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Ultrasonography of Carotidynia

Christian Arning

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MR Imaging Findings of Cortical Blindness Following Cerebral Angiography: Is This Entity Related to Posterior Reversible Leukoencephalopathy?

We read with interest Saigal et al's (1) article, *MR Findings of Cortical Blindness Following Cerebral Angiography*, in the February 2004 issue of the *AJNR.* The authors reported MR imaging findings in three cases of cortical blindness following cerebral angiography in which nonionic contrast media were used. These cases exhibited clinical and radiological findings that were relatively similar to those associated with posterior reversible leukoencephalopathy (PRLE), which suggests a common pathophysiology. On the basis of their experience, Saigal et al have hypothesized that the pathophysiological mechanisms of cortical blindness following cerebral angiography and PRLE may be related.

Posterior reversible encephalopathy syndrome, hypertensive encephalopathy, reversible posterior cerebral edema syndrome, and PRLE are all terms that have been used to describe a group of disorders that present clinically with headache, seizures, visual changes, altered mental status, and occasionally, focal neurologic signs (1). CT and MR imaging of patients with these disorders typically show symmetrically distributed areas of vasogenic edema predominantly within the territories of the posterior circulation (2–5). The abnormalities affect primarily the white matter, but the cortex is also involved. Localized mass effect and subtle enhancement within the lesions have been described, but are not seen consistently.

Endothelial cell damage is believed to be the central pathophysiology of posterior reversible leukoencephalopathy syndrome (2–5). Although reversible vasogenic edema due to cerebrovascular autoregulatory dysfunction is the underlying pathophysiological mechanism, irreversible lesions resulting from cytotoxic edema can be found, especially in patients with seizure (2–5). Diffusion-weighted (DW) imaging is useful to distinguish between reversible vasogenic edema and cytotoxic edema resulting in ischemic injury. It stands to reason that DW imaging could be used to distinguish PRLE from other disease entities or to monitor for ischemia as a complication of PRLE. DW imaging studies of PRLE have showed that apparent diffusion coefficient (ADC) values in areas of abnormal T2 signal intensity were high (2–5). In addition, low ADC values compatible with cytotoxic edema may be found, especially in severe irreversible lesions resulting from cytotoxic edema in patients with seizure (2–5).

We have reviewed all of the images presented by Saigal et al (1) and found no abnormalities on the DW images or the ADC maps. In their article, the authors state the following: "The absence of evidence of restricted fluid motion on the diffusion-weighted images in all cases seems to be an important finding" (Vol. 25, No. 2, p. 225). The authors did not address the issue of ADC values in the case report section.

As further evidence of the correlation between the pathophysiology affecting the patients in their series and that of PRLE, Saigal et al have noted the gyriform hyperintensities in the occipital cortices that appear on all fluid-attenuated inversion recovery and T2-weighted MR images in their series. Although PRLE is associated with cortical involvement, its effects are normally confined to subcortical white matter. Cortical involvement without subcortical white matter involvement is not normally associated with PRLE.

Because the ADC map and DW imaging findings and the cortical involvement patterns of cases presented by Saigal et al (1) are dissimilar to those associated with PRLE, we believe that the pathophysiological mechanisms of cortical blindness following cerebral angiography and PRLE are probably *not* related.

The true biochemical mechanisms of cerebral injury remains speculative in patients with cortical blindness following cerebral angiography. The incidence of transient cortical blindness is reported to range from 0.3–1% when nonionic contrast agents are used, but it can be as high as 4% when hyperosmolar iodinated contrast agents are used (6). Transient cortical blindness was reported in cerebral, vertebral, brachial, aortic arch, renal, and coronary angiography, translumbal aortography, and myelography (6). The highest incidence was reported following vertebral angiography (6). The highest incidence of cortical blindness following vertebral angiography and the higher risk of cortical blindness with nonionic contrast agent use makes us think that the possible explanation is a direct neurotoxicity of the contrast agent itself to sensitive occipital cerebral lobes.

Sait Albayram
Harun Ozer
Department of Radiology, Division of Neuroradiology
Cerrahpasa Medical School
Istanbul, Turkey

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Reply:

We appreciate Dr. Albayram's and Dr. Ozer's interest in our article on posterior reversible leukoencephalopathy (PRLE). The authors are correct in their comments about the findings of PRLE (paragraph 2 of their letter), which we did not elaborate on in our article. We did not comment on the apparent diffusion coefficient (ADC) values in this regard. ADC values are increased in cases of PRLE because of vasogenic edema. Cytotoxic edema may occur in some patients, in which case low ADC values would be seen.

We must, however, disagree with the comment about the biochemical mechanisms being speculative in patients with cortical blindness following cerebral angiography. Both experimental and clinical studies (1–5) have shown that contrast media (both ionic and nonionic) cause a disruption of the blood-brain barrier (BBB), with resultant leakage of the contrast media into the adjacent brain. It is this pathophysiological mechanism that explains the CT hyperattenuation seen in the occipital lobes following injection of contrast medium (5, 6). Disruption of the BBB leads to the vasogenic edema, which may progress to cytotoxic edema, as suggested in PRLE (7).

Similarly, disruption of the BBB leads to extravasation of contrast medium, with resultant contrast medium–induced neurotoxicity to the more sensitive occipital lobes. Extravasation of contrast material has been seen in both the cortical and subcortical areas in the occipital lobes (5, 8).

The reason why the posterior circulation is more commonly affected has also been extensively studied. Many theories exist, but the commonly accepted one is the paucity of sympathetic innervation of the vertebrobasilar system when compared with that of the internal carotid artery system (6, 7). That would also explain the higher incidence of cortical blindness in vertebral angiography, as we suggest.

The exact mechanism of neurotoxicity of contrast agents once they gain access to the brain parenchyma is open to speculation. It is thought that the toxicity may be related to the ability of the contrast agent to affect the membranes of the neurons or glia or idiosyncratic reaction, hypoxia, or edema (9). Hypertension has clearly been shown to be a factor that increases susceptibility of the endothelial cells to damage, and thus, increases the risk of both cortical blindness following angiography and PRLE (9, 10).

With regard to the imaging findings, we agree with the comment that ADC values are increased in cases of PRLE because of vasogenic edema. In addition, cytotoxic edema may occur in some patients, in which case low ADC values are seen. On a retrospective review of the cases we presented, the ADC values were slightly increased in two (cases 2 and 3). Unfortunately, because of the loss of data, we could not measure the ADC values in case 3. Also, one of the cases showed involvement of the subcortical white matter (case 1), and there was questionable involvement of the subcortical white matter in another (case 2). In case 3, however, only cortical involvement was noted. It is possible to have exclusive cortical involvement with pseudonormalization of the ADC values in PRLE, as suggested by Covarubbias et al (11). Also, there have been only two reports of exclusive cortical involvement in hypertensive encephalopathy syndromes (12, 13).

A relationship between these two entities has also been suggested by other authors on the basis of a similar pathophysiological mechanism (6, 10, 14).

Gaurav Saigal
Rita Bhatia
Sanjiv Bhatia
Ajay Wakhloo
Department of Radiology
University of Miami
Miami, FL

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An Explanation for Putaminal CT, MR, and Diffusion Abnormalities Secondary to Nonketotic Hyperglycemia

Wintermark et al (1) recently reported findings of high signal intensity on T1-weighted MR images and faint hyperattenuation of involved putamen on CT scans one month later. In addition, the putamen appeared hyperintense on the exponential diffusion-weighted (DW) images. The authors tried to propose an explanation by assuming that protein desiccation occurring in the course of Wallerian degeneration could explain the CT hyperattenuation and the MR imaging and DW imaging patterns in the early phase. I think this explanation is inadequate.

First, I had previously reported 10 cases of hemichoreahemiballism with similar CT and MR imaging findings and collected 13 more cases to date (2). A key finding in my article was that there was a mismatch between the size of lesions detected on CT scans and on T1-weighted MR images, and also a mismatch between their evolutions over time. It appeared that the lesions on CT and on T1-weighted MR images resulted from two different pathophysiological mechanisms running in parallel. It is not appropriate to make an attempt to give one explanation for two pathologic processes, although they may be triggered by the same event.

Second, it is now clear that the high signal intensity lesions on T1-weighted MR images are related to manganese accumulation in the reactive astrocytes after ischemia. In their article published in 1999, Fujioka et al (3) reproduced the MR imaging finding in rats 7 days after 15-minute occlusion of the middle cerebral artery but not after 60-minute occlusion. Histologic examination revealed that this specific ischemic change disclosed by MR imaging corresponded to selective neuronal death and gliosis, with preservation of the macroscopic structure of the brain, a finding similar to what I reported after histologic analysis of a biopsy specimen obtained from my patient. We both agreed that the MR imaging finding resulted from a progressive pathologic reaction in an incomplete infarction, which confirmed my hypothesis proposed in the 1998 article; that is, the MR imaging finding was related more to vascular compromise than to petechial hemorrhage or hyperglycemia (4). In their latest paper, Fujioka et al demonstrated a similar time course of the appearance of high signal intensity on T1-weighted MR images and the accumulation of tissue manganese accompanied by Mn-superoxide dismutase and glutamine synthetase induction in reactive astrocytes (3).

Third, the restricted diffusion on the DW images reported by Wintermark et al provided additional evidence that ischemia did occur early in the disease course. It is not required to discard such a good explanation for the purpose of keeping one explanation for both findings.

Fourth, while the mechanism responsible for the hyperattenuation on CT scans remains inconclusive, protein desiccation during Wallerian degeneration appears not to be the best explanation. Protein breakdown usually occurs from 4 to 14 weeks after injury, whereas the hyperintense lesions on CT scans in many of these patients occur early in the disease course. To the best of my knowledge, manganese deposition in the brain is not associated with hyperattenuation on CT scans. Microbleeds or "reversible calcium deposition or influx" remains a possible explanation.

Din-E Shan
Neurological Institute, Taipei Veterans General Hospital
National Yang-Ming University School of Medicine
Taipei, Taiwan

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Reply

We appreciate Dr. Shan for his interesting comments regarding our hypothesis that the observed CT and MR imaging findings in a patient with nonketotic hyperglycemia resulted from an initial hyperglycemic and/or hyperosmolar putaminal insult inducing in turn some degree of Wallerian degeneration of the internal white matter of the putamen.

Dr. Shan states that an attempt to give a single explanation for different pathologic processes is inadequate and raises several points that we would like to address. Relying on a recent reference (1), he suggests that the observed T1 hyperintensity results from manganese accumulation following incomplete brain infarction. He also suggests that the observed restriction of diffusion is a direct consequence of brain ischemia. Finally, Dr. Shan explains the CT hyperattenuation by microbleeds or a "reversible calcium deposit or influx."

Let us examine these possibilities one by one.

Dr. Shan's explanation of the CT hyperattenuation by microbleeds or a "reversible calcium deposit or influx" is contradicted by the absence of susceptibility effect on gradient-echo images, as observed in our patient and also as reported in previous articles (2). Furthermore, blood or calcification does not account for restricted diffusion.

Regarding the DW image abnormality, the persistence of restricted diffusion reported in nonketotic hyperglycemia, is different from that typically observed in cases of brain ischemia and suggests a different pathophysiological mechanism (2).

Finally, the accumulation of manganese, which results from an increase in the activity of an enzyme called manganese superoxide-dismutase, is not specific to brain ischemia (1) and can be triggered by multiple stimuli, including hyperglycemia (3, 4), as in the case of our patient.

Hence, the alternative explanation suggested by Dr. Shan for the imaging findings in the case of nonketotic hyperglycemia is not entirely convincing. Our hypothesis of an initial hyperglycemic and/or hyperosmolar putaminal insult, followed by some degree of Wallerian degeneration, has the advantage of proposing one single scientific explanation for the different CT and MR imaging findings. We agree that this has to be evaluated in a study involving longitudinal patient follow-up, which unfortunately could not be obtained in our patient. Considering our hypothesis, diffusion tensor imaging might represent a key tool for better understanding the constellation of brain imaging abnormalities in nonketotic hyperglycemia.

Max Wintermark
Nancy J. Fischbein
Pratik Mukherjee
Esther L. Yuh
William P. Dillon
Department of Radiology, Neuroradiology Section
University of California, San Francisco

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Absence of Memory Dysfunction after Bilateral Mammillary Body and Mammillothalamic Tract Electrode Implantation: Preliminary Experience in Three Patients

The anatomic bases for memory disorders have been widely debated. Lesions of the medial dorsal (MD) and anterior nuclei (AN) of the thalami and lesions of the mammillary bodies (MB) are most commonly involved in amnesic syndromes in humans (1, 2).

In a monkey model, Mishkin (3) conceptualized the existence of a double limbic circuitry supporting the memory function (i.e., the medial limbic [hippocampal] and the basolateral limbic [amygdaloid] circuits). In this functional scheme, the AN and the MD thalamic nuclei act as "nodal" points of convergence where bilateral tissue damage can result in memory function impairment, similar to bilateral lesions of the mammillothalamic tract (MTT), which connects the MB to the AN.

In an article in the June/July issue of the *AJNR,* Yoneoka et al (4) reported on the onset of a Korsakoff syndrome in a patient with acute ischemic damage of the left MTT assessed by positive diffusion-weighted imaging who had previously suffered from a contralateral right MTT mirror infarction. The authors suggested that bilateral MTT dysfunction may be sufficient to cause severe amnesic state, which matched Mishkin's hypothesis. Berger highlighted this in an editorial of the same issue of the *AJNR,* stating that the article added one more site of the hippocampal-limbic system—namely, the MTT—from which bilateral lesion may result in amnesia (5).

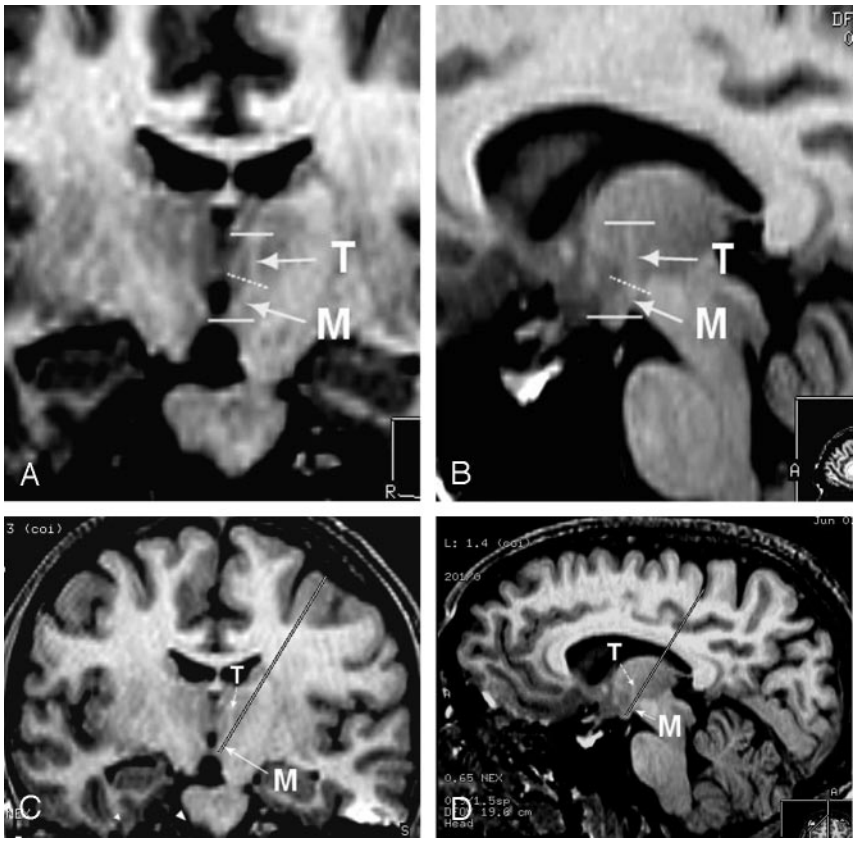

FIG 1. Anatomic study on healthy volunteers (reformatted images from unenhanced 3D T1-weighted SPGR acquisition).

A, Coronal oblique reformat (magnified view) clearly depicts the two segments of the MTT on both sides. The presence of a proximal "mammillary" (M) and of a distal "thalamic" (T) segment separated by an angulation (*dotted line*) is clearly shown.

B, Sagittal oblique reformat also shows the two segments of the left MTT in an orthogonal plane relative to that in the previous illustration.

C, Electrode trajectory simulation on frontal oblique reformat (similar but demagnified view as 1A) shows "catheterization" of the mammillary segment of the MTT and safe cortical entry point located in the posterior third of the middle frontal gyrus.

D, Electrode trajectory simulation on sagittal oblique reformat (similar but demagnified view as in 1B) showing "catherization" of the proximal MTT, but critical central sulcus entry point.

The neurosurgical team at our institution has initiated a clinical trial aimed at treating patients with chronic refractory epilepsy by bilateral stereotactic implantation of stimulation electrodes (DBS lead 3389, Medtronic, Minneapolis, MN) within the MB through the longest part as possible of the proximal segment of the MTT (6). We therefore performed a preliminary *in vivo* anatomic study and derived biometric measurements of the MTT by 3D processing of unenhanced 3D T1-weighted spoiled gradients data in a normative cohort of nine healthy volunteers without structural abnormalities of the brain (7).

In light of the functional directionality of the MTT projections from the MB to the AN, we clearly delineated 1) a proximal segment, named the "mammillary" segment, which has posterior-cranial orientation in the sagittal plane and lateral-cranial one in the coronal plane, and 2) a distal "thalamic" segment, which has frontal and caudal-cranial orientation (Fig 1A, B). An empirical method using the 3D capabilities of the Advantage Windows, release 4.0, software (GEMS, Milwaukee, WI) running on an off-line Ultra 60 Creator 3D station (Sun Microsystems, Santa Clara, CA) was designed to define the cortical entry point combining targeting of the MB epicenter together with so-called catheterization of the longest possible part of the mammillary segment of the MTT. We obtained the entry point by superimposing the straight line connecting the MB to cortical surface on the longitudinal axis of the mammillary segment of the MTT. It was located in the posterior third of the middle frontal gyrus in the coronal plane (Fig 1C) and in the central sulcus or in the precentral gyrus when drawing the line in the sagittal plane (Fig 1D). The first option was chosen to avoid primary functional areas, and the value of this empirical approach was assessed postoperatively by coregistrating the pre- and postoperative images in the similar juxta/supra-MB section location in all the three patients being recruited up to now (Fig 2). A passage through the most proximal segment of the MTT—at least partially—was demonstrated for the six electrodes, which have four stimulation contacts, the distal one (numbered 0) being located within the MB, and the three proximal ones (numbered 1–3) within the MTT.

None of the three patients experienced any memory deficit, neither immediately after surgical implantation, nor during global or elective stimulations (left side versus right side versus both sides; 0, 1, 2, or 3 only, versus any combination of the four stimulation contacts), which were performed under close neuropsychological monitoring. Additional comprehensive cognitive tests were repeatedly performed, all of which failed to reveal any early or delayed mental decline after implantation.

These data demonstrate the absence of significant memory dysfunction induced by bilateral MB/MTT implantation and electrical stimulation. They agree with the recent experimental work by Vann and Aggleton (8) on a rat model, and with Harding et al (9) on alcoholics with and those without Korsakoff psychosis, who demonstrated that AN degeneration or lesions by far more critically impaired memory than MB or MTT lesions.

The disconnection of the AN from its MB projections through the MTT (the so-called Delay and Brion connection) does not impair the direct fornical route from the hippocampus to the AN. This alternate pathway may explain why MB or MTT lesions, even if bilateral, are not as disruptive as AN lesions. Korsakoff syndrome in the patient reported by Yoneoka et al (4) could have been triggered by bilateral ischemic damage not limited to the MTTs. The newly infarcted left area and the old right lesion seem to extend far beyond the MTTs to involve the anterior thalamic nuclei (Figs 1A–D and 2A, B, p. 965; and Figs 4A, B, p. 967 [AJNR; Vol 25 No 6]). In accordance with the theoretical statement by Vann and Aggleton in their recent review on the topic (10), we think the presence of

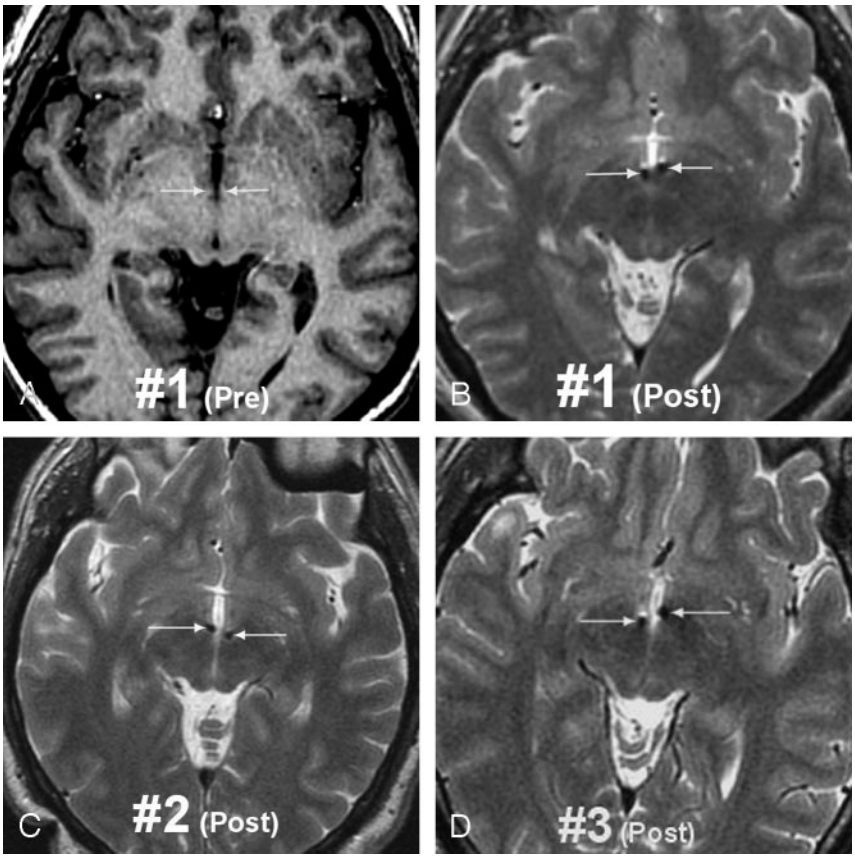

FIG 2. MR images in the three bilaterally MB implanted patients

A, Preoperative status in patient 1 (T1-weighted 3D SPGR image) showing the mammillary segment of the MTT on both sides (*arrows*).

B, Postoperative image (T2-weighted 3D fast spin-echo [FSE] image) in patient 1 showing the electrodes through the mammillary segment (*arrows*). FSE T2 weighting was preferred to minimize susceptibility artifacts due to ferromagnetic components of the electrodes.

C and *D,* Postoperative status in patients 2 and 3 (T2-weighted 3D FSE images) also showing "catherization" of the mammillary segment of the MTTs in both cases.

concurrent anterior thalamic disease to have outweighed the MTT damage in that patient.

Thierry P. Duprez
Basel Abu Serieh
Christian Raftopoulos
Departments of Radiology and Medical Imaging, and Neurosurgery
Université Catholique de Louvain
Louvain-en-Woluwé, Brussels, Belgium

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Reply:

We thank Duprez et al for their attention and valuable comments concerning our article "Acute Korsakoff Syndrome Following Mammillothalamic Tract Infarction" (1) and also for presentation of their advanced work in neuroradiology and neuromodulation (2, 3). Their excellent depictions of the mammillothalamic tract (MTT) and implanted neuromodulation electrodes with MR imaging allow us the opportunity to further discuss amnesia-initiating lesion(s) in our case.

Korsakoff syndrome is defined as a disproportionate impairment in memory, relative to other aspects of cognitive function, resulting from a nutritional (thiamine) depletion (4). In our case, however, the patient developed acute amnesia with features of Korsakoff syndrome that were not attributable to malnutrition but were the result of a left MTT infarction. MR imaging studies revealed acute ischemic damage of the left MTT assessed by diffusion-weighted imaging and a previous contralateral right MTT infarct on T2-weighted and fluid-attenuated inversion recovery images, which caused prolonged global amnesia manifesting as Korsakoff syndrome. Thus, we reported this rare case as an acute "Korsakoff syndrome" following MTT infarction. At clinical follow-up, he continued to suffer from the severe amnesic syndrome, 8 months after the onset of symptoms.

As Duprez et al suggested, we cannot completely rule out the anterior thalamic nucleus disease, at least on the left side, because the anterior thalamic nucleus (AN) lies in a similar vascular territory to the ipsilateral MTT (5–7). T2-weighted images (Figs 1B, 2B, p. 965 [AJNR; Vol 25 No 6]) may suggest the possibility of the left AN ischemic damage (1). It is unclear whether the right AN was affected or not on MR imaging (Figs 1, 2, p. 965; Fig 3, p. 966; and Fig 4, p. 967 [AJNR; Vol 25 No 6]) (1). We think, however, the MTT infarcts spared at least some of the left AN and most of the right AN because MR images examined 8 weeks after onset showed no significant changes of the ependymal contour around the foramen of Monro and there were unchanged AN volumes even in the left side (Fig 4, p. 967 [AJNR; Vol 25 No 6]) (1).

Concerning the crucial lesion(s) in our case of amnesic syndrome reported as acute "Korsakoff syndrome," there are two main candidates: 1) bilateral MTTs or 2) bilateral MTTs and AN(s). Clarke et al (8) reported a case of amnesia after unilateral left anterior thalamic infarction in which unilateral left thalamic infarction caused pure amnesia with slight frontal type dysfunction and thalamic aphasia. In this amnesic case of unilateral small ischemic lesion of the left thalamus, the patient's memory improved partially within 8 months. If perchance the left AN was affected by the left MTT infarction, there could be some improvement with resolution of the ischemic lesion during 8 months of follow-up. Although a small ischemic lesion caused prolonged global amnesia without aphasia in our case, the patient's amnesic symptoms hardly improved during the follow-up. MR images examined 8 weeks after onset demonstrated a right MTT lesion and a left MTT lesion, possibly involving some of the AN. For these reasons, we concluded that both MTT lesions cut off the hippocampal input through the mammillary bodies (MBs), into the AN, thereby producing a severe amnesic state.

T2-weighted images presented by Duprez et al show the remarkable accuracy of stereotactic procedures of deep-brain stimulation (DBS) as related to the MB/MTTs. There exist four general hypotheses to explain the therapeutic mechanism(s) of DBS: (1) stimulation-induced alterations in the activation of voltage-gated currents that block neuronal output near the stimulating electrode (depolarization blockade); (2) indirect inhibition of neuronal output by means of activation of axon terminals that make synaptic connections with neurons near the stimulating electrode (synaptic inhibition); (3) synaptic transmission failure of the efferent output of stimulated neurons as a result of transmitter depletion (synaptic depression); (4) stimulation-induced disruption of pathologic network activity (9). In view of these four DBS mechanisms, we have found it difficult to understand the reason(s) why their three patients never show any neuropsychiatric deficits when their DBS (2) affected MB/MTTs bilaterally.

We consider separately the following three propositions: (1) concurrent AN disease outweighed both MTT damage in our case, (2) no MB/MTT, but an AN lesion is crucial for alcoholic Korsakoff amnesia, and (3) bilateral MB/MTT functionally lessening modulation never causes neuropsychiatic deficits including amnesia: In other words, bilateral MTT dysfunction can spare memory. We have discussed the first proposition above. Concerning the second point, bilateral MTT lesions can cause amnesia closely resembling Korsakoff syndrome. For the third point, the evidence that an AN degeneration or lesion more critically impairs memory than MB/MTT lesions in Korsakoff psychosis (10) does not mean that bilateral MTT lesions do not affect memory. Further experiences of MB/MTT-DBS will clarify the role of the MB/MTT(s) in memory. On the assumption that the MB/MTTs play an important part in memory, it is difficult to explain why bilateral MB/MTT-DBSs never cause any neuropsychiatric deficits. Perhaps MB/MTT-alternative pathway(s) for memory exists in their three patients with chronic refractory epilepsy.

In any event, we are attentive to further studies of bilateral MB/MTT-DBS lead implantation by Duprez's colleagues (2). Their future work will advance the frontiers of amnesia research as more experience is obtained.

Y. Yoneoka
N. Takeda
A. Inoue
Y. Ibuchi
T. Kumagai
T. Sugai
K. Takeda
K. Ueda
Departments of Neurosurgery and Emergency Medicine
Yamagata Prefecture Central Hospital
Yamagata, Japan

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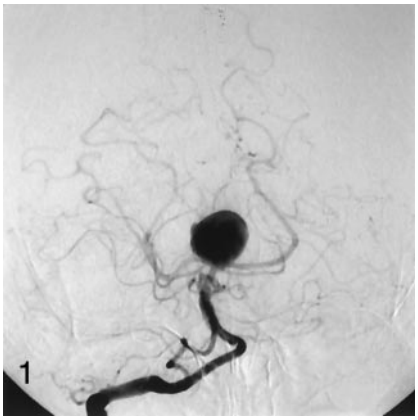
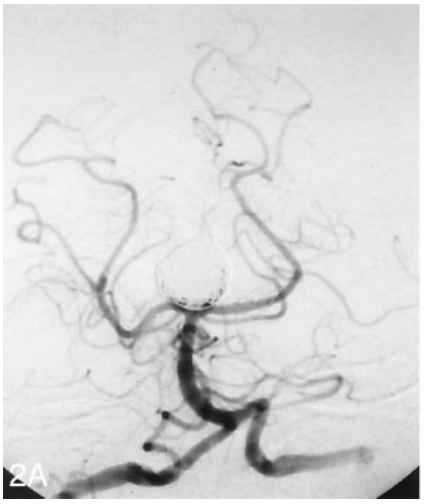
Desperate Appliance

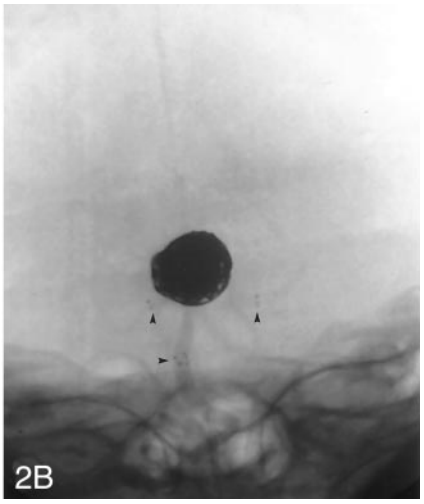
We read with interest the recent case report by Michael Chow and the Cleveland Clinic group regarding treatment of a wide-necked basilar bifurcation aneurysm by using the Y-configuration double-stent technique (1). This report and others (2) show the enormous potential of flexible intracranial stents for therapy of complex cerebral aneurysms. As stated by the authors, however, limitations of the technique exist (3), and more will be discovered. We relate one such cautionary tale.

A 62-year-old woman was referred to our institution for treatment of a large, wide-necked basilar bifurcation aneurysm (Fig 1). Both P1 segments of the posterior cerebral arteries were incorporated in the neck. Our knowledge of the Cleveland experience and the anticipated difficulties with conventional endovascular therapies for aneurysms in this location led us to proceed with the double-stent assisted coiling approach.

This was achieved by using two 3.5 × 20 mm Neuroform (Boston Scientific, Natick, MA) stents and 386 cm of GDC







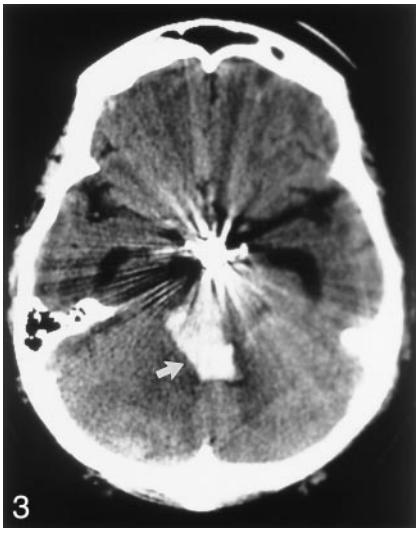


FIG 1. Right vertebral arteriogram (anteroposterior [AP]), shows a wide-necked basilar bifurcation aneurysm.

FIG 2. Postoperative arteriograms.

A, Right vertebral arteriogram (AP), obtained after treatment with stents and coils, shows near complete obliteration of the aneurysm.

B, Unsubtracted image obtained from the postoperative arteriogram shows the radiopaque markers at the ends of the stents (*arrowheads*).

FIG 3. CT head scan obtained 6 hours after treatment shows the pontine component of the acute brain stem hemorrhage (*arrow*).

(Boston Scientific) coils in a fashion similar to the method described by Chow et al (Figs 2). The procedure was performed under general anesthesia and full heparinization, with the standard pretreatment for Neuroform cases including aspirin and clopidigrel. There was near-complete obliteration of the aneurysm with preservation of both posterior cerebral arteries and no evidence of perforation, vessel dissection, or flow-limiting stenosis. The patient awoke from anesthesia with no focal neurologic deficit; however, approximately 6 hours later, she became progressively obtunded with left-sided weakness. A CT head scan showed a midbrain and upper pontine parenchymal hemorrhage (Fig 3), and cerebral angiography showed no change from the postprocedural study. The patient had a limited neurologic recovery but eventually succumbed to chronic hydrocephalus and pneumonia.

A subsequent review of the procedure showed considerable stretching of the right posterior cerebral artery during guidewire placement and positioning of the first Neuroform stent. There were no other technical problems encountered during the procedure. The postmortem neuropathologic examination showed no bleeding from the aneurysm (Fig 4) and no gross obstruction of pontine perforating vessels by the stents. There was a large right midbrain hemorrhage (Fig 5), with smaller hemorrhages in the right thalamus and right cerebellar hemisphere.

Unusual foreign body granulomas were also seen microscopically (Fig 6) associated with the hemorrhages, possibly due to microembolism from the hardware. Although the cause of the midbrain hemorrhage is unknown, it may be due to stretching of perforating vessels, infarction, and bleeding related to anticoagulation.

Despite an excellent angiographic result, this tragic clinical-outcome demonstrates the risks inherent in novel, aggressive interventional strategies. This, however, should come as no surprise. As William Shakespeare noted long ago,

> Diseases desperate grown
> By desperate appliance are reliev'd
> Or not at all (4)

David M. Pelz
Stephen P. Lownie
Max Kole
David Ramsay
Departments of Clinical Neurological Sciences (D.M.P., S.P.L., M.K.), Diagnostic Radiology (D.M.P.), Nuclear Medicine (D.M.P.), and Pathology (Neuropathology) (D.R.)
University of Western Ontario
London, Ontario, Canada

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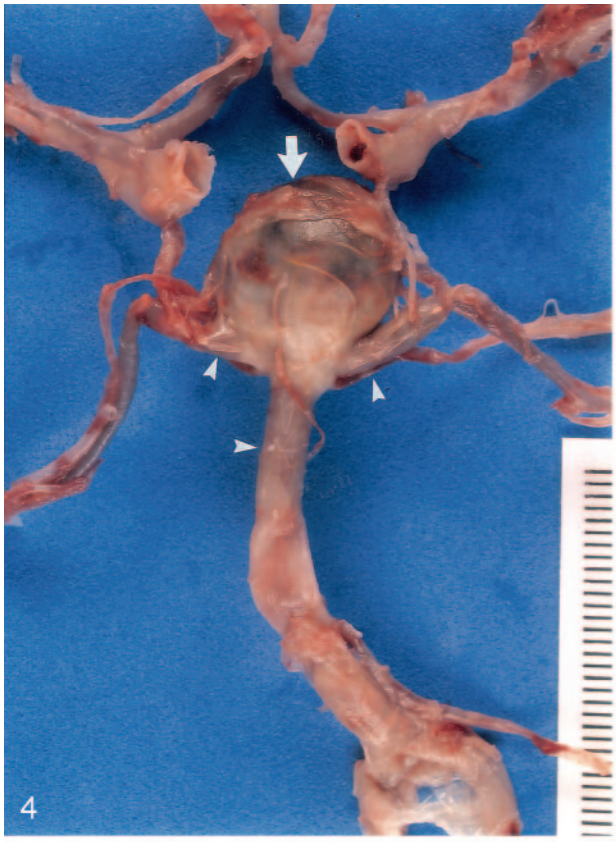

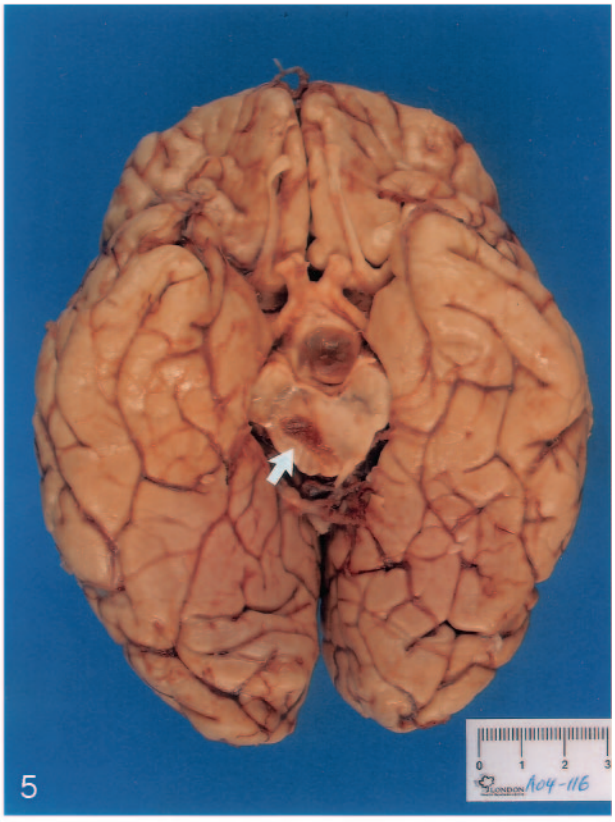

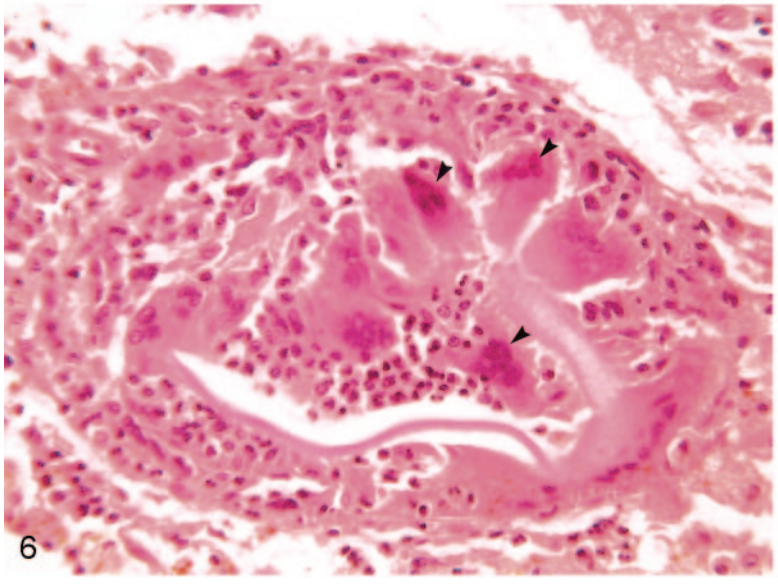

FIG 4. Postmortem dissection of the circle of Willis, showing the coils inside an intact basilar bifurcation aneurysm (*arrow*). The stents are visible through the basilar and posterior cerebral arteries (*arrowheads*).

FIG 5. Gross specimen. Section through the midbrain shows the hematoma (*arrow*)

FIG 6. Microscopic section through the midbrain hemorrhage shows unusual intravascular foreign body granulomas (*arrowheads*) (H&E, ×8).

Rupture of a Cerebral Aneurysm during MR Imaging: Leakage of Gadolinium into the Subarachnoid Space

Dear Editor: We wish to report a recent, notable case and present the remarkable images we obtained from it. A 62-year-old woman was undergoing routine MR imaging to investigate the cause of left arm and facial pain. While in the unit and shortly after the administration of gadolinium, she developed a severe headache and vomiting. After initial assessment, the radiologist obtained further MR images of her brain. These revealed extravasation of contrast medium into the subarachnoid space. The patient was taken immediately to the angiography suite, where the presence of a left internal carotid aneurysm was confirmed. This aneurysm was repaired by craniotomy, and the patient made a satisfactory recovery. There are reports in the literature documenting the MR imaging appearance of acute subarachnoid hemorrhage (1); however, our search of the literature yielded no reports of a subarachnoid hemorrhage (SAH) occurring dur-

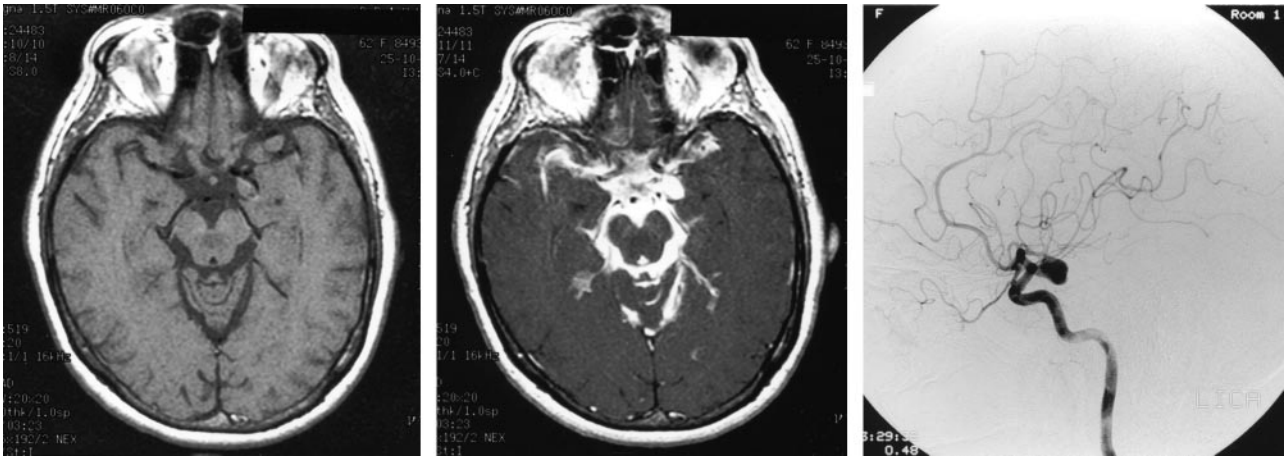

FIG 1. Noncontrast T1-weighted axial MR image of the area of the circle of Willis.
FIG 2. Postgadolinium T1-weighted axial MR image of the area of the circle of Willis.
FIG 3. Left internal carotid digital subtraction angiogram showing left internal carotid artery aneurysm.

ing MR imaging. This report illustrates the unique MR imaging appearances of gadolinium leakage into the subarachnoid space after development of an SAH.

Rodney S. Allan,
Westmead Hospital, Neurosurgery, Sydney, NSW, Australia
Royal Prince Alfred Hospital, Neurosurgery, Sydney, NSW, Australia
Geoffrey Parker,
Royal Prince Alfred Hospital, Radiology, Sydney, NSW, Australia
Martin McGee-Collett
Royal Prince Alfred Hospital, Neurosurgery, Sydney, NSW, Australia

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Ultrasonography of Carotidynia

Carotidynia is an unilateral neck pain syndrome associated with tenderness to palpitation over the carotid bifurcation, first described by Fay in 1927 (1). We performed ultrasonographic (US) investigation in six consecutive cases that fulfilled clinical criteria of idiopathic carotidynia according to the former International Headache Society classification (2). In all patients, hypoechoic wall thickening of the carotid bulb was found exactly in the region of tenderness, leading to a mild lumen narrowing and a large outward extension of the vessel wall. In some cases, the wall thickening was found in two different layers of the vessel wall (Fig 1A). Follow-up US 3–5 weeks later showed significantly fewer pathologic findings (Fig 1B). In two patients, MR imaging of the carotid bifurcation showed no evidence of intramural hematoma. The findings correspond to recently published MR imaging data that described abnormally enhancing tissue surrounding the symptomatic carotid artery in five cases of carotidynia (3); in one of these patients, MR imaging was repeated after resolution of symptoms and showed normal findings (3). In a recently published case of carotidynia, histologic findings of inflammation of the ca-

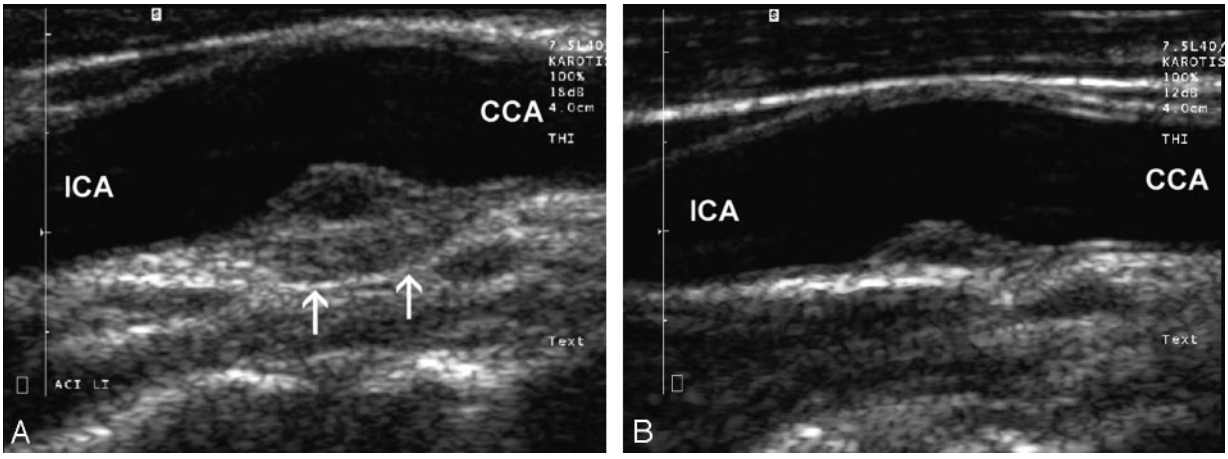

FIG 1. US findings in a case of carotidynia. CCA, common carotid artery. ICA, internal carotid artery.
A, Initial findings of wall thickening, leading to a mild lumen narrowing and a major outward extension of the vessel.
B, Follow-up findings 4 weeks later show significantly fewer pathologic findings.

rotid adventitia were presented (4); however, the cause of the inflammatory process remained obscure.

Our findings of a similar US pattern in six patients suggest that idiopathic carotidynia is a distinct entity, possibly caused by inflammation.

Christian Arning
Department of Neurology
AK Wandsbek
Hamburg, Germany

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