Arterial spin labeling demonstrates early recanalization after stroke

Recanalisation précoce après AVC démontrée par IRM de perfusion par marquage des spins

Case report

This healthy 52-year-old patient presented with mild language difficulties that brought him to our emergency department 2 h after symptom onset. Neurologically, there was no focal deficit of the extremities. The National Institute of Health (NIH) stroke scale score was 3. An emergency CT scan was performed and showed no hemorrhage, but a slight left frontal opercular hypodensity (Fig. 1). Based on this, it was decided to proceed to MRI, which was performed on a 3.0-T Magnetom Trio (Siemens; Erlangen, Germany). Arterial spin labeling (ASL) was carried out with a pulsed arterial spin labeling (PASL) sequence, using a QUIPS-II perfusion mode and the following parameters: 16 slices; voxel size: 3.4 × 3.4 × 6 mm; TA = 5/55 min; lambda = 0.9 mL/g; alpha = 95%; TE/TR/TI1/TI2/T1 (blood, 3T) = 15/5000/700/1800/1496.19 ms. Relative cerebral blood flow (relCBF) maps for ASL were calculated online by the MRI scanner, and offline for contrast-enhanced perfusion-weighted imaging (cePWI) using syngo perfusion (MR) software. Susceptibility-weighted imaging (SWI) was done using 3D acquisition with an in-plane resolution of 1 × 1 × 1 mm. cePWI was also acquired as well as diffusion-weighted imaging (DWI) with a 30°-direction scan. Initially, MRI showed a small focus of hyperintensity on DWI and hypoperfusion in the left middle cerebral artery (MCA) territory on gadolinium (Gd)-perfusion images (Fig. 1).

Angiography was performed using a transfemoral approach. The left carotid artery was supraselectively accessed and revealed an occlusion at the level of M2. Intra-arterial thrombolysis was carried out and was followed by revascularization of the vessel; indeed, the distal MCA branches were slightly hyperperfused afterwards (Fig. 2). On repeating MRI, the hypoperfusion was no longer evident. However, the ASL images revealed the presence of small cortical areas of hyperperfusion (Fig. 3). Clinically, the patient’s language problems were reversed and his Rankin score at discharge was 1.

We have shown here that ASL not only reproduces the imaging findings seen with Gd-based perfusion techniques, but can also demonstrate reperfusion. This is of great importance in cases where interventional neurovascular procedures have been undertaken. It is also of interest in the face of the challenges posed by nephrogenic systemic fibrosis (NFS).

Reperfusion is one of the aims of stroke therapy. Although neuroimaging in stroke has taken great strides in the last
Cerebral angiography shows an occlusion at the level of the M1 portion of the left middle cerebral artery (MCA) (A). This was supraselectively catheterized and thrombolysis was performed locally (B). Later angiography shows recanalization (C) as well as hyperperfusion in the distal MCA branches.

Angiographie cérébrale. Occlusion du segment M1 de l’artère cérébrale moyenne (ACM) gauche (A). Celui-ci a été cathétérisé sélectivement et une thrombolyse in situ a été réalisée (B). Le contrôle angiographique montre ensuite la recanalisation (C) ainsi que l’hyperperfusion dans les branches distales de l’ACM.

Arterial spin labeling before (A) and after (B) thrombolysis. A large perfusion deficit can be seen in the left middle cerebral artery territory (A). After recanalization (B), the deficit has regressed and additional cortical hyperperfusion can be seen (arrow).

Imagerie de perfusion par marquage des spins avant (A) et après (B) thrombolyse. Avant thrombolyse, il existe un déficit important de la perfusion dans le territoire de l’artère cérébrale moyenne gauche. Après recanalisation, celui-ci a régressé et il existe une hyperperfusion corticale additionnelle.

10 years, there remains much that needs to be improved [1,2]. In addition to demonstrating hypoperfusion and its reversibility, and the appearance—disappearance of lesions, imaging after interventions has been inadequate, as reperfusion is still not well characterized by MR techniques [3].

ASL is a promising new technique that may help to reveal the presence of small collaterals or cortical revascularization [4,5]. It is based on the possibility of performing MRI studies of brain perfusion (to obtain flow images) without the use of contrast agents. ASL can demonstrate collateral flow [5], and the presence or absence of collaterals plays an important role in the survival of tissue after stroke.

References


Optochiasmal apoplexy due to a cavernoma

A 25-year-old man was admitted with sudden right-sided vision loss. Neuro-ophthalmological findings included a temporal field defect in the right eye and blurred vision. There was a trace of optic nerve pallor with an increase in cup-to-disc ratio compared with the normal left side. The patient spontaneously recovered visual function within a week.

Ten days after onset, MRI of the anterior optic pathways demonstrated heterogeneous enlargement of the right half of the chiasm. The mass presented with central hypointensity on both T1- and T2-weighted images (WI) (Fig. 1A, B), without enhancement after gadolinium (Gd)-DTPA injection (not shown). This pattern suggested hemorrhage into an optic chiasm mass. The patient refused surgery despite being informed of the potential for recurrence and permanent vision loss.

Two years later, the patient again suffered an acute loss of eyesight. MRI (Fig. 1C–E) revealed the presence of an area of hyperintensity on T1-WI, with no enhancement after Gd-DTPA injection, and a peripheral hypointense rim on all sequences. Gradient-echo T2 (T2*) imaging was highly suggestive of a lesion containing hemosiderin with marked homogeneous signal loss. Surgical resection confirmed the diagnosis of cavernoma.

Whether sporadic or familial, cavernoma rarely occurs in the cranial nerve. Hemorrhage is a symptom in every case, and patients typically present with sudden-onset focal neurological deficit associated with the afflicted cranial nerve. Only 32 cases of optic tract cavernoma have been reported, including the present case [1—3]. In all of these cases, patients presented with either acute (18 cases) or subacute (14 cases) impairment of the visual field as a consequence of intratumoral hemorrhage. Along with the sudden visual loss and bitemporal hemianopsia, acute frontal or retro-orbital headache was present in 21 of the 32 cases. Fourteen patients had previous episodes of transient blurred vision, reflecting repeated episodes of bleeding. The differential diagnoses include optic chiasm apoplexy, and involvement or compression of the optic chiasm due to pituitary macroadenoma, meningioma, craniopharyngioma, glioma and metastases, or optic chiasm hematoma as a

![Figure 1](image-url)

**Figure 1** Initial coronal T1- (A) and T2-weighted images (WI) (B) and, taken two years later, coronal T1-WI (C), T2-WI (D) and gradient-echo T2 or T2* (E) images. The initial MRI examination showed a heterogeneous mass in the right side of the optic chiasm, with central hypointensity on both T1- and T2-WI. Two years later, there was a slight enlargement of the mass, with a hyperintense spot superiorly on T1-WI and a central hyperintensity on T2-WI with a peripheral hypointense rim. On coronal T2*, the mass appeared to be very hypointense, a consequence of the presence of hemosiderin (E). The evolution of the signal and pattern on T2* are highly suggestive of a bleeding lesion — in particular, a cavernoma.

Coupes coronales T1 (A) et T2 (B) initiales. Deux ans plus tard, coupes coronales T1 (C), T2 (D) et T2* (E). Sur l’IRM initiale, masse hétérogène de la partie droite du chiasma optique, avec portion centrale en hyposignal T1 et T2. Deux ans plus tard, augmentation discrète des dimensions de la masse, qui présente une portion supérieure spontanément en hypersignal T1, une portion centrale en hypersignal T2 avec un anneau périphérique en hyposignal. En T2*, la lésion apparaît en hyposignal marqué dans son ensemble, lié à la présence d’hémosidérine (E). L’évolution du signal et l’aspect en T2* suggèrent une lésion hémorragique et particulièrement un cavernome.