Vertebral Pseudo-occlusion Due to Acute Basilar Artery Thrombosis

Sir,

The concept of acute arterial pseudo-occlusion (PO) defines a flow artifact in CT angiography (CTA) or digital subtraction angiography (DSA) that gives the appearance of occlusion at a certain level when the vessel is, in fact, permeable and the occlusion is localized at a more distal segment.[1] This finding is associated with a slow flow of unopacified blood due to the lack of drainage pathways.[2]

PO has been described in recent years at the extracranial internal carotid artery (ICA) by thrombi located mainly in the intracranial segment, appearing in up to 46% of these patients.[3] However, there are no previous reports of arterial PO in the posterior circulation.

A 56-year-old woman presented with new-onset gait unsteadiness, vomits, and a decreased level of consciousness, requiring orotracheal intubation. Unenhanced brain CT revealed a proximal hyperdense basilar artery and CTA showed an unopacified left vertebral artery that is filled distally (V4 and V3 segments) in a retrograde manner, suggesting proximal occlusion [Figure 1a and b].

Being contraindicated the intravenous thrombolysis due to an unclear-onset time, we performed a DSA to assess mechanical thrombectomy. Through the right vertebral artery, thrombus was localized in the proximal segment of the basilar artery with no evidence of competent posterior inferior cerebellar
arteries (PICAs). The left vertebral artery was permeable, without any sign of occlusion or dissection [Figure 1c and d]. Notwithstanding basilar thrombus removal, angiographic control images showed clot fragmentation and distal embolization to terminal branches of both posterior cerebral arteries, as well as basilar artery stenosis at initial thrombus site, which needed a balloon angioplasty. PICAs were still no patent. In the end, mTICI 2b recanalization was achieved.

Despite the reperfusion, clinical worsening occurred in the following hours, confirming on CT an extensive infarction in the territory of the basilar and both posterior cerebral arteries, subsequent oedema with mass effect, and closure of the IV ventricle, evolving into brain death.

Acute arterial PO is first described in 2010 by Kim et al.[4] as a CTA finding at extracranial ICA due to rapid acquisition that outpaces the slow flow of contrast-opacified blood. This produces a false image of complete occlusion at the extracranial level or even arterial dissection, with the characteristic “flame sign.”[3]

The ability to discern between sluggish flow and true extracranial occlusions remains difficult with current stroke neuroimaging protocols. This could influence the management of these patients if we misdiagnose either tandem occlusion or arterial dissection.[2]

Our patient clinical picture and the basilar artery hyperdensity in unenhanced CT, with no patent PICAs in CTA, made us think on the possibility of a vertebral PO instead of the CTA suspicion of proximal left vertebral occlusion.

We report the first documented case of artery PO not related to the carotid territory. Recent research on anterior circulation has delved into the pathophysiology of this phenomenon, which refers to the lack of drainage vessels proximal to the true location of the thrombus as the cause of the slow flow.[2] We should, therefore, understand PO as a physical phenomenon that can affect any artery and not just the already described extracranial ICA. In our case, the lack of a competent drainage pathway could explain the CTA images.

Concerning to prognosis, different case series have linked acute carotid PO to worse results of endovascular recanalization and poor functional prognosis.[3] The search for new cases will also allow us to assess the influence of this finding in posterior circulation events.

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Conflicts of interest
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