Increased relative cerebral blood volume (rCBV) in brain lymphoma

In clinical imaging, it can be difficult to distinguish primary central nervous system lymphoma (PCNSL) from high-grade glioma with conventional magnetic resonance imaging (MRI) [1,2]. However, the correct diagnosis of brain lymphoma is important because the treatment and the prognosis are different from other intracranial tumors [2,3]. The development of functional MRI has made it possible to differentiate cerebral lymphomas and high-grade tumors [4]. In several studies, lymphomas were hypovascular on perfusion MRI and their rCBV ratio were much lower than those of malignant tumors [1—4], mean value 1.68 [3]. We described two patients with cerebral lymphoma presented high value of rCBV.

The first patient was a 27-year-old woman with a TPCNSL and the second a 45-year-old women with a large B-cell lymphoma. The lesions presented a hypersignal in T2 and FLAIR sequences, which enhanced in post-gadolinium T1 sequence. In perfusion imaging, these lesions were hypervascularized with rCBV max of 698% (Fig. 1) and 501%, respectively (Figs. 2 and 3). The patients did not received corticoids prior to perfusion imaging. For the acquisition, a first bolus of gadolinium was injected before acquisition T2 sequence. This sequence is followed by a second bolus injection of gadolinium at a concentration of 0.1 mmol/kg and a rate of 10 mL/sec for the acquisition of the perfusion sequence with the following parameters: TR/TE = 1500 ms/35 ms, 24 slices, thickness 5 mm, 128 × 128 matrix, gap 1 mm.

Perfusion imaging has proven effective for the differentiation between brain lymphoma and high-grade glioma [5]. Specifically, low CBV allows identifying a large number of lymphomas [1,3,6]. This low rCBV is in agreement with histological data, which showed the absence of neovascularization in lymphomas [1,3,4,6]. Besides, lymphoma lesions usually enhance strongly due to disruption of the blood brain barrier [3,6].

The two patients described in our study presented a rCBV max in perfusion MRI superior to 500%. Our observation is in agreement with two publications [4,6], not using the double bolus injection.

Figure 1  A 45 years old woman with a diffuse large B-cell lymphoma. A. T1 weighted post-gadolinium sequence showing the strong enhancement of the lesions in the left frontoparietal lobes. B. On the perfusion parametric map using a colorimetric scale for estimation of the rCBV, these lesions were hypervascularized with rCBV five to seven times superior in the normal.
Figure 2  A 27 years old women with a T primary central nervous system lymphoma. A. T1 weighted post-gadolinium sequence showing a strongly enhancement lesion in right temporal lobe. B. On the perfusion parametric map using a colorimetric scale for estimation of the rCBV, this lesion was hypervascularized (rCBV of 501%).

Figure 3  A 27 years old women with a T primary central nervous system lymphoma. A. Perfusion parametric map showing a right temporal hypervascularized lesion (rCBV 310%). B. Graph showing rCBV curves for normal white matter (green curve) and for tumor lesion (purple curve).

injection method for the perfusion acquisition, indicating that 20–25% of the lymphomas presented an increased rCBV ratio comparable to that of high-grade gliomas. As similar results are obtained with or not the double injection method, we think that this last one not falsified values of rCBV. The values of rCBV can be falsified because of the important disruption of the BBB. Indeed, the accumulation of the contrast medium into the extravascular space during the first pass shortens the T1, leading to an increase of the signal intensity and simultaneously decreases the T2* effects by decreasing the contrast agent concentration gradient between intra- and extravascular space [1,3,6]. In principle, the double injection of contrast agent realized before the acquisition of the perfusion sequence should eliminate biases due to increased permeability of the BBB. Importantly, our observations have shown that a high rCBV can be observed in cases of lymphoma even when a double injection is performed. In such cases, the differentiation between lymphoma and high-grade gliomas remains a challenge, relying on careful investigation of conventional MR images.

Conflict of interest statement

No conflict of interest.

References


V. Dandois a,∗
B. De Coene b
P. Laloux b
C. Godfraind c
G. Cosnard d

a Department of Medical Imaging, MRI Unit, cliniques universitaires de Mont-Godinne, 1, avenue du Docteur-Gaston-Thérasse, 5530 Yvoir, Belgium
b Department of Neurology, cliniques universitaires de Mont-Godinne, 1, avenue du Docteur-Gaston-Thérasse, 5530 Yvoir, Belgium
c Department of Anatomopathology, université catholique de Louvain, cliniques universitaires Saint-Luc, 10, avenue Hippocrate, 1200 Brussels, Belgium
d Department of Medical Imaging, MRI Unit, université catholique de Louvain, cliniques universitaires Saint-Luc, 10, avenue Hippocrate, 1200 Brussels, Belgium

∗Corresponding author. Tel.: +32 71 59 68 94; fax: +32 71 59 68 94.
E-mail address: vinciane.dandois@skynet.be (V. Dandois).
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Spontaneous bilateral intrapetrous carotid dissection complicated by a ruptured dissecting aneurysm

A 49-year-old black woman, with a history of treated arterial hypertension, complained of pulsatile right eye pain, occipital headache and nausea for 1 week with normal clinical examination. Cranial computed tomography (CT) without injection was interpreted as normal. The next day, she consulted an ophthalmologist who disclosed a right Horner’s syndrome and homolateral ophthalmic nerve hypoesthesia. Axial T2 WI and magnetic resonance angiography (MRA) showed a hyperintense signal surrounding a narrowed right intrapetrous internal carotid without above stenosis, compatible with an intramural hematoma (Figs. 1 and 2); the controlateral intracranial internal carotid showed no irregularities or stenosis. Blood tests showed a white-cells count at 15,000 per millimeter cube, C-reactive protein level at 467 mg/l, antinuclear antibodies at 1/160; hemocultures, rheumatoid factor, complement investigations and antineutrophil cytoplasmic antibodies were negative. She was managed by intravenous heparin. The following day, she noticed diplopia and complained of worsening headache. Neurological examination revealed complete right oculomotor nerve palsy. A computerized tomography angiography (CTA) showed an intracavernous aneurysm of the right carotid artery without subarachnoid haemorrhage (SAH). The next day, a pulsatile right exophthalmia was disclosed, followed a few hours later by a generalized seizure and coma. Heparin treatment was stopped. CT showed massive SAH, a temporal haematoma, ventricular haemorrhage, and acute hydrocephalus (Fig. 3). A digital substracted angiography (DSA) disclosed an intracavernous aneurysm of the right carotid artery and bilateral narrowed intrapetrous internal carotids (Fig. 4) without abnormalities of their supraclinoid segment. An endovascular coiling of the aneurysm was performed. An external ventricular shunt was inserted and corticosteroid therapy initiated. Despite sedation, refractory intracranial hypertension due to diffuse brain oedema occurred, and she died 3 days later. Her family refused medical autopsy. Considering the association of bilateral narrowed arteries, an intracavernous aneurysm and a rapid clinical course, we suspected an intrapetrous carotid artery dissection (IPCAP).

Figure 1 AngioMR: intrapetrous narrowing of the right internal carotid artery, the left carotid showing a normal calibre.

Figure 2 Axial T WI: hyperintense intramural hematoma of the right intrapetrous carotid.