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BEFORE SYMPTOMATIC INTERNAL CAROTID ARTERY STENOSES CAN BE ATTRIBUTED TO GIANT CELL ARTERITIS, OTHER CAUSES MUST BE RULED OUT

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Giant cell arteritis, vasculitis, ischaemic stroke, endovascular therapy, cardiovascular risk profile

LETTER TO THE EDITOR

We read with interest Orfao et al.'s article about a 68-year-old female diagnosed with giant cell arteritis (GCA) and presenting with multiple ischaemic strokes[1]. GCA was diagnosed based on clinical presentation, ultrasound examination of extracranial arteries, and magnetic resonance angiography and computed tomography angiography (CTA)[1]. The patient did not benefit from glucocorticoids, but significantly from stenting of both supraclinoidal internal carotid artery stenoses[1]. The study is excellent, but some points need discussion.

The first point is that the diagnosis of GCA was made based on clinical presentation, ultrasound (halo sign) and blood tests, but not by biopsy. There is also no ophthalmologic examination documenting retinal involvement in GCA[1]. GCA should be documented by temporal artery biopsy and on ophthalmologic examination.

The second point is that there is no discussion as to how atherosclerosis was excluded as a cause of the supraclinoidal stenoses of the internal carotid arteries as shown in Figure 4[1]. The patient was of advanced age and had at least hyperlipidaemia as one of the classic cardiovascular risk factors.

The third point is the discrepancy between the statement in the abstract that 'computed tomography angiography (CTA) and ultrasound showed severe vascular disease involving multiple territories, without significant intracranial involvement' and the conclusions that 'the case is outstanding for the importance of endovascular treatment (EVT) as an effective therapy in patients with medically refractory GCA with symptomatic intracranial stenosis'[1]. This discrepancy should be resolved. We should know whether intracranial arteries were normal or affected by vasculitis or atherosclerosis. At least Figure 4 in the report suggests that the supraclinoidal portion of the internal carotid artery was affected[1].

The fourth point is that cardiac embolism has not been sufficiently ruled out. We should know the results of echocardiography and 24-hour ECG monitoring to assess whether there was endocarditis, intraventricular thrombus formation, Takotsubo syndrome or atrial fibrillation. There is evidence that non-infective endocarditis may be a complication of GCA[2]. Therefore, it is imperative that cardiac embolism is definitively ruled out as a cause of multiple ischaemic strokes.

The fifth point is that the index patient was not evaluated

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for malignancy. There is evidence that GCA can be a paraneoplastic phenomenon and the first manifestation of malignancy[3]. Therefore, it would have been imperative to examine the patient for malignancy, particularly lung carcinoma, carcinoid tumour and breast carcinoma[4].

The sixth point is that the pathophysiology of multiple strokes has not been sufficiently discussed assuming that GCA was the underlying aetiology. Were strokes the result of an arterio-arterial embolism, vasospasms, local thickening of the vessel walls or local thrombosis?

In summary, the excellent study has limitations that should be addressed before final conclusions are drawn. Clarifying the weaknesses would strengthen the conclusions and improve the study. Before bilateral, symptomatic internal carotid artery stenoses can be attributed to GCA, alternative causes must be ruled out.

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