after a stroke raise the question of being able to repeat IVT within a short interval without increasing the bleeding risk. Interestingly, hyperacute lesions in ADC maps may reverse fully and permanently if full recanalization is rapidly achieved after stroke onset [2–4]. The shorter the delay for IVT and probably the smaller the clot size, the higher the frequency of early full recanalization and ADC decrease reversal with an early complete neurological recovery [3,4]. Some subtle histopathological changes might correspond to these reversible DWI anomalies. But, the absence of sequelae of recent ischemic episodes on successive MRI several days apart does not advocate this hypothesis and was the basis of the decision to repeat IVT.

Conflict of interest statement

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References


Hypoglossal artery associated with homolateral internal carotid artery dissection

Artère hypoglossée associée à une dissection de l’artère carotide interne homolatérale

Case report

A 37-year-old woman, with no history of either trauma or risk factor for cerebrovascular disease, presented with acute aphasia and right lower-limb weakness. Neurological examination showed right hemiparesis, Horner’s sign and Broca’s aphasia. The patient complained of episodic left-sided neck pain and frontal headache. Cerebral ischemia was suspected. Emergency 3-T magnetic resonance imaging (MRI) was performed to confirm the diagnosis of focal brain ischemia and to assess the supra-aortic vessels [1]. Diffusion images showed several foci of acute stroke in the left internal carotid artery (ICA) territory (Fig. 1A), associated with decreased flow in branches of the left intracranial carotid on intracranial 3D time-of-flight (TOF) images. Axial proton density fat-saturated images revealed a recent parietal hematoma of the left extracranial ICA (Fig. 1B).

Gadolinium-enhanced magnetic resonance angiography (MRA) of the supra-aortic vessels revealed occlusion of the left extracranial ICA and an abnormal presentation of the supra-aortic vessels, with persistence of a primitive left hypoglossal artery (Fig. 2). A tubular structure was also found in the enlarged left hypoglossal foramen (Fig. 1A). The three-month follow-up MRA showed complete recanalization of the left ICA (Fig. 3).

Persistent primitive hypoglossal artery (PPHA) is a rare vascular variant and the second most common carotid–basilar anastomosis, after persistent trigeminal artery, with a prevalence of 0.02–0.1% [2,3]. The presence of PPHA has been explained as failure of the embryological anastomosis to regress between the primitive ICA and bilateral longitudinal neural arteries [4]. The PPHA originates from the ICA at the C1–C3 vertebral body levels. It anastomoses with the basilar artery after its course through the hypoglossal canal, thereby differentiating it from the proatlantal artery, the third most frequent type of persistent carotid–basilar anastomosis, which passes through the foramen magnum.

Vertebral arteries are hypoplastic in 78% of cases, as in the present case, in which no left vertebral artery was seen. Posterior communicating arteries are also hypoplastic in 79% of cases. These findings are related to the variation in blood supply caused by the carotid–basilar anastomosis [5]. Despite occlusion of the ICA, the patient’s ischemic stroke was minor. The flow compensation can be explained by the patent left posterior communicating artery and the cross flow provided by the anterior communicating artery.

PPHA is usually an incidental finding [6], but persistent carotid–basilar anastomoses are linked to higher rates of arteriovenous malformation and cerebral aneurysm [5]. In rare cases, it has also been implicated in cranial nerve symptoms, such as glossopharyngeal neuralgia and hypoglossal nerve paralysis. To our knowledge, this is the first reported case of persistent hypoglossal artery associated with homolateral ICA dissection. In this case, the hypoglossal artery
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Figure 1  Axial diffusion images (A) show multiple hypersignals resulting from several acute foci of ischemia, while axial proton density fat-saturated images (B, C) show an acute parietal hematoma of the left extracranial internal carotid artery (arrow), suggesting arterial dissection, and a vascular structure in the hypoglossal canal (arrowhead).

Figure 2  Gadolinium-enhanced magnetic resonance angiography (MRA) of the supra-aortic vessels shows the left hypoglossal artery (arrowhead) arising from the initial segment of the left internal carotid artery, which is not visible downstream due to dissection.

remained normally perfused despite the acute ICA dissection, probably because the parietal hematoma in the spontaneous carotid dissection was located just below the petrous bone, whereas the origin of the PPHA was located 2 cm above the origin of the ICA. The resulting angiographic image is somewhat misleading and, thus, emphasis should be placed on analyzing the origins and courses of the supra-aortic vessels as well as the hypoglossal foramen caliber, which is generally larger.

Conflict of interest statement
None.

References
Magnetic resonance spectroscopy of a cerebral parasitic cyst

Spectroscopie par résonance magnétique d’un kyste cérébrale parasitaire

A 9-year-old boy, living in France under substandard conditions of hygiene, presented with right hemiparesis and left oculomotor nerve palsy that had progressively worsened over 3 weeks. Magnetic resonance imaging (MRI) disclosed a well-defined, round 6-cm lesion in the left frontoparietal region with cerebrospinal fluid (CSF)-like signal intensity and a mass effect (Fig. 1). A thin hypointense peripheral rim was noted on T2-weighted images, and there was no diffusion restriction, peripheral edema or enhancement (Figs. 2 and 3). Magnetic resonance spectroscopy ([MRS]; Fig. 4) revealed a succinate peak (2.4 ppm), an inverted lactate peak (1.3 ppm), a smaller alanine peak (1.48 ppm) and an acetate peak (1.9 ppm). These features suggested an infectious cyst. However, hydatidosis and cysticercosis serology and hepatic ultrasound examination were negative.

Surgical removal via a left frontal craniotomy and hydrodissection of the cyst was performed to avoid rupture (Fig. 5). The postoperative recovery was complete,