Bilateral Infarction in the Anterior Cerebral Artery Vascular Territory

To the Editor:

Borggreve et al reported a case of bilateral infarction in the anterior cerebral artery (ACA) territory due to a unique anomaly of the circle of Willis, the absence of the postcommunicating part of the right ACA. In fact, this anatomic variant is not an anomaly of the circle of Willis in the strictest sense, but consists more of a unique postcommunicating ACA. The circle depicted in Willis' Cerebri Anatom shows the ACAs proceeding medially and coming together in a confluence, a possible but most unlikely anomaly; the anterior communicating artery was not depicted and was unknown to Willis. The impression is of a drawing undertaken with insufficient care to faithfully reproduce the arterial anatomy. Possibly the unknown anterior communicating artery was included by Willis' draftsmen in the precommunicating part of the right ACA; so the unique anomaly described by Borggreve et al might in fact have first been reported by Willis himself in 1664.

The main clinical features reported by Borggreve et al were quadriplegia with akinetic mutism and ophthalmoplegia, an association that mimics a basilar artery occlusion. In fact, such an association corresponds to the tetraparetic mutism, described by De Smet et al, that combines a locked-in-like de-afferentation with an akinetic mutism-like deafferentation. Finally, the cases reported by Borggreve et al and Ferbert and Thron suggest the possibility of an almost complete quadriplegia that includes the face and plantar reflexes in extension with bilateral infarction of the ACA cortical (gyrus precentralis), subcortical (corona radiata), and striatocapsular (anterior limb) territories. Such a deficit is quite different from the nonpyramidal hemispheric syndrome seen in cases of ACA striatocapsular territory infarction. It thus implies the possibility of aberrant pyramidal fibers in the anterior limb of the internal capsule or the extension of the ACA cortical and subcortical territories beyond their classic boundaries.

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References


Response

We appreciate the interest shown by Dr De Smet in our patient who had bilateral infarction in the ACA vascular territory, and welcome his attempt to add some historical perspective to this unusual case. However, we can only partially agree with the issues raised. The described anomaly did not involve the circle of Willis as such but actually involved an absence of the precommunicating part of the right ACA; in addition, the postcommunicating part of the left ACA had an increased diameter and split into two arteries.
that supplied both frontal lobes. The circle of Willis as an anatomic structure can be defined as a network of blood vessels absorbing 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.14 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 Anato mie" showing "the ACAs proceeding medially and coming together in a confl uence, a possible but most unlikely anomaly..." Furthermore, the statement that the unique anomaly we described could in 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,6 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 tetraparetatic mutism he described,8,10 which combines a locked-in-like deafferentation with an akinetic mutism-like deafferentation. 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-like 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, but because of the state of akinetic mutism we were unable to prove this hypothesis.

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 described11 and is worth reiterating.

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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 this T2-weighted MR image as well. The enhanced signal intensities were most intense at the level of the lower medulla (bottom panel), which diminished as the section planes went up. Unfortunately this figure, presented to demonstrate the MR imaging findings of three different cuts of the medulla, was not from a patient with lateral medullary syndrome but from one with a supratentorial lacunar infarct. In this patient, the T2-weighted MR image showed a normal vertebral FV pattern, arguing strongly against the possibility of thrombus formation.

The FV pattern in MR imaging has usually been assessed using a T2-weighted image,2 where altered FV signal is more reliably detected. In our daily practice, we occasionally observe the abnormal vertebral FV pattern on a T2-weighted image unaccompanied by corresponding changes on the T2-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 phenomenon, and that a decisive conclusion should not be made unless the T2-weighted MR image shows compatible findings.
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