In most textbooks, intracranial artery dissections are considered rare, difficult to diagnose, and associated with a high risk of subarachnoid hemorrhage (SAH). Three different clinical presentations are described: (1) cerebral ischemia, when the mural hematoma remains intimal, ie, located between the media and the elastica interna, leading to vessel stenosis or occlusion; (2) SAH when the mural hematoma is subadventitial, ie, located between the media and the adventitia, or transmural, leading to vessel rupture; and (3) acute and isolated headache. Cerebral ischemia and headache are not specific, because they are also present in most patients with cervical artery dissections. Bleeding is a specificity of intracranial artery dissections. It is explained by structural differences between cervical and intracranial arteries: the latter has no external elastic membrane, and thin muscular and adventitial layers.

There is no clear evidence-based data that anticoagulation, or even antiplatelet therapy, may be beneficial in patients with cervical artery dissection, even if there is a strong rationale for their use, and some indication of a good safety profile. In pure intracranial artery dissections, or in intracranial artery dissections patients, there is no proof—in the absence of randomized trials—that it provides a benefit over antiplatelet therapy or even no treatment at all. Thirdly, the natural history of intracranial artery dissections with cerebral ischemia remains unknown in terms of recurrence risk. This is an important issue because anticoagulation is supposed to prevent early recurrences, not to reduce the severity of stroke.

This study provides important results on the safety profile of anticoagulation in patients with intracranial artery dissections, but this is not enough to recommend anticoagulation. Assuming that early recurrence rate is very low, no case having been reported in 81 patients, if we hypothesize that anticoagulation reduces by \( \approx 25\% \) early recurrences, >10 000 patients would have to be randomized in an early prevention trial, which is unrealistic for such a rare disease. Therefore, in the absence of randomized trials the decision will be left to the clinical judgment of the neurologist. This study provides 2 important clinical messages: (1) anticoagulation is safe in the absence of bleeding, and (2) recurrences being rare anticoagulation may have a limited indication.

Another major interest of this study is to report a large series of intracranial artery dissections associated with cerebral ischemia. As stated by the authors, most of the literature on intracranial artery dissections has been published by neurosurgical teams, explaining a probable overrepresentation of cases with SAH and aneurysms. This series, although retrospective, is a good representation of intracranial artery dissections in the general population because the authors included all consecutive patients admitted in the only neurological and neurosurgical emergency unit of Helsinki and surroundings.

We are not sure that clinicians will change their practice after this study. Those who believe anticoagulation is useful will continue and argue that it is safe, and those who are more reluctant to use anticoagulation will probably not change their strategy, arguing that the action of anticoagulation is to prevent early recurrences, which are rare, if they exist. As often with good studies, we have more questions at the end than before. Larger multicenter registries, with different strategies left to the opinion of the clinicians, may help to better define the natural history of the disease. The study reported by Metso et al is obviously the first significant piece of the puzzle.

**Disclosures**

None.
References


**Key Words:** dissection ■ intracranial aneurysm ■ intracranial disease ■ intracranial stenosis ■ subarachnoid hemorrhage
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