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To the Editor:

We read with great interest the article by Metso et al¹ and congratulate the authors for their important work for 2 main reasons: (1) previous studies about intracranial artery dissections (IAD) were based either on patients recruited in neurological departments after experiencing mainly ischemic events and/or local symptoms and signs (eg, headache, Horner syndrome), or in neurosurgical departments after a subarachnoid hemorrhage. Metso et al¹ included all consecutive patients from the neurological as well as the neurosurgical units of the Helsinki University Central Hospital and avoided the inclusion bias mentioned above. Second, the authors report the largest series of patients with IAD.

The authors conclude that IAD presenting with cerebral ischemia and showing no aneurysm at angiography can be safely anticoagulated, and their conclusion is shared by Leys and Debette² in their Editorial Comment. We think that the data presented are not sufficient to prove that anticoagulation is safe in IAD.

The reliable in vivo diagnosis of cerebral arterial dissection is based on the detection of an intramural hematoma, a double lumen, an intimal flap, or an aneurysm at a nonbifurcation site.^{3,4,5} In contrast, detection of a flame-shaped occlusion, a string sign or a segmental stenosis at angiography is nonspecific. In our experience, IAD are difficult to firmly diagnose because of the rarity of specific signs. This is illustrated in the Metso et al series because they found an intracranial wall hematoma in 3 (13%) and an intracranial intimal flap or double lumen in 8 (35%) of 23 patients with "solely intracranial dissection." The remaining patients showed nonspecific angiographic signs such as occlusion or stenosis which may be of atherothrombotic or embolic origin. The corresponding figure for extracranial dissections extending intracranially are unclear because the authors did not state whether the wall hematoma, the intracranial intimal flap and double lumen were also detected intracranially.¹ This differentiation between IAD and other varieties of occlusion or stenosis, however, is crucial because anticoagulation of an intracranial thrombus or embolus is not expected to cause a vessel rupture and subarachnoid hemorrhage, whereas a dissected intracranial artery might indeed carry a risk of rupture. Therefore, it is of paramount importance that Metso et al report

how many of their patients had radiological findings permitting the unequivocal diagnosis of IAD.

We would also like to comment on the diagnosis of subarachnoid hemorrhage, which in Metso et al's series was exclusively based on brain CT or MRI. Some patients with IAD have a normal CT but blood in cerebrospinal fluid. This is why we routinely perform—in the absence of contraindications—a lumbar puncture in patients with IAD before starting heparin or aspirin. In conclusion, we agree that it is likely that anticoagulation in IAD is safer than previously thought, but it is too early to firmly conclude that it is safe.

Disclosures

None.

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