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LETTER TO THE EDITOR

Epistaxis as unusual presentation of carotid siphon aneurysms, evolved in subacute carotid thrombosis

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Background

Epistaxis as the first clinical manifestation of intracranial aneurysm is a rare event. Moreover, coexistence of aneurysm rupture and progressing stroke due to spontaneous aneurysm thrombosis generates a complex decision-making discussion.

Case presentation

An 88-year-old man with history of mild hypertension since age 65 and two remote transient ischemic attacks at age 66 and 72, in treatment with Clopidogrel, presented to the emergency department for a severe epistaxis with acute onset.

He had experienced right facial pain for the previous month and had a neurological evaluation two weeks prior to the episode of epistaxis that had not found any neurological abnormality. However, during the neurological evaluation, he had an initial episode of epistaxis, which was treated with nasal packing.

Epistaxis continued for at least 3 h, requiring anterior and posterior nasal packing and two blood transfusions of concentrated red blood cells. Rhinoscopy showed only blood clots in rhinofarinx. Clopidogrel was then discontinued, and the patient was discharged home.

One week later, a new episode of severe epistaxis required again hospitalization, and, in addition to packing and transfusions, he was also given tranexamic acid. Cerebral and facial MRI showed possible bleeding within the right sphenoid sinus, associated to a possible dilation of the ipsilateral carotid siphon. Two days later, he developed a mild left hemiparesis with dysarthria (NIHSS 3), first witnessed at wake-up time.

A non-enhanced CT scan excluded cerebral hemorrhage and did not show acute ischemic changes; over the following hours, he partially recovered, with a residual left arm paresis. He was not deemed a candidate for systemic thrombolysis, due to recent major hemorrhage, nor to mechanical thrombectomy due to low NIHSS score and unknown hour of stroke onset (wake-up stroke).

Three days after, CT angio revealed a large, 15-mm aneurysm of the siphon of the right internal carotid, with a large neck; it was located in the right sphenoid sinus, through an erosion of the bone wall of the sinus itself. No other carotid pathology was reported (Figs. 1 and 2).

Both epistaxis and subsequent ischemic stroke were thus thought to be related to the aneurysm, the first due to fissuration of the aneurysm sac in the extracerebral space, and the second due to artery-to-artery embolism caused by initial thrombosis of the aneurysm itself.

Although the large neck of aneurysm and the old age of patients, considering the high risk of rupture and the impending thrombosis, the patient was scheduled for endovascular treatment, for the next morning.

During the night, his neurological condition worsened: he developed left hemianopia and complete palsy of the left arm, associated with moderate paresis of the left leg and fluctuating state of consciousness. Cerebral CT scan and CT-angio scan, done 24 h after the previous one, showed complete occlusion of left internal carotid artery, 1 cm form the origin to carotid siphon, with complete exclusion of the aneurysm from cerebral circulation. Both MCA and ACA were patent, with good Willis circulation and collaterals (Figs. 3 and 4).

Thus, clinical worsening was due to infarction in the vascular territory of anterior choroidal artery (with initial ischemic lesion on cerebral CT) and the patient was given antiplatelet therapy (acetil salicilic acid, 300 mg/day).

Unfortunately, carotid thrombosis evolved, and in the next three days extended to both MCA and ACA, causing a

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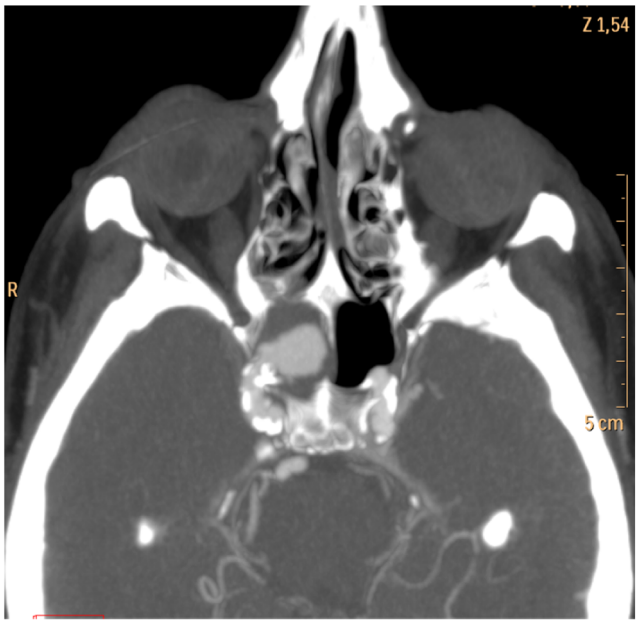

Fig. 1 CT-angio showing right carotid siphon aneurism, located in the sphenoid sinus

massive cerebral infarction. Clinical worsening finally ended to rostrocaudal deterioration and death.

Discussion and conclusions

Our case presents several points of interest: the first is epistaxis related to carotid aneurysm. This is an uncommon cause of epistaxis, and it is important to be known and to be suspected when massive epistaxis requiring blood transfusion occurs, especially when it recurs at short time or in subjects with a previous history of traumatic brain injury (more than half of the cases in literature are in fact pseudoaneurysms due to trauma) [1–4].

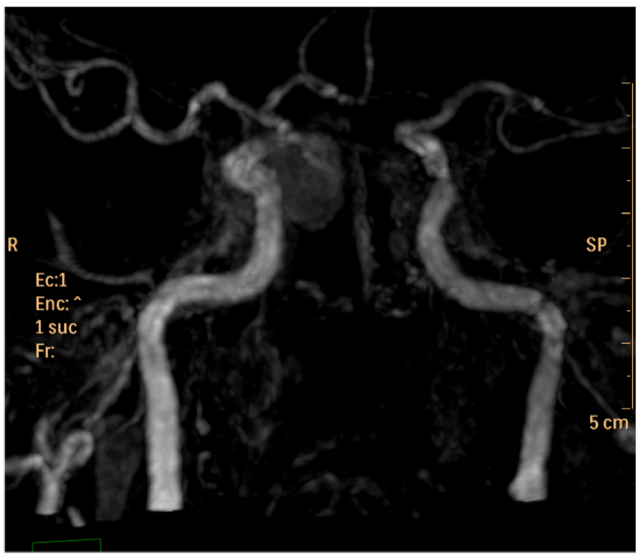

Fig. 2 MR-angio (TOF 3D) showing right carotid siphon aneurysm

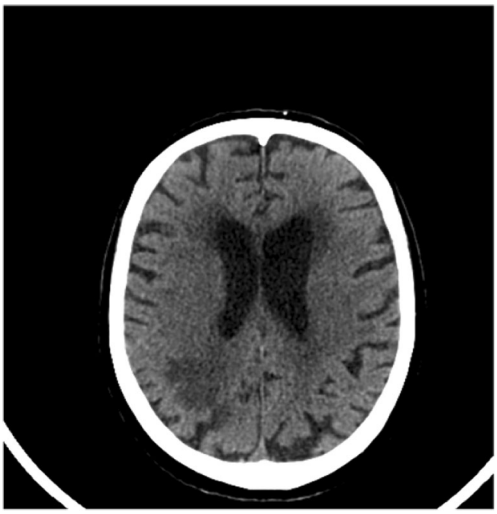

Fig. 3 Recent right parietal infarction on CT scan

The second point of interest of the case is the spontaneous thrombosis of the aneurysm, visualized "real time" by imaging. The previous transient episode could have been a distal embolism, coming from the intra-aneurysm thrombosis, then progressed to the whole carotid parent artery.

Spontaneous thrombosis of aneurysm sac causing large stroke is extremely rare, and only a few cases have been described in literature so far [5–8].

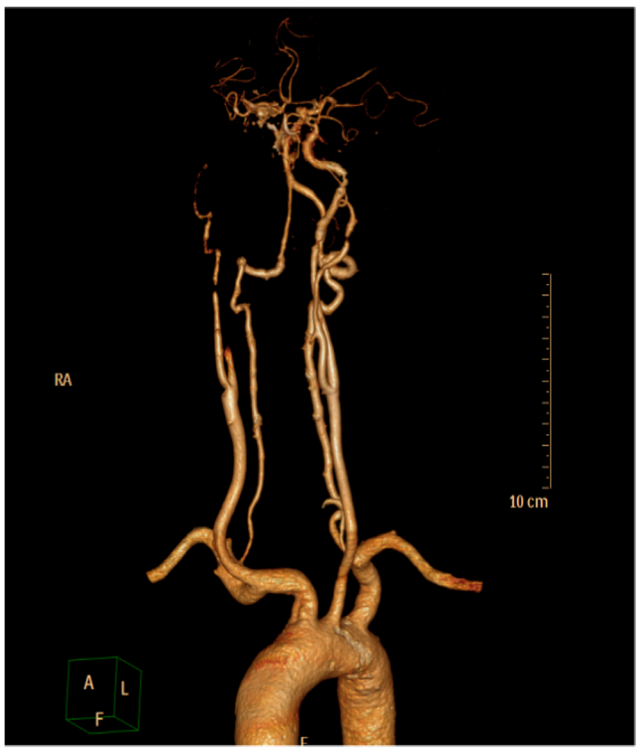

Fig. 4 Right internal carotid artery occlusion on CT-angio

Finally, our case underlies the importance of evaluating, for clinical decision-making, the risk of rupture of an aneurysm, even in cases of extra-dural fissuration, thus without subarachnoid hemorrhage. Recent data suggest that calculation of wall shear stress (WSS) of the aneurysm could predict risk rupture of the aneurysm [9]. In particular, mean aneurysm parent WSS ratio could predict the rupture risk of narrow-necked giant aneurysms. These computational fluid data could be helpful for clinical decision-making in these patients. In our case, the old age and the relatively high risk of endovascular treatment [10] did not stop our neuroradiologist of considering intervention, unfortunately not done because of rapid clinical deterioration.

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