that supplied both frontal lobes. The circle of Willis as an anatomic structure can be defined as a network of blood vessels affording communication between the carotid system and the basilar system, encompassing an anterior and two posterior communicating arteries and proximal portions of the anterior and posterior cerebral arteries. However, this description is still not fully satisfying because part of the middle cerebral artery and the top of the basilar artery also contribute to this communicating circle. It was our deliberate choice to list the anomaly among anomalies of the circle of Willis to avoid confusing the reader, who will find virtually all other previously reported variations of the postcommunicating ACA in articles about anomalies of the circle of Willis, as is illustrated in several important papers considering large populations.1-4 Moreover, De Smet illustrates the ambiguous use of the term “circle of Willis” even by Willis himself when De Smet refers to “the circle depicted in Willis’ Cerebri Anatome” showing “the ACAs proceeding medially and coming together in a confluence, a possible but most unlikely anomaly...” Furthermore, the statement that the unique anomaly we described could fact have first been reported by Willis himself in 1664 is extremely speculative, as is also illustrated by the use of the conditional tense by De Smet in his letter. Even if, as he suggests, the draftsman who contributed to Willis’ 1664 paper included “the unknown anterior communicating artery” in the precommunicating part of the right ACA, the anomaly in our patient still differs from this would-be variant because of the existence of a plain communicating ACA.

In the second part of his letter De Smet addresses the clinical features of our case. Erroneously, however, he writes that our patient had ophthalmoplegia. This was the case for one of the two patients discussed by Ferbert and Thron,1 who rightly conclude that bilateral ACA territory infarction should be considered in the differential diagnosis of basilar artery occlusion, even when it is accompanied by vertical gaze palsy. De Smet proposes that an association of quadriplegia with akinetic mutism and ophthalmoplegia corresponds to the tetraptaratic mutism he described,8,9 which combines a locked-in-like deafferentation with an akinetic mutism. For our patient, we prefer to use only the term “akinetic” mutism and agree with Ferbert and Thron that variations of a classical akinetic mutism syndrome may exist. Communication by means of a code of eye blinking or vertical eye movements, as in the locked-in syndrome, was not possible for our patient. We further suggest an association with receptive aphasia, because of the state of akinetic mutism we were unable to prove this hypothesis.

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References


Vertebral Flow Void and Lateral Medullary Syndrome

To the Editor:
The outstanding descriptions by Kim et al1 of magnetic resonance (MR) imaging findings in the lateral medullary syndrome include three fabulous MR imaging sections through the medulla (Fig 2). The figure legend does not provide details of the neuroradiological findings. One “pearl” overlooked in the text is that all three images showed absence of the normal left vertebral flow void (FV) phenomenon. This finding has been previously described2 and is worth reiterating.

Joseph S. Jeret, MD
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References


Response

Dr Jeret noticed the abnormal vertebral FV pattern in the left vertebral artery presented in Fig 2 of our article.1 In fact, the right vertebral artery showed enhanced FV signal on a T2-weighted image unaccompanied by corresponding changes on the T1-weighted image in patients with various types of stroke. This phenomenon might suggest reduced blood flow velocity in vertebral arteries. However, this almost always occurs bilaterally, and exclusively in vertebral arteries (but not the basilar artery) seen in less than three consecutive cuts from the lowest image. Therefore, we think that this phenomenon is more likely to be caused by tilted cutting of the vertebral artery or entry slice phenomenon7 and that a decisive conclusion should not be made unless the T2-weighted MR image shows compatible findings.
Nevertheless, after receiving this interesting correspondence, we reviewed the vertebral FV pattern seen on MR imaging in the 26 patients with lateral medullary syndrome included in our study. In five patients we noted the absence of normal vertebral FV on the T2-weighted image, all on the same side of the lesion. Enhanced FV signal in the same vertebral artery was noted on the T1-weighted image in all five patients. We also examined the vertebral FV pattern in a control group of 20 patients with unilateral pontine basis infarction who were of comparable mean age and had vascular risk factors similar to those of the patients with lateral medullary syndrome. We found that none showed the absence of vertebral FV. Therefore, although it is infrequently seen, the altered vertebral FV pattern judged from both T2- and T1-weighted MR images appears to be of value in the evaluation of the vascular status of patients with lateral medullary syndrome.

References

Vertebral flow void and lateral medullary syndrome.

J S Jeret

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