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Fatal Recurrent Subarachnoid Hemorrhage after Endovascular Aneurysm Occlusion from Overdistention of the Aneurysm Wall

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measurement or the relative luminal reduction. MR imaging provides noninvasive methods of qualifying carotid plaque.[9] CTA shows some promise in the qualification of plaques as well, showing plaques as being fatty or calcified or having varying densities.[4]

Halo artifacts and indistinct edge definition are a challenge in CTA, as they are with other angiography techniques whether performed by MRA, digital subtraction angiography (DSA), or old film-screen techniques. This is inherent in x-ray physics as well in that of digital imaging display. There is inherent limitation to how much magnification one can do to carry out a measurement because magnification will blur the edges. For NASCET, measurements obtained showed an extremely high kappa of consistency,[8] despite this limitation. In our study, we placed our measurement calipers half-way between the visualized attenuated CTA contrast luminogram edge and the outer halo,[1-4] mimicking measurements acquired in NASCET, which then used a jeweler's eye piece to look at stenosis on angiographic films and DSA.[8]

CTA is now the preferred angiographic technique at many sites to quantify carotid stenosis due to the lack of stroke risk, ease of standardization of CTA, the quick time for the examination (seconds to acquire images from arch to vertex), and the high-quality data produced. It is understandable that those in favor of duplex sonography carotid imaging could be concerned about the capabilities of carotid CTA. Duplex sonography is an excellent screening technique to detect carotid plaque within a narrow window in the neck, with correlations to percentage stenosis from angiography that generally have rather wide numeric ranges. Due to the stroke risk and the resource-intensive nature of conventional angiography, the decision to perform endarterectomy is based on sonography data in some centers, without more accurate angiographic measures. Carotid duplex sonography scanning, however, is also relatively labor-intensive, requiring very highly skilled technologists to achieve accuracy. Adding orbital and transcranial Doppler is required if some distal information of the intracranial circulation is desired.

CTA can be performed within a few seconds without stroke risk and with excellent visualization of all vessels from arch to vertex. Compared with MRA, sonography, and conventional angiography, CTA is the fastest, is easily standardized (between patients, scanners, and technologists), and provides high-resolution images of the intracranial/extracranial vessels as well as the surrounding soft tissues. Additionally, CTA has no stroke risk and demands little time of labor-intensive resources.

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We read with great interest the article by Bendszus et al[1] in which successful coil embolization was followed by fatal re-rupture 2 weeks later.

In reviewing the case presentation, we made a number of observations from which we would like to propose an alternative explanation of the eventual outcome. Although there are obviously institutional differences in how patients like these are managed, we noted a number of unique points that may have contributed to an increase in periprocedural risk.

The conventional and CT angiographic images submitted are of high quality and demonstrate an aneurysm of the basilar terminus measuring approximately two thirds of the diameter of the basilar artery. It is not clear how the aneurysm was determined to be 3 mm in diameter, but because the average basilar artery typically measures approximately 3 mm in diameter, one would suspect that the actual dimension of the aneurysm would more likely be in the range of 2–2.5 mm. The neck of the aneurysm is not well demonstrated by angiography but appears rather well defined by CT angiography and would not be considered wide-necked. On the basis of the images submitted, we would suggest that the coils selected may have been too large and that the aneurysm might well have been successfully treated with fewer 2- or 2.5-mm coils, without the need for balloon assistance. We do not routinely proceed directly to the balloon-assist technique until we have first attempted direct unassisted coil embolization. We would also suggest that although balloon assist is useful in the placement of the coils within wide-necked aneurysms, using a balloon might lead to overpacking in small aneurysms. This can result in a relatively greater degree of pressure against the wall of the aneurysm that could, in turn, lead to an increased risk of rupture. We have long since learned, from the experience of endoluminal balloon embolization of aneurysms, that increased and asymmetric stresses on the wall of an aneurysm predispose to rupture.

One principal advantage of the detachable coil technique over balloon embolization is a lower and more symmetric distribution of radial forces within a treated aneurysm and proved association of a lower incidence of aneurysm rupture. We would point out that the dimensions of the coil mass in Fig 2*A* are significantly larger than those of the untreated aneurysm in Fig 1*A*. It would, therefore, appear likely that the aneurysm was overdistended, resulting in a tear of the ventral wall, by use of 12-cm oversized coils and the balloon-assist technique. The patient was subsequently diagnosed with vasospasm. Assuming that the patient was being volume expanded and was hypertensive at the time of her re-rupture, it is quite possible that the increased volume and pressure in the face of an overpacked aneurysm

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.

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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