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Letter to the editor

Elongated Styloid Process Can be Made Responsible for Recurrent Embolic Stroke Only if Eagle Syndrome is Present

Josef Finsterer[1*], Sounira Mehri[2]

[1]Neurology and Neurophysiology Center, Vienna, Austria.
[2]Disciplina de Neurociência. Universidade Federal de São Paulo/Escola Paulista de Medicina (UNIFESP/EPM). São Paulo, Brasil.

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Corresponding author:
Josef Finsterer
fifigs1@yahoo.de

With interest we read the article by Xu et al. on a 59 year-old male with recurrent episode of left hemispheric, embolic stroke [1]. Elongated styloid process (ESP) and carotid web pocket were identified as causes of recurrent stroke [1]. After resection of the ESP and resection of the carotid web, the patient became asymptomatic during a 6 months follow-up [1]. The study is excellent but has limitations that are cause of concerns and should be discussed.

The main limitation of the study is that alternative causes of embolic stroke were not entirely ruled out. We should know whether there were any indications for alternative causes of embolic stroke, such as heart failure, atrial fibrillation, Takotsubo syndrome. noncompaction, endocarditis, valve abnormalities, or myocarditis. Missing in this respect are echocardiography and ultrasound of vertebral arteries. As long as all these alternative sources of embolism were not adequately ruled out, ESP and carotid web pocket cannot be made responsible for recurrent stroke with certainty. There is also no mention of inflammatory parameters, blood coagulation parameters, platelet counts, troponin values, and pro brain-natriuretic peptide (proBNP).

A strong argument against the ESP as the cause of recurrent stroke is that ESP presumably existed already for a long time. No explanation is provided why it became symptomatic just at age 58 years. Where there ever any sudden neurological deficits recognised during previous years (>1.5 years before)?

Another argument against ESP as the cause of recurrent stroke is that the index patient did not present with typical Eagle syndrome also referred as stylohyoid syndrome, styloid syndrome, or styloid–carotid artery syndrome [2]. Eagle syndrome is clinically characterised by orofacial and cervical pain often triggered by neck movements, by dysphagia, facial palsy, slurred speech, and vertigo [2]. Eagle syndrome can be associated with ankyloting spondylitis [3], which should have been ruled out in the index patient.

The diffusion weighted imaging (DWI) lesions shown in figure 1 panel A do not explain right-sided weakness of the upper limb and amaurosis. The precentral lesions are too small and not directly in the area of the cortical upper limb representation to explain limb weakness.

Regarding unilateral amaurosis, we should know whether ophthalmologists ever documented occlusion of the central retinal artery. Amaurosis triggered by head

flexion suggests that each time the head is flexed embolization into the left central retinal artery occurs, which is rather unlikely. Clamping of the carotid artery by head flexion is unlikely since no cerebral symptoms with head flexion were reported [1]. Another argument against amaurosis as a manifestation of ESP is that no such cases have been previously reported.

Overall, the interesting study has limitations that call the results and their interpretation into question. Addressing these issues would strengthen the conclusions and could improve the status of the study. Before attributing recurrent embolic stroke to ESP and carotid web, it is crucial to adequately rule out all alternative causes of embolic stroke. Recurrent unilateral amaurosis triggered by head flexion cannot be easily explained by ESP.

Keywords: Stroke, Elongated Styloid Process, Carotid Artery, Carotid Web

Declarations

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Consent to publish: NA

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