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Comparison Between Diffusion Tensor Imaging and Conventional MR Imaging Sequences in the Detection of Spinal Cord Abnormalities

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could have contributed to the rebleed. The coil mass likely began to be displaced and finally prolapsed across the torn ventral wall of the aneurysm, resulting in a fatal rebleed.

We cannot disagree with the conclusion that coiling of aneurysms cannot protect all patients from rehemorrhage. There are many examples of how undercoiling can lead to rehemorrhage; however, we believe this to be an example of how overcoiling can lead to rehemorrhage. The hypothesis of re-rupture as a consequence of recanalization of a partially thrombosed aneurysm cannot be entirely excluded; however, we would not expect extrusion of coils into the subarachnoid space if this were the etiology.

Although we agree that so-called bioactive coils may improve permanence of coil embolizations, we disagree with your conclusion that bioactive coils would have made any difference in the outcome in this patient. The stated absence of organized thrombus within the aneurysm lumen after a re-rupture should not lead the reader to believe that no fibrotic reaction occurred at all during the 2 weeks that the coils were in place. It is entirely possible that the more mature thrombus surrounding the coil mass may have extravasated into the subarachnoid space along with the coils.

In conclusion, we do not believe that this case represents a failure of detachable coils but rather a consequence of incorrect selection of coil size and overly aggressive delivery technique.

Reference

1. Bendszus M, Hagel C, Maurer M, et al. **Fatal recurrent subarachnoid hemorrhage after complete endovascular aneurysm occlusion.** *AJNR Am J Neuroradiol* 2006;27:2058–60

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Reply:

In summary, it is stated in this letter regarding our case presentation[1] that we used oversized coils with balloon assistance, which, in combination, resulted in delayed aneurysm rupture due to increased wall stress. This letter relies on several incorrect assumptions that we cannot accept. First, this aneurysm measured 3 mm as determined by intra-arterial 3D angiography (not CT angiography as stated by the authors of the letter). The assumption of a size of 2–2.5 mm is simply incorrect. Second, this aneurysm did indeed have a wide neck. At our institution, we never use balloon assistance as the first line of treatment. Rather, we always attempt deploying a spheric coil first and use a balloon only when this deployment is unsuccessful. Before blaming an incorrect or overly aggressive technique, one should read the manuscript carefully. ("Because of the wide neck of the aneurysm, it was not possible to place a coil in the aneurysm without it prolapsing into the basilar artery."[1]).

Third, the assumption that the coil mass in Fig 2*A* is larger than the aneurysm in Fig 1*A* is incorrect. Figures 2*A* and 2*B* are more magnified than Fig 1*A*. Looking at Figs 3*A* and 3*B* with a magnification similar to that in Fig 1*A*, one realizes that the coil mass very closely corresponds to the initial aneurysm size. Fourth, the argument that delayed re-hemorrhage occurred as a result of volume expansion and induced hypertension as a cause of overdistention of the aneurysm is wrong. As we stated in the manuscript, re-rupture occurred 14 days after initial rupture, when the patient had stabilized and was scheduled for rehabilitation the next day. Vasospasm had completely subsided and re-rupture occurred 5 days after cessation of hypervolemic/hypertensive therapy. Fifth, extrusion of coils outside the aneurysm sac is a frequent finding in coiled aneurysms undergoing surgery later and must not be mistaken for overpacking.[2]

Finally, as we stated in the article, there was no histologic evidence for tissue response such as thrombus organization, macrophages, or fibrin formation. We are amazed at why the authors, without providing evidence, stated that the reader should not be led to believe that no fibrotic reaction occurred at all during the 2 weeks that the coils were in place. As we stated in our article,[1] this case differed histologically from findings reported for aneurysms at a similar time after coil embolization.[3-5] Re-rupture may have occurred for several reasons in this patient, but we cannot accept the allegation that this was most likely caused by incorrect or overly aggressive treatment.

References

1. Bendszus M, Hagel C, Maurer M, et al. **Fatal recurrent subarachnoid hemorrhage after complete endovascular aneurysm occlusion.** *AJNR Am J Neuroradiol* 2006;27:2058–60
2. Veznedaroglu E, Benitez RP, Rosenwasser RH. **Surgically treated aneurysms previously coiled: lessons learned.** *Neurosurgery* 2004;54:300–03
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4. Stiver SI, Porter PJ, Willinsky RA, et al. **Acute human histopathology of an intracranial aneurysm treated using Guglielmi detachable coils: case report and review of the literature.** *Neurosurgery* 1998;43:1203–08
5. Bavinzski G, Talazoglu V, Killer M, et al. **Gross and microscopic histopathological findings in aneurysms of the human brain treated with Guglielmi detachable coils.** *J Neurosurg* 1999;91:284–93

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Comparison Between Diffusion Tensor Imaging and Conventional MR Imaging Sequences in the Detection of Spinal Cord Abnormalities

We read with interest the articles by Renoux et al[1] and Facon et al,[2] respectively, in the October 2006 and in the June–July 2005 issues of the *AJNR*.

The authors evaluated the diagnostic accuracy of diffusion tensor imaging (by using the apparent diffusion coefficient and fractional anisotropy) in inflammatory diseases of the spinal cord[1] and in spinal cord compression.[2] In these 2 articles, diffusion tensor imaging was compared with T2-fast spin-echo (FSE)–weighted sequences. The authors found a higher sensitivity in the detection of spinal cord abnormalities with diffusion tensor imaging than with T2-FSE–weighted sequences in both articles.[1,2]

We draw the authors' attention to the previously published reports about the diagnostic accuracy of spinal cord abnormalities with conventional MR imaging sequences. We cite only 2 of them because of the restriction on the number of references. These reports showed that short τ inversion recovery FSE (STIR-FSE) sequences may have a higher sensitivity than T2-FSE–weighted sequences in the detection of spinal cord lesions.[3,4] Campi et al[3] and Rocca et al[4] concluded that STIR-FSE sequences had a higher sensitivity in the detection of demyelinating lesions. Furthermore, Campi et al showed a better demarcation and maybe a better sensitivity in the detection of spinal cord abnormalities in a group of patients with myelopathy of unknown etiology with STIR-FSE sequences.

We hypothesize that the best conventional sequence, with the

highest sensitivity in the detection of spinal cord abnormalities, may be the STIR-FSE sequence. We think that future studies evaluating the diagnostic accuracy of diffusion tensor imaging in the detection of spinal cord abnormalities should include STIR-FSE sequences.

References

1. Renoux J, Facon D, Fillard P, et al. **MR diffusion tensor imaging and fiber tracking in inflammatory diseases of the spinal cord.** *AJNR Am J Neuroradiol* 2006;27:1947–51
2. Facon D, Ozanne A, Fillard P, et al. **MR diffusion tensor imaging and fiber tracking in spinal cord compression.** *AJNR Am J Neuroradiol* 2005;26:1587–94
3. Campi A, Pontesilli S, Gerevini S, et al. **Comparison of MRI pulse sequences for investigation of lesions of the cervical spinal cord.** *Neuroradiology* 2000;42:669–75
4. Rocca MA, Mastronardo G, Horsfield MA, et al. **Comparison of three MR sequences for the detection of cervical cord lesions in patients with multiple sclerosis.** *AJNR Am J Neuroradiol* 1999;20:1710–16

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Reply:

Short τ inversion recovery (STIR) MR imaging has a better sensitivity to detect spinal cord lesions compared with regular spin-echo (SE) T2-weighted imaging as stated in this letter. The purpose of our work in the 2 cited articles[1,2] was mostly based on how diffusion tensor imaging (DTI) and fractional anisotropy (FA) could help locate lesions within the spinal cord and, at the same, how to better understand the pathophysiology of inflammatory myelitis as well as spinal cord compressions. Sensitivity of MR SE T2-weighted imaging sequences was only assessed to hypothesize pathophysiologic patterns of these diseases. Indeed, our observations led us to draw a scheme of FA value changes that may correlate with patient outcome. If conventional MR imaging sequence sensitivity plays an important role in detecting spinal cord lesions, this sensitivity could not be used to assess understanding of the neuronal cluster regeneration that occurs in such diseases, contrary to the use of DTI and FA values. We used SE T2-weighted imaging instead of STIR because we focused more on pathophysiology than on MR imaging accuracy.

The authors of the letter are right: STIR is better than SE T2. However, only DTI, FA, and fiber tracking may help to understand these diseases at the water molecule level.

References

1. Renoux J, Facon D, Fillard P, et al. **MR diffusion tensor imaging and fiber tracking in inflammatory diseases of the spinal cord.** *AJNR Am J Neuroradiol* 2006;27:1947–51
2. Facon D, Ozanne A, Fillard P, et al. **MR diffusion tensor imaging and fiber tracking in spinal cord compression.** *AJNR Am J Neuroradiol* 2005;26:1587–94

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Erratum

The authors regret the following errors that appeared in "A Preliminary Report of Brain Edema in Patients with Uremia at First Hemodialysis: Evaluation by Diffusion-Weighted MR Imaging" (*AJNR Am J Neuroradiol* 2007;28:68–71):

1. The first author, C.L. Chen, is affiliated with Division of Nephrology, Kaohsiung Veterans General Hospital, Kaohsiung and the Department of Medicine, National Yang-Ming University School of Medicine, Taipei, Taiwan and the Institute of Clinical Medicine, National Cheng Kung University School of Medicine, Tainan, Taiwan.
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