AJNR

Diffusion-Weighted Imaging of Thromboembolic Events Associated with Coil Embolization of Intracranial Aneurysms

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AJNR Am J Neuroradiol 2004, 25 (10) 1861-1862
http://www.ajnr.org/content/25/10/1861

Diffusion-Weighted Imaging of Thromboembolic Events Associated with Coil Embolization of Intracranial Aneurysms

I read with interest the article by Soeda et al (1) in the January 2003 issue of the *AJNR* reporting on the incidence and appearance on diffusion-weighted (DW) imaging of thromboembolic events associated with Guglielmi detachable coil (GDC) embolization of intracranial aneurysms. DW imaging studies showed hyperintense lesions in 40 of 66 patients after 66 embolizations. Of these presumed thromboembolic events, 16 (24%) were symptomatic and 24 (36%) were clinically silent.

In another article, the same authors reported the incidence of thromboembolic events associated with GDC embolization of asymptomatic posterior circulation aneurysms (2). DW imaging studies, performed in 26 patients (26 aneurysms) showed hyperintense lesions in 69% of patients, of which 27% were symptomatic and 42% were silent (2). According to the authors, the overall risk of thromboembolic events after GDC treatment of unruptured aneurysms varies from 61% to 69% (approximately 25% are symptomatic and 40% are silent) (1, 2). These results are at odds with those reported by others, including us (3). If the results of Soeda et al reflect the true percentage of thromboembolic complications, they are worrisome. If, as I think, they reflect some bias in their methodology, their data need to be addressed because they may cast an unfavorable shadow on what is now the treatment of choice for many cerebral aneurysms.

An important consideration is that these authors did not perform DW imaging before their procedures. It is conceivable that, in patients with subarachnoid hemorrhage and vasospasm, preprocedural DW imaging may have shown small infarctions. Recent ischemia may also be due to embolic fragments migrating from an aneurysm or from other origins. In addition, Soeda et al evaluated only DW imaging but did not provide apparent diffusion coefficients. Thus, it is not clear whether what they saw were true infarctions. Soeda et al also used systemic anticoagulation to maintain the activated clotting time at 2–2.5 times above baseline throughout their procedures (1). In my opinion, this degree of anticoagulation may not be sufficient during some of the more complex procedures such as remodeling technique and may account for the fact that ischemic lesions were detected at 73% of their patients after the balloon-assisted technique. In their report dealing with posterior circulation aneurysms, hyperintense lesions were seen in all three patients treated with the balloon-assisted technique (2).

Also, in the *AJNR,* we reported our initial experience by using DW imaging in patients treated with GDC for ruptured and unruptured intracranial aneurysms (3). Silent acute infarctions were observed in two of 21 patients. We continue by using DW imaging to evaluate most of our patients who have aneurysm embolization, and our data (not published) are in accordance with our initial results.

In an editorial, Nichols commented on the discrepancy in the frequency of DW imaging abnormalities seen after GDC treatment between our series and that of Rordorf et al (3–5). I agree with Nichols that, after coil treatment of aneurysms, many variables may account for the disparities in the percentage of DW imaging abnormalities, both symptomatic and silent. Factors include differences in anticoagulation regimens during and after treatment, administration of intravenous aspirin, aneurysm characteristics, degree of final aneurysm occlusion, number of guiding catheters and microcatheters, clinical status of patients, and operator experience. In addition, it is conceivable that some complications may arise from the "angiographic phase" of the procedure, especially in elderly patients or in those with a vascular risk profile. Bendszus et al (6) used DW imaging to evaluate a silent embolism after diagnostic angiography and surprisingly reported hyperintense lesions in 17 (26%) of 66 patients. Conversely, Britt et al (7) studied 20 patients and reported no evidence of new DW imaging abnormalities after cerebral angiography.

In conclusion, I believe that many factors other than just coil embolization may account for the high number of acute strokes described by Soeda et al. Their articles (1, 2) remind us that embolization of intracranial aneurysms is not without risks and that a meticulous technique is needed to avoid them.

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Reply

We thank Dr. Biondi for the interest and comments regarding our articles (1, 2). Although she agreed that our articles suggested that GDC embolization of intracranial aneurysms is not without risks and a "meticulous" technique is needed to avoid thromboembolic events, there was doubt concerning our methodology. First, although five patients with ruptured aneurysms might have had infarctions before GDC treatments, only one of them had hyperintense lesions after treatments. Second, although embolic fragments from small aneurysms might cause thromboembolism, in our second study, 78% of patients showed hyperintense lesions proximal to treated aneurysms. These results suggested most of the emboli were due to intravascular devices during the procedures rather than aneurysm clot before, during, or after treatment. Third, although cardiac arrhythmia and complicated plaques from carotid artery or aorta may cause thromboembolism, no patient had such complications on preprocedural workup.

Dr. Biondi pointed out that our thromboembolic rate was higher than their series and comments that our data are worrisome. It is true, however, that our "negative" data will prompt us to change management of unruptured intracranial aneu-

rysms, including antithrombotic regimens, degree of final occlusion, number of coils or catheters used, and patient selection. In addition, we stated previously that these data would stimulate the development of new coils, catheters and other embolic agents such as liquid materials. In fact, we changed management including antithrombotic regimens such as per OS antiplatelet agent before treatment, as a result and with the advent of coils and catheters, thromboembolic complications were observed less frequently than our previous published series (data not published).

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Carotid Pseudofenestration: The Double-Barrel Peril

We enjoyed reading the article, "Pseudofenestration of the Cervical Internal Artery: A Pathologic Process that Simulates an Anatomic Variant" by Gailloud et al (1). We would like to add two more cases to their series in support of their argument that apparent fenestrations of the carotid artery are acquired lesions and not congenital anomalies. Furthermore, we would like to raise the issue that there may be prognostic features that portend a more benign or malignant course, which should be highlighted in any discussion with the treating physician.

Case 1. A 69-year-old man was referred for evaluation of two recent transient ischemic attacks. The first attack, which occurred while he was taking aspirin for coronary artery disease, consisted of left hemiparesis. He was placed on Plavix (clopidogrel), but 2 months later experienced a transient episode of diplopia. MR angiography findings obtained at an outside institution were interpreted as showing carotid stenosis, so he underwent diagnostic cerebral angiography for further evaluation (Fig 1). This study showed the entire right internal carotid artery (ICA) was markedly abnormal, with focal and short segment strictures and areas of fusiform dilatation, which suggest an underlying dysplastic process with superimposed pseudoaneurysms. At the cervicopetrous junction, the ICA divided into two lumens of nearly symmetrical caliber that ran nearly parallel to each other, approximately 2 mm apart, and then joined distally at approximately the level of the anterior carotid genu. The limbs of this apparent fenestration were also irregular, with a small pseudoaneurysm at the proximal bifurcation. The angiogram also showed that the other ICA had a small ulcerated plaque at the bulb, and both vertebral arteries were occluded at their origins and segmentally reconstituted from muscular branches from the ascending cervical artery. This patient failed a balloon occlusion test and is scheduled to undergo a right extracranial-to-intracranial arterial bypass followed by sacrifice of the diseased vessel.

Case 2. A 33-year-old woman developed "pressure" headaches after a difficult delivery of twins, followed 4 days later by vision changes, difficulties understanding, and numbness over the left face and hand. Angiography (Fig 2A) demonstrated dissections of both ICAs at the craniocervical junction as well as the proximal left vertebral artery, accompanied by a dysplastic appearance of the distal left ICA consistent with fibromuscular dysplasia, and a small, unruptured, wide-necked anterior communicating artery aneurysm. This aneurysm was uneventfully clipped, after which she was anticoagulated with coumadin for 6 months and then switched to aspirin. Her symptoms resolved during this time. Follow-up angiography showed the left ICA dissection had healed with two smooth lumens of nearly equal caliber, spiraling around each other and communicating both proximally and distally without any restriction in flow (Fig 2B, -C). The dissections in other vessels had also improved in appearance. This patient continues to be conservatively treated with aspirin and 10 months later remains asymptomatic with stable anatomy on follow-up studies.

The location of an apparent fenestration at a site prone to spontaneous dissections, in the setting of an underlying vasculopathy, further supports the theory that a "double-barreled" carotid artery is likely the result of an injury that dissected through a segment of the vessel to communicate both proximally and distally. Our second case illustrates a progression of changes that lead to this phenomenon. It is instructive to note the high frequency of strokes in Gailloud's series and ours to remind us of the need to notify our clinical colleagues quickly of the potential danger if left untreated. Not all diseased vessels, however, must be necessarily sacrificed. Angiographic features that would suggest a more worrisome course would include marked irregularities in the vessel walls, restricted flow, the presence of a pseudoaneurysm, and extension over time. Without these

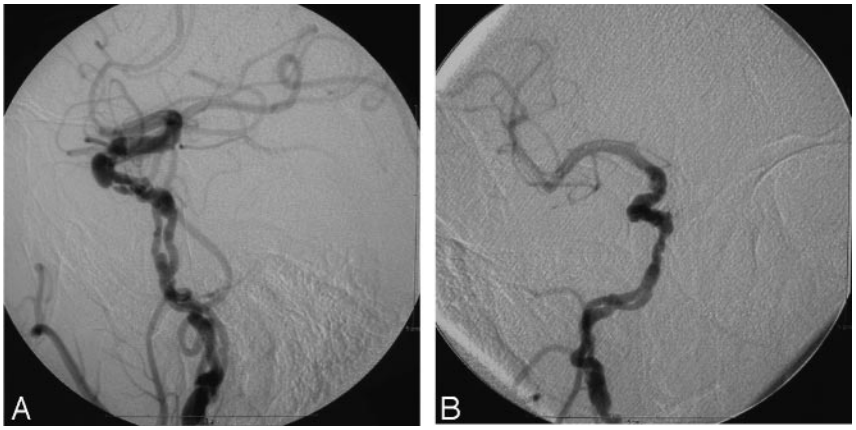

FIG 1. A 69-year-old man with right ICA pseudofenestration. Digital subtraction angiography (DSA), right common carotid injection, lateral (*A*) and frontal (*B*) views. Both lumens of the fenestrated segment are markedly irregular with focal stenoses alternating with areas of fusiform dilatation. The ICA has a narrow caliber and striking dysplastic changes throughout its entirety.

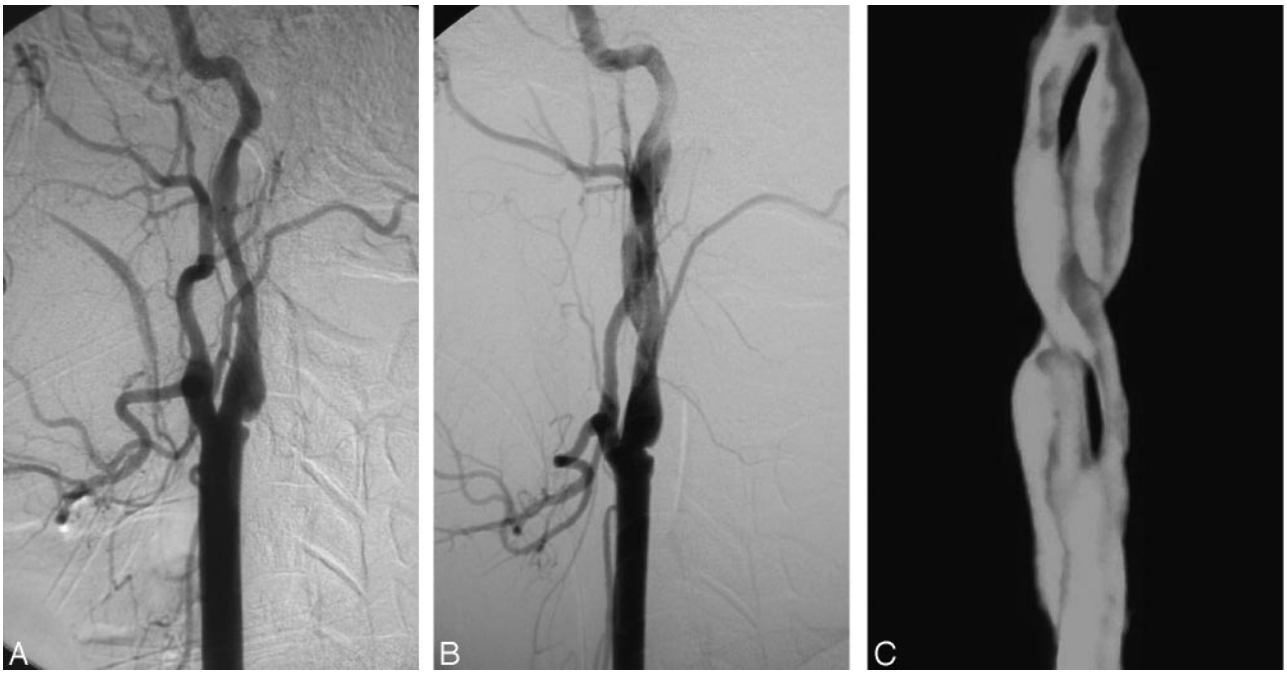

FIG 2. A 33-year-old woman with left ICA dissection, characterized by irregular caliber changes of the single lumen just below the skull base (*A,* left common carotid injection, lateral DSA). After 6 months of anticoagulation, the dissection has healed with the formation of a relatively smooth, spiraling, double lumen configuration communicating both proximally and distally without any flow limitation (*B,* lateral DSA; *C,* 3D rotational angiogram with the external carotid artery cut away to show the ICA).

features, a patient may be conservatively treated with an anticoagulant or antiplatelet regimen as in our second case.

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Influenza-Associated Encephalitis-Encephalopathy with a Reversible Lesion in the Splenium of the Corpus Callosum: Case Report and Literature Review

We read with interest the article by Takanashi et al (1) in the May 2004 issue of the *AJNR*. The authors reported two cases involving lesions of splenium of the corpus callosum in patients with influenza-associated encephalitis/encephalopathy. Although they described a reversible lesion in the splenium of the corpus callosum, they did not investigate causes other than encephalitis/encephalopathies. We would like to emphasize previously published reports regarding such lesions.

For instance, Kim et al (2) initially described transient splenial lesions, as revealed on MR imaging, in 1999. Focal nonhemorrhagic lesions of the corpus callosum are rare, but they have been described in various clinical conditions, and little is known about the underlying mechanisms. Although the pathogenesis is not understood, awareness of the fact that this is a reversible focal lesion may help prevent unnecessary invasive diagnostic and therapeutic intervention. Various pathologic conditions, such as multiple sclerosis, trauma, neoplasm, infarct, leukodystrophies (especially adrenoleukodystrophy), AIDS dementia complex, and Marchiafava-Bignami disease, may also involve the corpus callosum (3).

Kim et al (2) reported such lesions in six patients with focal epilepsy. They classified the lesion as demyelination and hypothesized that anticonvulsive drug toxicity was the cause. Similar lesions have been described in epileptic patients in the postictal period as well. These lesions have been interpreted as transient focal edema due to transient physiologic alteration in the callosal fibers caused by seizure activity. Association of such splenial lesion with acute cerebellitis due to herpes simplex, and its regression within 72 hours, has also been reported (4).

In support of these observations, we herein briefly report our experience with a 24-year-old man who presented with headache and apathy lasting for 5 days. He had no remarkable medical history, he was not receiving any medication, and he denied alcohol abuse. Scalp electroencephalograms were normal, and CSF testing showed slight elevation of protein levels. Results of blood tests were unremarkable for demyelinating or other systemic conditions, metachromatic leukodystrophy, antiphospholipid antibody syndrome, or HIV infection. Titers of serum immunoglobulins against human herpes viruses, herpes simplex virus, Epstein-Barr virus, varicella zoster virus, rubella virus, measles virus, papovavirus, enterovirus, influenza virus, and rotavirus, and *Mycoplasma pneumonia* were normal. Cranial MR images showed a focal nonenhancing lesion in the splenium of the corpus callosum, which totally regressed within 4 weeks without medication (Figs 1–2). We believe that this case represented an instance encephalitis/encephalopathy, but this could not be proved.

The case that Takanashi et al (1) reported was believed to have same underlying cause as ours, but to our knowledge the association of such a lesion with apathy or headache has not

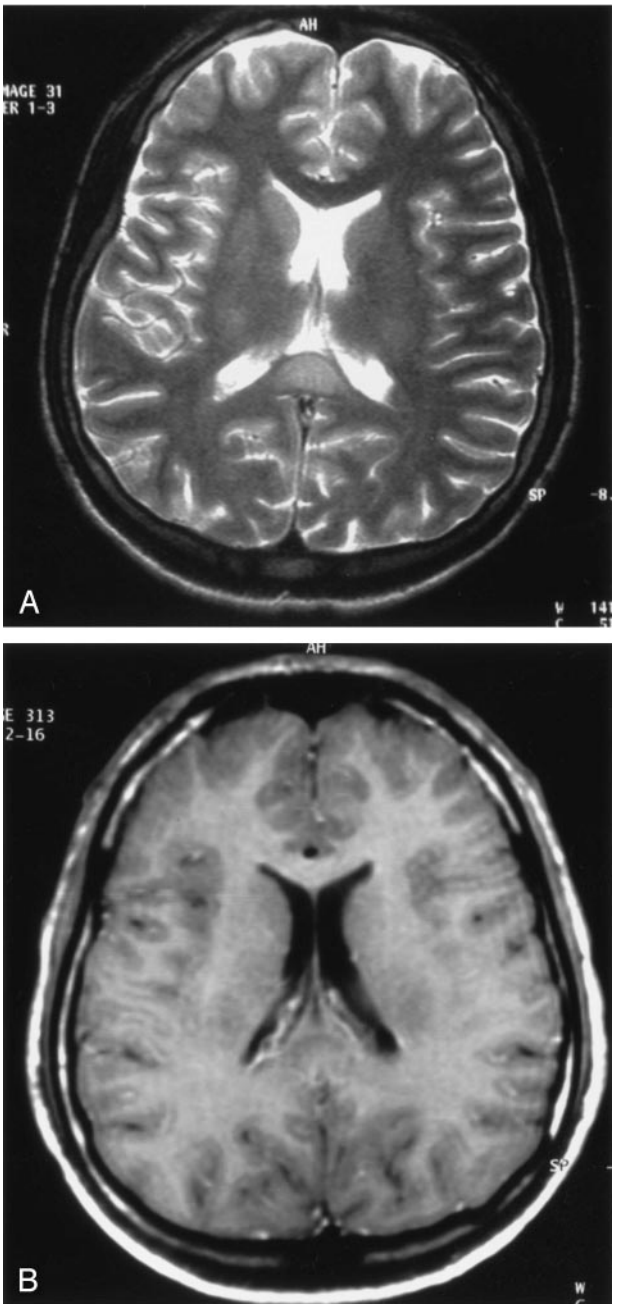

FIG 1. Cranial MR images obtained in a 24-year-old man show a focal nonenhancing lesion.

A, Axial T2-weighted turbo spin echo image (TR 5000 ms, TE 99 ms; NEX 2) shows hyperintense lesion in the splenium of the corpus callosum.

B, Axial T1-weighted, contrast administered spin echo image (TR 600 ms, Te 14 ms; NEX 2) reveals hypointense character of the lesion, without contrast enhancement.

been reported. Polster et al (5) have suggested that such a transient splenial lesion appears to be a nonspecific endpoint of a different disease processes leading to vasogenic edema. This seems the most probable pathophysiologic explanation of such splenial lesions, as in our case.

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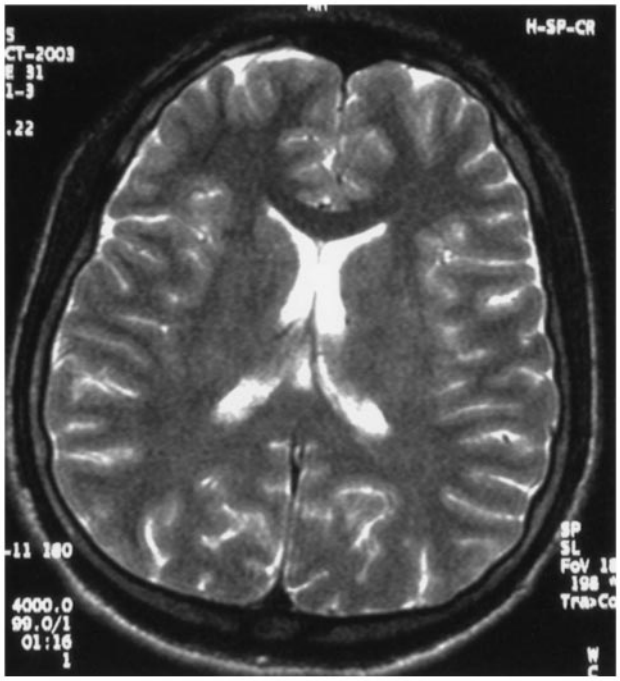

FIG 2. Axial T2-weighted turbo spin echo image (TR 5000 ms, TE 99 ms; NEX 2) shows resolution of the lesion in the splenium of the corpus callosum.

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Abnormal Fluid-Attenuated Inversion Recovery Signal Foci in the Splenium of a Patient with Presenilin-1 Mutation

A recent article associated discrete regions of abnormal T2 prolongation in the splenium of the corpus callosum with aging and radiation therapy (1). We recently encountered a patient with autosomal dominant, early-onset Alzheimer disease (AD) due to presenilin-1 (PS-1) mutation with an identical callosal appearance.

The patient's cognitive decline began at 39 years of age. Within a year, her job performance deteriorated, forcing employers to assign her progressively less challenging duties before termination. Her parents reported no family history of neurologic or psychiatric disorders. Initial evaluation in 2001 found moderate global cognitive impairment on the Mini–Mental State Examination (MMSE 16/30). Routine studies, including brain MR imaging and electroencephalography, were reported as normal or nondiagnostic. Subsequent evaluation at

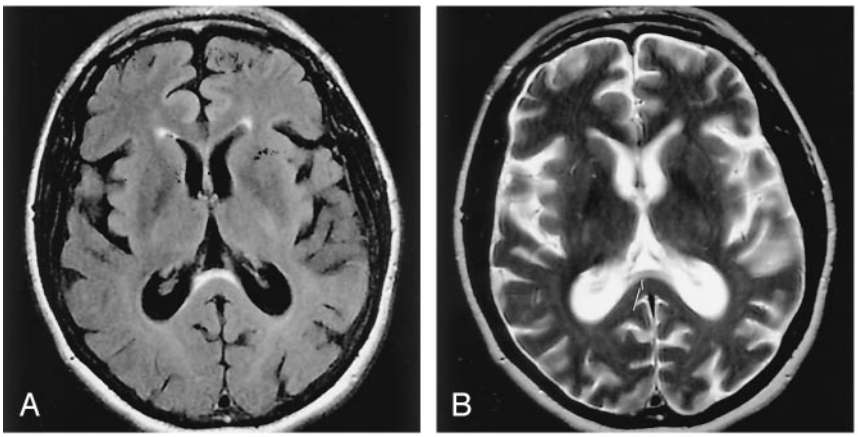

FIG 1. Suppression of ventricular fluid signal by using the FLAIR sequence unmasked the abnormality hidden by the conventional T2-weighted sequence.

A, Axial FLAIR image, revealing a discrete thin band of signal intensity hyperintensity in the anterior subependymal region of the splenium.

B, T2-weighted image, showing that CSF signal intensity obscured assessment of the anterior splenium (*arrow*).

45 years of age found the patient completely dependent on her parents for basic self-care activities. Physical examination revealed irregular, generalized myoclonic movements and unsteady gait. Cognitive testing showed severe impairment (MMSE 3/30). Blood tests detected a point mutation (M146 L) in the PS-1 gene that has been previously reported in association with early-onset AD.

Brain MR imaging (1.5T) at age 45 revealed diffuse cerebral and cerebellar cortical atrophy. Axial fluid-attenuated inversion recovery (FLAIR) images revealed a discrete thin band of hyperintense signal in the anterior subependymal region of the splenium (Fig 1A). Corresponding views on T2-weighted images showed that CSF signal intensity obscured assessment of the anterior splenium (Fig 1B). Axial FLAIR images also showed two thin, paired linear foci of increased signal intensity in the vicinity of the medial lemniscus in the pons, as well as a narrow (<5 mm) rim of hyperintensity around the margins of the superior aspect of the lateral ventricles. More superior FLAIR sections failed to demonstrate definite reduced signal intensity in the motor cortex, a finding anecdotally associated with AD at 3T. Other white matter structures were normal on all additional sequences. No sagittal FLAIR images were obtained. There were no areas of abnormal restriction on diffusion-weighted images or abnormal enhancement after intravenous administration of gadolinium-diethylenetriamine pentaacetic acid contrast medium.

Pekala et al (1) reported that focal signal intensity abnormalities in the splenium were common in otherwise normal older patients and in those who have undergone brain radiation therapy. In both groups, splenium hyperintensity correlated with leukoaraiosis elsewhere. The authors noted that FLAIR sequences allowed detection of juxtaventricular lesions that were obscured by adjacent high signal intensity from the ventricles on T2-weighted images. Observations from the present case support their hypothesis: volume-averaged ventricular fluid caused high signal intensity on conventional T2-weighted images that eclipsed the distribution of hyperintensity in the anterior splenium. The authors concluded that splenium lesions do not necessarily indicate diseases such as glioma or multiple sclerosis and admonished radiologists to be aware of this common incidental finding.

On the other hand, our patient, a young woman without history of radiation therapy, showed an identical callosal appearance. Although available radiographic descriptions indicate that some patients with early-onset AD due to PS-1 mutations show distinctive white matter abnormalities, abnormal callosal signal intensity has not been reported (2, 3). In retrospect, the images depicted in Aoki et al (2) may merely represent prominent perivascular spaces in the posterior white matter. Unfortunately, they only displayed T1- and T2-weighted images, not FLAIR or proton density–weighted studies. Accordingly, the paucity of abnormal white matter signal intensity closely resembles that in our patient. These previous reports attributed leukoaraiosis to ischemia secondary to vascular amyloid deposition. The lack of other typical deep white matter signal intensity changes, coupled with the uncommon incidence of ischemic injury in the posterior corpus callosum, make this mechanism less tenable for the case described here. Another explanation is that FLAIR hyperintensity in the anterior subependymal splenium merely represents the same benign process responsible for causing the thin rim of hyperintensity around the lateral ventricles that is commonly observed with MR imaging, especially in older patients, as Pekala et al (1) demonstrated in Figure 1. Alternatively, gliosis in this region may plausibly result from focally severe axonal attrition from parieto-occipital or posterior cingulate zones linked via the splenium. This conjecture suggests that, beside cases representing "incidental" findings, abnormal FLAIR signal intensity might be detectable in other degenerative conditions with a similar distribution of neocortical injury (e.g., prion disease, posterior cortical atrophy) (4).

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Enlarging Vertebral Pneumatocysts in the Cervical Spine

We read with interest the report by Kitagawa et al about enlarging vertebral pneumatocysts in the cervical spine (1). It would seem to have been appropriate for these authors to cite

our earlier report about vertebral pneumatocysts (2), because it is the only report indicating the prevalence of vertebral pneumatocysts and contains the largest series of such patients. Without citing our report, the authors' report seems to have some major handicaps. For example, including our patients, there are 22 cases of intravertebral pneumatocysts, instead of 12 cases. Also with reviewing features of our cases, authors could have given more appropriate documentation of reported cases in the English-language literature.

The authors cited the Hall and Turkel study, but they missed the fact that Hall and Turkel also mentioned subsequent enlargement of a pneumatocyst in the ilium by CT (3). So Kitagawa et al's case is not the first enlarging pneumatocyst reported in the literature, but it is the first enlarging cervical vertebral one.

Also, writing and publishing scientific papers is facilitated by the wide accessibility of such electronic search tools as Pubmed and Medline, through which major journals are easily accessible. Statements carrying the disclaimer "to our knowledge," in our opinion, should be used only when full and careful literature searches have been achieved. Nevertheless, we congratulate the authors on their well-written and superbly illustrated report.

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Reply

We appreciate Drs. Arslan and Cubuk's letter regarding inappropriate references in our report.

We also apologize to Dr. Arslan and colleagues for our not citing their previous comprehensive report regarding vertebral pneumatocysts.

As they noted, because multisection CT is widely used in clinical settings, we realize that vertebral pneumatocysts exist more frequently than previously reported, especially in severely degenerated spine. Nevertheless, the course of vertebral pneumatocysts is still unclear, as is their relationship to vertebral cystic lesions. We have still to follow the patients in our report and have observed other patients with pneumatocysts by using multisection CT. We hope we can report on this topic in the foreseeable future.