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Optimizing Brain MR Imaging Protocols with Gadopentetate Dimeglumine: Enhancement of Intracranial Lesions on Spin-Density- and T2-Weighted Images

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To determine whether long TR MR imaging is best performed before or after IV administration of gadopentetate dimeglumine, we obtained spin-density- and T2-weighted images before and after contrast administration in 21 patients with known intracranial enhancing lesions. Of 25 lesions demonstrating enhancement on T1-weighted sequences, 21 showed mild or moderate enhancement on spin-density-weighted sequences and 20 showed mild enhancement on T2-weighted sequences. Importantly, no spin-density or T2 information was obscured by the administration of gadopentetate dimeglumine, and no T2 shortening effects were visible. Two new foci of enhancement were visible on postcontrast spin-density- and T2-weighted images that were missed on postcontrast T1-weighted images and on precontrast spin-density- and T2-weighted studies. Visualization of new areas of enhancement is the main advantage provided by the long TR images obtained after IV injection of gadopentetate dimeglumine. The most likely reason for the appearance of these newly visualized lesions is thought to be delayed enhancement. This imaging protocol also allows the display of adjacent edema or gliosis and enhancing lesions on a single image. Additionally, in three cases, posterior fossa phase-shift artifacts raised the suspicion of an enhancing lesion on postcontrast T1-weighted images, but the cerebellum was shown to be normal on the postcontrast spin-density- and T2-weighted studies.

On the basis of our results, we recommend obtaining long TR images after rather than before the administration of gadopentetate dimeglumine in patients with intracranial enhancing lesions.

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Several reports have documented the importance of gadopentetate dimeglumine in MR imaging of the brain [1–5]. Although enhancing lesions in the brain are best demonstrated on postcontrast T1-weighted images, long TR pulse sequences (i.e., spin-density- and T2-weighted images) remain essential to identify many nonenhancing abnormalities with increased water content, such as low-grade tumors or areas of edema, infarction, or demyelination. It has been recognized that some contrast enhancement occurs with spin-density-weighted imaging sequences [3]; however, little attention has been directed toward the relative advantages or disadvantages of combining contrast administration with long TR pulse sequences. In view of this, we undertook a study to compare pre- and postcontrast spin-density- and T2-weighted images to determine whether it is more advantageous to obtain long TR images before or after the administration of contrast material. Our aims were to determine the frequency with which contrast enhancement was discernible on spin-density- and T2-weighted images and whether such enhancement provided any additional information to that obtained with postcontrast T1-weighted images. We also analyzed the postcontrast long TR images to determine whether any new areas of enhancement were detected and, most importantly, whether any spin-density or T2 information was obscured after administration of gadopentetate dimeglumine.

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Materials and Methods

Twenty-one randomly selected patients with a variety of intracranial lesions that had demonstrated enhancement on previous CT or MR examinations were studied prospectively. Nine patients had recurrent gliomas, three patients had metastatic lesions, two patients had postoperative enhancement, two patients had meningiomas, one patient had a low-grade glioma, one patient had an inflammatory lesion, one patient had a pineal germinoma, one patient had a venous angioma, and one patient had an acute demyelinating plaque. Of the three patients with metastatic disease, one patient had three lesions while the other two patients had two lesions each. Thus, a total of 25 enhancing lesions were identified in the study population.

All patients were examined on a 1.5-T MR unit (GE, Milwaukee, WI). Imaging sequences prior to contrast injection consisted of sagittal and axial T1-weighted sequences, 600/20/2 (TR/TE/excitations), followed by axial spin-density- and T2-weighted images (2700/30–80/1). Following IV administration of 0.1 mmol/kg of gadopentetate dimeglumine (Magnevist, Berlex, Inc., Wayne, NJ), the axial T1-weighted and the axial long TR sequences were repeated with identical imaging parameters. Postcontrast images were completed within 20 min of injection. Slice thickness was 5 mm with a 2.5-mm interslice gap and 256 × 192 matrix.

All studies were reviewed by three neuroradiologists in a nonblinded fashion; a consensus opinion was reached as to the presence or absence of enhancement on each pulse sequence. Enhancement was graded as mild, moderate, or marked. Pre- and postcontrast spin-density- and T2-weighted images were compared to determine the degree of enhancement and the location and extent of all pathologic findings, both enhancing as well as nonenhancing. The enhanced spin-density- and T2-weighted images were also compared with the postcontrast T1-weighted images to determine if any areas of enhancement were obscured or if any additional areas of enhancement were identified on the long TR images. The presence and extent of

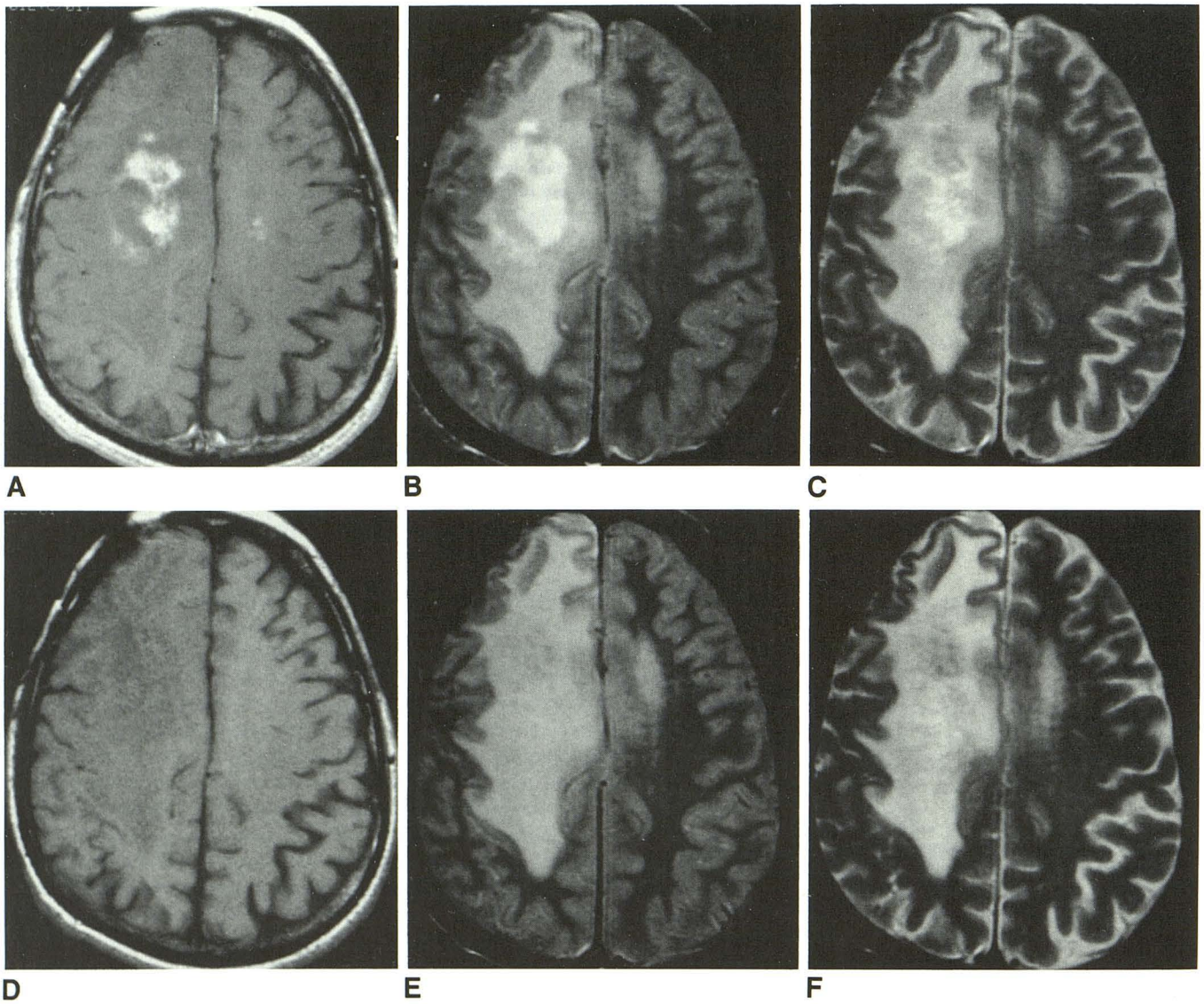

Fig. 1.—Patient with recurrent astrocytoma.
A–F, Postcontrast T1-weighted (*A*), spin-density-weighted (*B*), and T2-weighted (*C*) MR images distinguish enhancement of underlying lesion from adjacent edema as compared with unenhanced T1-weighted (*D*), spin-density-weighted (*E*), and T2-weighted (*F*) images.

nonenhancing abnormalities (such as edema or gliosis) were also evaluated on pre- and postcontrast long TR images to determine if contrast administration had any effect on these abnormalities.

Results

In this series of 21 patients, 25 enhancing lesions were identified on T1-weighted postcontrast MR images. Most of these lesions also demonstrated enhancement on the long TR pulse sequences. Twenty-one of 25 lesions enhanced on spin-density-weighted images and 20 of 25 lesions showed some perceptible enhancement on T2-weighted images. Of the four lesions that showed no discernible enhancement on postcontrast spin-density- or T2-weighted images there was only mild enhancement on postcontrast T1-weighted images. All lesions that demonstrated moderate to marked enhancement on the postcontrast T1 sequence also showed enhancement on the postcontrast long TR sequence.

In 14 of 16 cases with significant associated edema, concurrent visualization of the enhancing nidus of the lesion as well as the adjacent edema was possible on both the postcontrast spin-density- and T2-weighted images (Fig. 1). In these cases, nidus identification was generally not possible on the precontrast spin-density- and T2-weighted images. In six of nine cases with proved recurrent astrocytoma, the postcontrast spin-density- and T2-weighted images allowed improved differentiation of the enhancing recurrent tumor from an adjacent postsurgical cavity when compared with the precontrast spin-density- and T2-weighted images. In two cases with intracerebral hemorrhage, the postcontrast spin-density- and T2-weighted images showed an enhancing lesion that proved to be the origin of the hemorrhage. These lesions were not seen on precontrast long TR images, although they were seen on the postcontrast T1-weighted images.

Most importantly, direct comparisons of pre- and postcontrast long TR images demonstrated that no spin-density or T2 information was obscured after the administration of gadopentetate dimeglumine. Despite the theoretical concern that the T2 shortening effect of contrast medium might obscure pathologic changes, this did not occur in any of our cases. As expected, enhancement on the spin-density-weighted images was either equal to or, in most cases, less than that observed on T1-weighted images. Also as expected, more marked enhancement was observed on the spin-density-weighted images than on the corresponding T2-weighted images.

In two cases, new areas of enhancement were visualized on the postcontrast long TR pulse sequences that were not visualized on the postcontrast T1-weighted images. In one case of recurrent astrocytoma, spin density- and T2-weighted images demonstrated a new finding of dural enhancement (Fig. 2). In another patient with metastases from a renal cell carcinoma, spin-density images demonstrated enhancement within a hemorrhagic tumor that was not evident on enhanced T1 images. The visualization of these new areas of enhancement was the single most important advantage to obtaining the long TR pulse sequences after rather than before contrast administration. These new abnormalities are most likely related to delayed enhancement within these lesions rather than to increased sensitivity of the spin-density sequences to contrast enhancement.

We also noted three cases in which suspicious or equivocal lesions within the brainstem or cerebellum were seen on enhanced T1-weighted images. These were later proved to represent posterior fossa phase-shift artifacts and not pathologic abnormalities. In all three cases, these suspicious areas of abnormality were shown to be entirely normal on the postcontrast spin-density- and T2-weighted images that were

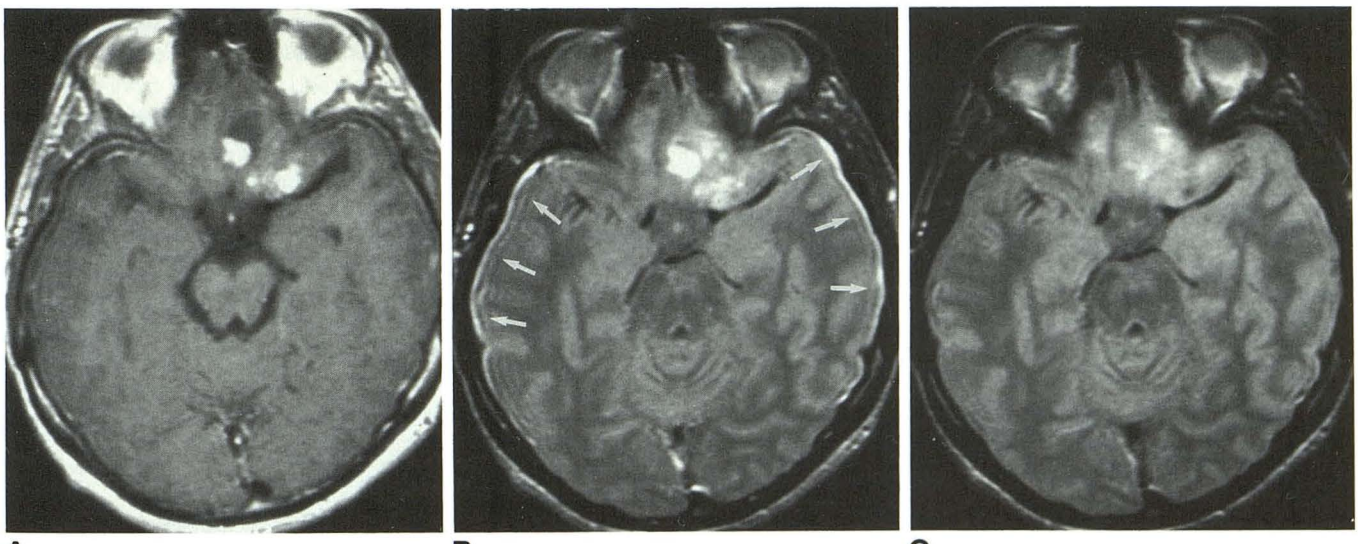

Fig. 2.—*A–C*, Lesion enhancement in patient with recurrent astrocytoma is demonstrated on postcontrast T1-weighted image (*A*) and postcontrast spin-density-weighted image (*B*). In addition, postcontrast spin-density-weighted image shows a new finding of bilateral dural enhancement (*arrows*) as compared with precontrast spin-density-weighted image (*C*). Finding was not apparent on *A*, and is most likely the result of delayed enhancement of the dura.

obtained with flow-compensation techniques (Fig. 3). These findings were later confirmed by follow-up MR imaging in all three cases.

Discussion

In clinical practice, long TR images are sometimes obtained and evaluated to determine whether contrast material should be administered. In addition, a small portion of MR brain examinations are performed to investigate such entities as chronic multiple sclerosis plaques that do not require further characterization by the administration of contrast material. Clearly, the results of this study will have little impact on either of these subsets of MR examinations. However, in an ever-increasing percentage of cases, it is known before the start of the MR examination that the clinical considerations can only be addressed with the use of contrast material. It is for this group of cases that our study was designed. While the most commonly used protocol consists of unenhanced T1 and long TR sequences followed by a T1-weighted enhanced examination, our study demonstrates several advantages to obtaining the long TR images after rather than before contrast administration.

There are two major reasons for the common practice of obtaining spin-density- and T2-weighted images before the administration of contrast medium. First, since the predominant effect of gadopentetate dimeglumine at the clinically used dosage is T1 shortening [6], there is no obvious advantage to obtaining T2-weighted images after its administration. Second, at high concentrations, gadopentetate dimeglumine has an additional effect of T2 shortening that could theoretically mask T2 abnormalities by causing a "paradoxical" decrease in signal intensity [6].

In the present study, contrast enhancement was visible in the vast majority of cases on both spin-density- and T2-weighted images. This enhancement was either equal to or, in most cases, less than that observed on T1-weighted images. The predominant mechanism for this enhancement is the residual T1 effects that are present on the spin-density-weighted and, to a much lesser degree, the T2-weighted images caused by incomplete recovery of the longitudinal magnetization vector during the finite TR interval of 2700 msec. This residual T1 effect is amplified by the use of a high-field-strength magnet (1.5 T), which prolongs the T1 relaxation time of tissues compared with a low- or mid-field-strength MR system. Although the T1 information displayed on these spin-density-weighted images would be demonstrated better by a T1-weighted image obtained at the same time, the spin-density images provide the advantage of displaying both contrast enhancement and T2 information on a single image. This improves the appreciation of the spatial relationships between different components of a pathologic entity, such as tumor nidus or edema. Most significantly, no T2 shortening effects were observed in our study. Direct comparisons of the pre- and postcontrast long TR images demonstrated that no spin-density or T2 information was obscured after the administration of contrast material. Nonenhancing abnormalities such as edema and gliosis were equally well shown on the pre- and postcontrast long TR sequences in all cases. Thus, equivalent T2 information can be obtained with both pre- and postcontrast imaging.

We also analyzed these studies to determine if additional abnormalities could be identified on the postcontrast long TR images. New lesions with enhancement were defined in two of our 21 patients on the postcontrast spin-density- and T2-weighted images that were definitely not present on the precontrast long TR images or on the short TR images, with or without contrast enhancement. In one case, bilateral areas of postoperative dural reaction with enhancement were noted, and in another case an enhancing metastatic lesion adjacent to an area of hemorrhage was noted. These findings are most likely the result of delayed enhancement within these lesions, a phenomenon noted previously on MR images of intracranial disease [7, 8]. It should be noted that in this study the postcontrast long TR images were always acquired after the postcontrast T1-weighted images. Thus, a delay of approxi-

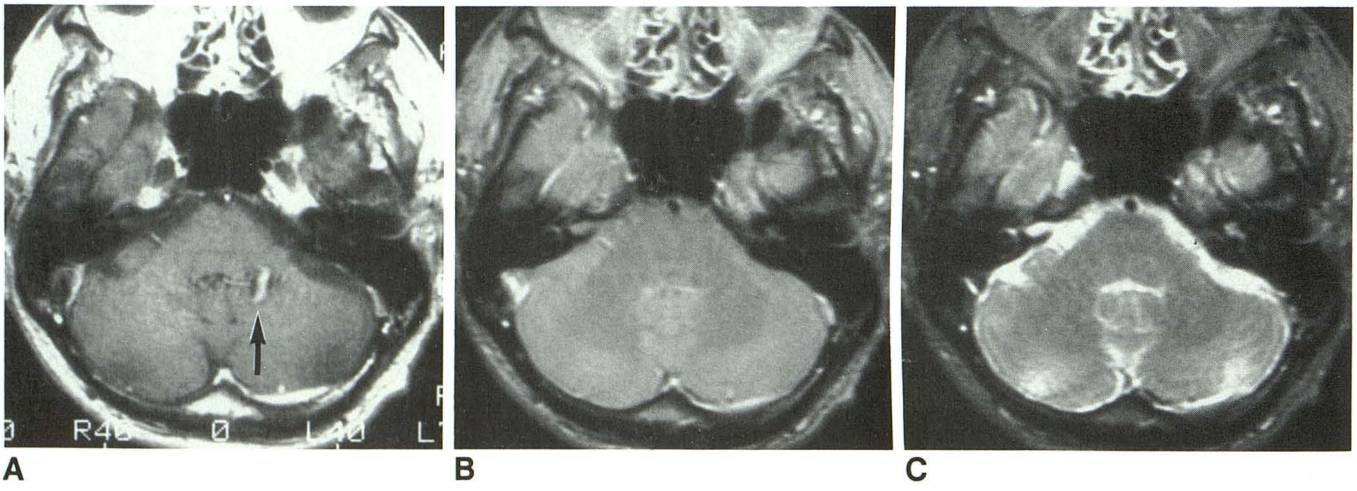

Fig. 3.—*A–C*, Postcontrast T1-weighted image (*A*) shows area of increased signal (*arrow*) adjacent to fourth ventricle, representing flow-related artifact. Postcontrast spin-density-weighted (*B*) and T2-weighted (*C*) images show that this area is normal and confirm that the finding is an artifact.

mately 10–15 min occurred between the time of contrast injection and the acquisition of long TR images, and a relative delay of 7–10 min occurred between postcontrast T1- and T2-weighted images. Our results indicate that the delay necessitated by obtaining postcontrast long TR images can provide additional diagnostic information without prolonging the overall imaging time for the patient. This was the main advantage to obtaining the long TR images after rather than before contrast administration. While information similar to that found in our two patients could probably be obtained by using delayed postcontrast T1-weighted sequences, such a protocol would add to the imaging time.

Another major advantage of the flow-compensated postcontrast long TR images is the improved differentiation of lesions from posterior fossa phase-shift artifacts on enhanced T1-weighted sequences. These artifacts are caused by misregistration of signal from enhanced flowing blood in the sigmoid and lateral sinuses that propagates across the brainstem and/or cerebellum along the phase-encoding axis. When these artifacts fortuitously reinforce one another, they may simulate enhancing lesions on postcontrast T1-weighted images, causing a confusing diagnostic dilemma. The routine use of flow-compensation techniques on postcontrast T1-weighted images has been proposed to eliminate these artifacts [9]. However, in our experience, the use of these techniques has not been entirely successful for several reasons. First, flow-compensating, gradient-moment-nulling techniques reduce the number of slices possible for a given TR, thus precluding coverage of the entire head with the use of a single, short TR sequence. This limitation can be rectified by either increasing TR, which has the disadvantage of reducing the T1-weighting of the image, or performing two separate axial T1 sequences to cover the entire head, which has the disadvantage of increasing the examination time. Second, flow compensation produces increased signal within superficial veins due to improved refocusing of signal from enhanced, slow-flowing blood. This can sometimes mimic leptomeningeal enhancement. Third, flow compensation increases signal from CSF due to reduced pulsation-related signal loss. This results in decreased image contrast between CSF and brain. Finally, and most importantly, the flow compensation used with our scanner is only a first-order (velocity) correction. Although velocity-compensated techniques reduce posterior fossa phase-shift artifacts, they do not completely eliminate them, since residual artifacts are still produced as a result of acceleration and "jerk" motions of flowing blood. For these reasons, and because of our own previous experiences, we do not routinely use gradient-moment-nulling techniques on postcontrast T1-weighted images.

Long TR images, on the other hand, show significantly fewer of these artifacts through a combination of gradient-moment-nulling techniques (used routinely with our long TR imaging sequences in the head and spine), prolonged echo-delay time that results in a fewer number of flowing protons remaining within the imaging volume between application of the 90° and 180° RF pulses, and decreased signal from blood due to T2 decay effects. Thus, absence of increased signal on the long TR images can clarify an area of suspicion on an enhanced T1-weighted study. The presence of increased signal on both sequences would confirm a lesion. This point is valid, since postcontrast long TR images, as demonstrated by this study, show enhancement in virtually all lesions that have moderate or intense enhancement on T1-weighted images. The only cases that did not show enhancement on the long TR studies had only mild degrees of enhancement on the corresponding postcontrast T1 weighted images. Thus, routine use of postcontrast long TR imaging protocols will help to identify posterior fossa artifacts with a greater degree of certainty.

Conclusions

In view of the findings that contrast enhancement is visible on spin-density- and T2-weighted sequences in most cases and that no T2 information is obscured by gadopentetate dimeglumine, our results indicate several advantages for obtaining spin-density- and T2-weighted images after rather than before contrast administration. The primary advantages are improved detection of some lesions that enhance slowly because of slow blood-brain-barrier leakage and improved differentiation between enhancing lesions and flow-related phase-shift artifacts in the posterior fossa. Importantly, the total MR examination time is not prolonged. This imaging protocol also provides the ability to display both enhancing lesions and edema or gliosis on a single image. Therefore, the recommended imaging protocol for cranial MR imaging with gadopentetate dimeglumine consists of a sagittal localizing sequence and a baseline T1-weighted axial sequence before contrast administration, followed by axial T1-, spin-density-, and T2-weighted images after the administration of contrast material. Additional supplemental views can be added as needed.

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