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Letter to the Editor

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Asymptomatic Common Carotid Artery Dissection Requires Comprehensive Clarification of the Underlying Cause so as not to Miss Treatment Options

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LETTER TO THE EDITOR

We were interested to read the article by Takahashi et al. about a 70-year-old man with a spontaneous dissection of the left common carotid artery (CCA), which was discovered incidentally during an ultrasound examination of the thyroid gland to clarify a thyroid nodule and was asymptomatic [Takahashi, T. *et al.*, 2024]. The patient underwent a successful endarterectomy and recovered well without developing a clinically manifest or subclinical stroke [Takahashi, T. *et al.*, 2024]. The study is impressive, but some points should be discussed.

The first point is that the cause of the CCA dissection was not clarified [Takahashi, T. *et al.*, 2024]. To prevent recurrence of the dissection or the occurrence of a dissection in a different arterial bed, it would have been imperative to systematically search for possible causes of the dissection. Among the causes that should have been excluded, as they have been reported in association with dissection of extra- or intracranial arteries, are vasculitis [Jamil, O. *et al.*, 2016], fibromuscular dysplasia [Gonzalez, F. E. *et al.*, 2024], a mitochondrial disorder [Kalashnikova, L. A. *et al.*, 2010] or paraneoplasia. Therefore, we should know whether the vasculitis parameters were indicative of ANCA-positive/negative large vessel vasculitis. It would also be interesting to know the results of the renal mass examination, in particular whether it was a benign or malignant tumour. Was there any evidence of a mitochondrial disorder in the family history or did the index patient exhibit typical phenotypic characteristics of a mitochondrial disorder? As the case may have occurred during the pandemic, it is also important to know whether the index patient was SARS-CoV-2 positive in the weeks prior to the discovery of the dissection. SARS-CoV-2 infections are known to be complicated by dissection of the carotid artery [Noble, O. *et al.*, 2024].

The second point is that the medical history of the index patient was sparsely reported. We should also know what kind of medication the index patient was taking regularly. Was there evidence of a rheumatologic disease, a metabolic disorder, a chronic infectious disease or a connective tissue disease such as Ehlers-Danlos syndrome, Marfan syndrome or osteogenesis imperfecta? It must also be checked whether the index patient suffered from hyperhomocysteinemia [Lusawat, A, 2011].

Thirdly, it is incomprehensible why the postoperative magnetic resonance angiography (MRA) still showed a double lumen in the CCA despite endarterectomy. Why was the section of dissection not resected and replaced by a patch? One would expect only a single lumen to be present after successful endarterectomy.

The fourth point is that the results of preoperative cerebral MRI were not reported. To assess whether the CAA dissection was truly asymptomatic, we should know whether the preoperative cerebral MRI was truly normal as reported for the postoperative MRI.

In conclusion, the index study has limitations that put the results and their interpretation into perspective. Addressing these limitations could strengthen the conclusions and support the results of the study. CCA dissection requires a comprehensive workup of the underlying cause of the dissection, not overlooking an etiology amenable to more causal treatment of the dissection.

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