



Providing Choice & Value

Generic CT and MRI Contrast Agents



CONTACT REP

AJNR

**In Re: Arterial Dissection Complicating
Cerebral Angiography and Cerebrovascular
Interventions**

Jiri Vitek

AJNR Am J Neuroradiol 2002, 23 (4) 740-741

<http://www.ajnr.org/content/23/4/740>

This information is current as
of July 29, 2025.

MR Imaging in Comatose Survivors of Cardiac Resuscitation

We read with interest the article by Wijndicks et al (1) on the role of MR imaging as a confirmatory study in survivors of cardiac arrest. The authors, using fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted (DW) imaging, demonstrated diffuse signal intensity abnormalities in the cerebellum, thalamus, frontoparietal cortices, and hypothalamus that were not initially visualized on CT scans. This study and several others stress the important new role of neuroimaging in predicting the neurologic outcome after global ischemia (2–4).

We would like to draw the attention of the authors to a previously published study (3) in which CT findings were used as predictors of the outcome after cardiac arrest. Wijndicks et al report an interesting discrepancy between MR imaging and CT results. This discrepancy supports our previous findings (5) that a loss of gray matter (GM)–white matter (WM) differentiation (GWMD) on CT scans, when visually assessed, is not a reliable predictor of a poor outcome or death after cardiac arrest. We found a poor interrater reliability ($\kappa = 0.3$). Interrater agreement was only 58% with an actual agreement beyond chance of 20%. This finding prompted us to use a more quantitative assessment of the loss of GWMD. Hence, we measured the Hounsfield unit density of GM and WM in several regions of interest on nonenhanced CT scans of the brain. The most important area was at the basal ganglial level (BGL). Using an analysis of the receiver operating characteristic curve, we determined that a Hounsfield unit ratio of GM (caudate) to WM (posterior limb of the internal capsule) of less than 1.18 at the BGL was 100% predictive of death with an odds ratio of death of 21.67 (3).

Although there is no doubt that MR and DW imaging are far more accurate than CT scanning in identifying early ischemic changes, the use of this technique is still limited by several factors, such as the lack of availability and expertise needed to interpret DW images in several centers and the long duration of the test, which may expose critically ill patients to undue risks. Wijndicks et al were able to obtain MR images in only 37% of their patients, with additional advantage of having the anesthesia and MR imaging departments located on the same level as the intensive care unit. This level of support is not readily available in most hospitals.

We believe that, until MR imaging technique overcomes some of these limitations, the quantitative measurement of the loss of GWMD with CT may be a good substitute in several medical centers.

Michel T. Torbey, MD, MPH and Anish Bhardwaj, MD
Neurosciences Critical Care Division
The Johns Hopkins University
School of Medicine
Baltimore, MD

References

1. Wijndicks EF, Campeau NG, Miller GM. **MR imaging in comatose survivors of cardiac resuscitation.** *AJNR Am J Neuroradiol* 2001; 22:1561–1565
2. Arbelaez A, Castillo M, Mukherji SK. **Diffusion-weighted MR imaging of global cerebral anoxia.** *AJNR Am J Neuroradiol* 1999;20: 999–1007
3. Torbey MT, Selim M, Knorr J, Bigelow C, Recht L. **Quantitative analysis of the loss of distinction between gray and white matter in comatose patients after cardiac arrest.** *Stroke* 2000;31:2163–2167
4. Chalela JA, Wolf RL, Maldjian JA, Kasner SE. **MRI identification of early white matter injury in anoxic-ischemic encephalopathy.** *Neurology* 2001;56:481–485
5. Torbey MT, Paydarfar D, Bigelow C, Recht L. **Loss of gray-white matter differentiation (GWMD) predicts poor patient outcome after cardiac arrest.** *Neurology* 1999;52(suppl 12):A61

Management of Aneurysm Perforation during Guglielmi Electrodetachable Coil Placement

We read with interest the case report presented by Willinsky and terBrugge (1) regarding a technique of using a second microcatheter to treat perforation of a ruptured paraophthalmic aneurysm during initial catheterization. Having had experience with a similar situation during endovascular treatment of a cerebral aneurysm, we felt obliged to report on our procedure with some modification and to contribute to the validation of the simple technique to treat a perforation of a cerebral aneurysm.

A 66-year-old man presented with subarachnoid hemorrhage. Angiography showed a $6 \times 5 \times 4$ -mm saccular aneurysm of the basilar-superior cerebellar artery. A Fastracker-10 microcatheter (Boston Scientific/Target) was navigated into the aneurysmal lumen through a guiding catheter that was placed into the left vertebral artery. When we were attempting to push the remaining 10-mm length of a second coil after successful placement of a first coil, the microcatheter and coil suddenly moved forward and perforated the dome, with the distal part of the coil extending beyond the aneurysm (Fig 1A). Angiography performed immediately after the event showed no apparent extravasation of contrast material. The microcatheter and coil were left in place, and the heparin treatment was promptly reversed. An antihypertensive agent was intravenously administered because the patient had a history of transient systemic hypertension, and his blood pressure was maintained at 80 mm Hg. We decided to pack the aneurysm with a Guglielmi detachable coil via a second microcatheter to minimize extravasation at withdrawal of the original microcatheter and Guglielmi detachable coil. With the help of a second microcatheter placed through the contralateral vertebral artery, we succeeded in packing

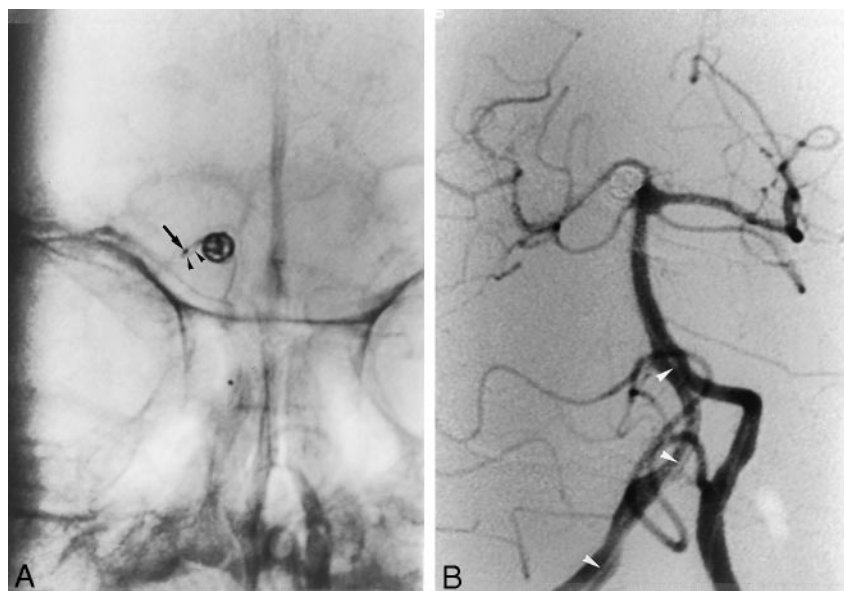


FIG 1. Images show perforation of the dome and obliteration of the lumen.

A, Frontal scout view obtained immediately after aneurysmal perforation, shows the original microcatheter with the second Guglielmi detachable coil (arrowheads) extending beyond the aneurysm. The arrow indicates the tip of the microcatheter.

B, Angiogram of the left vertebral artery, obtained after final embolization through the second microcatheter (arrowheads), shows complete obliteration of the aneurysmal lumen.

the aneurysm without removing the original microcatheter (Fig 1B). Although additional minimal subarachnoid hemorrhage was observed, the patient was discharged in good clinical condition.

Several strategies to manage the life-threatening situation have been suggested (1–5). Prompt recognition of the aneurysmal perforation by using the road-mapping technique, immediate reversal of anticoagulation treatment, reducing the systemic blood pressure, and emergency angiography are essential. Immediate treatment decisions should be guided by the results of emergency angiography, with the material perforating the aneurysmal wall temporarily remaining in place.

If the emergency angiogram reveals extravasation, rapid packing of the aneurysm with Guglielmi detachable coils should be performed (2, 3). If the patient manifests an acute increase in intracranial pressure and persistent systemic hypertension despite rapid embolization of the aneurysm, emergency ventriculostomy may be required (4). If, on the other hand, the emergency angiogram shows no or minimal extravasation, some other methods should be considered. Placement of a bridging coil that prevents extravasation through the hole of the aneurysm may be useful (5). The use of a second microcatheter, as described in our case, may be a valuable technique. Willinsky and terBrugge successfully treated the perforation of a paraophthalmic aneurysm by using a second microcatheter. Several conditions were different between our case and the reported case. Because both the microcatheter and the second coil perforated the aneurysm in our case, the size of the hole might have been larger in our case than in theirs. The location of the aneurysm was also different. For the posterior circulation aneurysm, an approach through the contralateral vertebral artery is necessary because the diameter of the vertebral artery is much smaller than that of the carotid artery. If the aneurysm is located in the posterior circulation, we suggest that angiograms of the bilateral vertebral arteries be obtained before the start of endovascular treatment to iden-

tify a route for the possible introduction of a second microcatheter.

Toshinori Hirai
Kenji Suginothara
Shozaburo Uemura
Amakusa Medical Center

Jun-ichiro Hamada
Yukunori Korogi
Mutsumasa Takahashi
Kumamoto University School of Medicine
Kumamoto, Japan

References

1. Willinsky R, terBrugge K. Use of a second microcatheter in the management of a perforation during endovascular treatment of a cerebral aneurysm. *AJNR Am J Neuroradiol* 2000;21:1537–1539
2. Viñuela F, Duckwiler G, Mawad M. Guglielmi detachable coil embolization of acute intracranial aneurysm: perioperative anatomical and clinical outcome in 403 patients. *J Neurosurg* 1997;86:475–482
3. McDougall CG, Halbach VV, Dowd CF, Higashida RT, Larsen DW, Hieshima GB. Causes and management of aneurysmal hemorrhage occurring during embolization with Guglielmi detachable coils. *J Neurosurg* 1998;89:87–92
4. Ricolfi F, Le Guerinel C, Blustajn J, et al. Rupture during treatment of recently ruptured aneurysms with Guglielmi electrodetachable coils. *AJNR Am J Neuroradiol* 1998;19:1653–1658
5. Halbach VV, Higashida RT, Dowd CF, Barnwell SL, Hieshima GB. Management of vascular perforations that occur during neurointerventional procedures. *AJNR Am J Neuroradiol* 1991;12:319–327

The Petrosquamosal Venous Channel

We read the article by Marsot-Dupuch et al (1), “The Petrosquamosal Sinus: CT and MR Findings of a Rare Emissary Vein,” with great interest. We would like to take this opportunity to emphasize the two following points.

First, our evaluation of 13 anatomic corrosion casts of the cerebral venous system corroborates

the existence of a petrosquamosal venous channel, commonly referred to as the petrosquamosal sinus (PSS). This structure was present in five of the 26 corrosion cast sides we studied (Figs 1 and 2). In three instances, the PSS had the typical appearance of a diploic channel (ie, rounded irregular contours and a tortuous course), which seems to correlate with the CT scans presented in the article by Marsot-Dupuch et al. However, we found no association with venous anomalies such as described by these authors. Considering that a petrosquamosal sinus was observed in five of 26 specimen sides (19%) and that not all the corrosion casts indicated complete filling of the cerebral and meningeal venous system, we think that the petrosquamosal sinus can not be considered a rare anatomic entity. Although no investigational procedure is perfect, endovascular moldings obtained with the corrosion cast technique remain a powerful anatomic tool for the study of vascular structures, particularly when they are difficult to access, as is the case for the venous system at the skull base.



FIG 1. Left lateral corrosion cast of the cerebral venous system shows the PSS (arrows), the transverse sinus (1), the sigmoid sinus (2), the cavernous sinus (3), the pterygoid plexus (4), and the mastoid emissary vein (asterisk).

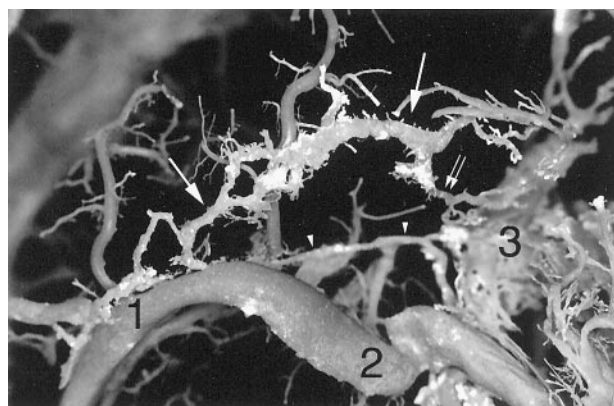


FIG 2. Right lateral corrosion cast of the cerebral venous system (different specimen). Note the presence of the superior petrosal sinus (arrowheads) and the medial connection of the petrosquamosal sinus to an emissary vein of the middle cranial fossa (double arrow). 1 indicates the transverse sinus; 2, sigmoid sinus; 3, cavernous sinus.

Second, in their discussion, Marsot-Dupuch et al. mention that emissary veins connected to the sigmoid sinuses (ie, the posterior condylar vein, the mastoid emissary vein, and the petrosquamosal sinus) play a minor role in healthy persons, acting as a safety valve. This conclusion is based on the assumption that the only normal drainage pathway for encephalic blood flowing through the sigmoid sinuses occurs via the internal jugular veins. This assumption is correct for a person who is lying supine. However, in the upright position, encephalic drainage occurs mainly through the internal and external vertebral venous plexuses (2, 3). This drainage pattern is rendered possible by connections linking the vertebral venous plexuses to the transverse and sigmoid sinuses and to the bulb of the internal jugular vein. These connections occur via the anterior, lateral, and posterior condylar veins and via the mastoid and occipital emissary veins (4). Emissary veins, therefore, play a major role in encephalic and neurocranial venous drainage. We agree, however, that the petrosquamosal sinus most likely plays a minor role in encephalic drainage because it is not connected to the vertebral venous plexus.

Diego San Millán Ruíz, MD
Jean H. D. Fasel, MD
University of Geneva
Geneva, Switzerland

Philippe Gailloud, MD
The Johns Hopkins Hospital
Baltimore, MD

References

1. Marsot-Dupuch K, Gayet-Delacroix M, Elmaleh-Berges M, Bonneville F, Lasjaunias P. **The petrosquamosal sinus: CT and MR findings of a rare emissary vein.** *AJNR Am J Neuroradiol* 2001;22:1186-1193
2. Eckenhoff J. **The physiologic significance of the vertebral venous plexus.** *Surg Gynecol Obstet* 1970;131:72-78
3. Valdueza JM, von Munster T, Hoffman O, Schreiber S, Einhaupl KM. **Postural dependency of the cerebral venous outflow.** *Lancet* 2000;355:200-201
4. San Millán Ruíz D, Rüfenacht DA, Delavelle J, Fasel JHD. **The craniocervical venous system in relation to cerebral venous drainage: an anatomical contribution.** *Proceedings of the 39th Annual Meeting of the American Society of Neuroradiology*, Boston, Mass; ASNR; 2001

In Re: Arterial Dissection Complicating Cerebral Angiography and Cerebrovascular Interventions

Belatedly, I read the above-mentioned article written by Cloft et al (1) and published in the March 2000 issue of *AJNR*. It seems that our younger colleagues do not pay enough attention to the older medical literature: in 1975, an article (2) dealing with exactly the same topic was published. It enumerated exactly

the same causes of damage in the brachiocephalic arteries due to the catheter angiography and had exactly the same conclusions. At that time, we redesigned the multipurpose catheter that we used for brachiocephalic angiography in such a way that the tip of the catheter did not direct the injected contrast against the vessel wall but into the bloodstream and that it was difficult to wedge into the vessel wall. Other work (3, 4) also deserves to be mentioned. The value of Dr Cloft's article is that he reminded younger neuroradiologists of the possibility that iatrogenic damage to the brachiocephalic arteries during diagnostic or interventional procedures may occur and discussed how to avoid them.

Jiri Vitek, MD, PhD
*Lenox Hill Heart and Vascular Institute
New York, NY*

References

1. Cloft HJ, Jensen ME, Kallmes DF, Dion JE. **Arterial dissections complicating cerebral angiography and cerebrovascular interventions.** *AJNR Am J Neuroradiol* 2000;21:541-545
2. Vitek JJ, Powell DF, Anderson RD. **Damage of the brachiocephalic vessels due to catheterization.** *Neuroradiology* 1975;9:63-67
3. Lang EK. **A survey of the complications of percutaneous retrograde arteriography.** *Radiology* 1963;81:257-263
4. Gilbert GJ, Melnick GS. **Pathophysiology of subintimal hematoma formation during retrograde arteriography.** *Radiology* 1965;85:306-319

Reply:

I apologize to Dr Vitek for overlooking his earlier work. I appreciate his pointing this out to the neuro-radiology community so that we can all learn from his work and the work of others.

Harry J. Cloft, MD, PhD
*Mayo Clinic
Rochester, MN*