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Tips and traps in brain MRI: Applications to vascular disorders


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Abstract The French Society of Radiology’s guide to good use of medical imaging examinations recommends MRI as the first-line examination for exploring cerebrovascular events or disorders. This paper will discuss the main traps in the images when stroke is suspected and provide the technical tips or knowledge necessary for an optimal radiological report. © 2012 Éditions françaises de radiologie. Published by Elsevier Masson SAS. All rights reserved.

The radiologist’s daily routine confronts him with a certain number of difficulties when interpreting MRI of the brain. They may be due to an acquisition protocol inappropriate to the requirements of the examination, to the presence of artifacts or trap images, to poor knowledge of the signs of brain diseases or to non-systematic reading of the images. We shall not in this review consider the MRI signs of the main vascular brain diseases, since the reader can find them in a great many articles [1–3], but rather focus on the ‘traps’ most often encountered when undertaking MRI of such brain diseases. For each of these traps, practical solutions (tips or tricks-of-the-trade) will be offered. We will study, one by one, each of the sequences routinely used in these indications that comprise our ‘vascular protocol’: fluid attenuated inversion recovery (FLAIR), T2, time of flight (TOF) magnetic resonance angiography (MRA) of the circle of Willis, T1 gradient-echo scout images. We shall not deal here with the more recent techniques such as T2 perfusion [4,5], perfusion imaging by arterial spin labelling [6,7] or dynamic MRA sequences [8].

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The FLAIR sequence

This is the key sequence in exploring the brain and has largely replaced real T2 weighting. An inversion pulse allows the high signal intensity from the cerebrospinal fluid (CSF) to be nulled, offering excellent sensitivity and reading comfort for detecting parenchymal hyperintense signals close to fluid structures, the subarachnoid space or the vascular structures abnormally visible distal to a stenosis or arterial occlusion. The FLAIR sequence presents few difficulties for analysing vessels or parenchymal oedema (Fig. 1) but this is not the case for analysing the subarachnoid space. Flow artifacts are indeed common on FLAIR sequences (Fig. 2). They are not a problem if the patient has been referred for a first epilepsy seizure, or a suspected arterial ischaemic stroke (Fig. 2). On the other hand, they are the source of diagnostic difficulties in an examination for headaches. The tip is to use FLAIR sequences devoid of flow artifacts in MRI protocols for exploring headaches. For this there are two solutions (Fig. 3): 1/2D FLAIR, in which the inversion pulse is applied on either side of the slice of interest so that the protons entering the imaged slice due to the effect of CSF pulsatile flow have all been subjected to the inversion pulse; 2/3D FLAIR, in which there are no flow artifacts since the inversion pulse is applied to the entire cerebral volume [9]. Flow artifacts are eliminated at the expense of a slightly longer acquisition time (Fig. 3). It is then easy to detect pathological high signal intensity from the subarachnoid space (Fig. 4). In a FLAIR sequence, magnetic field inhomogeneities, particularly if metal is present, cause failure in CSF suppression. This can be the source of trap images that can mimic subarachnoid haemorrhage (Fig. 5). The tip is to check that there are no metal artifacts on the adjacent slices, ideally on a T2* sequence or, failing this, on diffusion images.

T2* sequence

The first trap is not having a T2* sequence at hand, which, remember, should be part of any MRI performed to explore

![Figure 1](image1.png) A FLAIR sequence and parenchymatous, subarachnoid or vascular abnormality: a: oedema in a patients suspected of having an arterial ischemic stroke the cortical parenchyma in the context of an arterial ischaemic stroke; b: diffuse hyperintense signals from the subarachnoid space: acute subarachnoid haemorrhage; c: high signal intensity from tubular structures in the left lateral sulcus indicating slow flow in the branches of the left middle cerebral artery. Note that the flowing vessels are not normally visible in a FLAIR sequence (right lateral sulcus).

![Figure 2](image2.png) Flow artifacts in FLAIR imaging: a: flow artifacts visible around the 4th and 3rd ventricles. They do not interfere with interpreting the images acquired because of a first epilepsy seizure; b: flow artifacts around the basal cisterns, the 4th ventricle and the lateral ventricles (arrows). These artifacts do not interfere with visualising the ischaemic lesion of the right hemisphere.
Tips and traps in neurological MRI: Applications to vascular disease

**Figure 3.** FLAIR sequence without flow artifacts. In a 2D FLAIR sequence it is advisable to apply the inversion pulse on either side of the slice of interest. The 3D FLAIR sequence where the inversion pulse is applied over the entire volume to be explored is devoid of artifacts.

**Figure 4.** Subarachnoid haemorrhage. Irrespective of whether we view the 2D or 3D FLAIR images, high signal intensity from the subarachnoid space is visible here, related to subarachnoid haemorrhage due to a ruptured aneurysm [thumbnail image: TOF MRA showing an aneurysm of the anterior communicating artery (arrow)].

headache or the sudden onset of a focal neurological deficit. The risk is indeed of not correctly recognising a recent intraparenchymal haematoma, which gives a high intensity signal on FLAIR imaging (Fig. 6). Any search for blood must therefore include a T2' sequence [10], whether related to a recent or earlier haemorrhage (Fig. 6). Spontaneous petechial haemorrhagic changes are common at the subacute stage of ischaemic accidents, or following fibrinolysis. Their abundance is overestimated in T2' [11] compared with a CT scan (Fig. 7). Even though petechial haemorrhagic changes can be impressive in T2' (Fig. 7) they do not imply immediate discontinuation of anticoagulants, if they are necessary, for example, for cardiac arrhythmia. These haemorrhages are classified based on the T2' images [11].

T2' is also useful for analysing the vascular structures, arteries and veins. When blood flow is normal, their signal is intermediate in T2'. A fresh intra-arterial thrombus can appear in T2' as a low intensity signal (the blooming effect) [12] (Fig. 8), but this can vary and depends on the constituents of the thrombus. Similarly, a thrombus in the venous sinuses (Fig. 9) or cortical veins will give a clear low intensity signal [13] (Fig. 10). If there is any doubt, a 3D sequence with injection of gadolinium should also be
Defective CSF nulling in FLAIR imaging. 37-year-old woman referred for unusual headaches. Hyperintense FLAIR signal from the bifrontal subarachnoid space as well as from the lateral ventricles. Symmetrical signal and anterior topography abnormalities (frontal horns) may be related to subarachnoid haemorrhage. Analysis of the subjacent slices shows a major magnetic susceptibility artefact due to the presence of dental material responsible for a loss of signal perfectly visible in T2*. 

Undertaken to confirm that the venous structures are not opacified (Fig. 9) [14]. While the T2* sequence is the best sequence for viewing cortical venous thromboses, dural calcification can produce traps when imaging the vertex (Fig. 10). The lack of a hypointense signal in T2* is also a factor thwarting most traps falsely appearing to indicate thrombophlebitis (Fig. 11): arachnoid granulations (Pachioni’s granulations), or a hypoplastic lateral sinus, may appear as a hyperintense FLAIR signal, but venous thrombosis can be eliminated due to the intermediate signal in
**Figure 7.** T2* and haemorrhagic change. Petechial haemorrhagic change in an ischaemic lesion visible in T2* images as a pronounced low intensity signal here in the deep middle cerebral artery territory, causing the high intensity signal in diffusion imaging to disappear. This hypointense T2* signal does not correspond to a real haematoma since there is no mass effect or haematoma on the CT scan. The CT scan shows pseudo-normalisation of the left lentiform nucleus because of the existence of haemorrhagic petechiae.

**Figure 8.** T2* and intra-arterial thrombus. The fresh thrombus appears in T2* images as a hypointense signal distal to the arterial occlusion. TOF MRA merged with the T2*-weighted sequence.

**Figure 9.** T2* and thrombophlebitis. The thrombus of the superior sagittal sinus appears as a clear hypointense T2* signal and is seen as defective opacification of the sinus following gadolinium injection.

**Figure 10.** Venous thrombosis or dural calcification? Low intensity curved T2* signals following the pathway of the cortical veins and the superior sagittal sinus related to venous thromboses. Dural calcifications are responsible for sometimes misleading hypointense T2* signals around the vertex.
Figure 11. False thrombophlebitis: a: hyperintense FLAIR/T2 signal from the left lateral sinus with no hypointense signal in T2* weighting, a hypointense signal after gadolinium injection, a non-obstructive rounded opacification defect in the sinus (pink arrow): arachnoid granulations (Pacchioni’s granulations); b: hypoplasia of the left lateral sinus responsible for a high intensity FLAIR signal with no low intensity signal in T2* with perfect opacification of the sinus following gadolinium injection (orange arrow). Rounded image in the right lateral sinus (pink arrow), intermediate signal in FLAIR and diffusion imaging, with no low signal in the T2*-weighted image; c: on the T1-weighted 3D sequence following gadolinium injection, coronal reconstruction shows a hernia of the occipital parenchyma within the sinus displacing the right lateral sinus.

Figure 12. Sequences sensitive to the effect of magnetic susceptibility. A comparison here, in the same patient, of 2D gradient-echo or T2* sequences, T2* echo planar imaging and a magnetic susceptibility weighted imaging (SWI) sequence. In this patient with suspected amyloid angiopathy with a right temporal haematoma, microhaemorrhages are best visible on the SWI sequence, as are also the deposits of haemosiderin (arrowhead).

T2* (Fig. 11). Several sequences are available that are sensitive to the effects of magnetic susceptibility (Fig. 12) [4]. Which should be chosen for a given indication? Conventional T2* or 2D gradient-echo T2* can last less than a minute, are good enough to exclude a haematoma in patients suspected of having had a stroke. The longer the echo time, the more marked will be the magnetic susceptibility effect. T2* echo planar imaging (EPI) lasts
Figure 13. Diffusion imaging and haemorrhage: a: intra-parenchymatous haematoma responsible for clear high signal intensity in a diffusion sequence that could be confused with an arterial ischaemic lesion. Only with T2* weighting can the diagnosis of a haematoma be confirmed (thumbnail image on left); b: subarachnoid haemorrhage as a hyperintense signal in FLAIR (thumbnail image) and diffusion imaging. The hyperintense signal in the diffusion-weighted image is solely due here to the presence of blood and not to the presence of ischaemia caused by vasospasm; c: venous thrombosis in the superior sagittal sinus responsible for a hyperintense signal in diffusion imaging.

Figure 14. Calculating the ADC. Faced with any FLAIR/T2 and diffusion imaging hyperintense signal, it is essential to calculate the ADC to discern: a: low-grade glial tumour, when confronted with this lesion shown as a high intensity signal in diffusion imaging with an elevated ADC. Note the increase in the choline peak within the lesion on spectroscopy; b: an ischaemic lesion as a hyperintense signal in FLAIR and diffusion imaging with a lowered ADC.
Figure 15. Calculating the ADC and a punctiform lesion. Patient suspected of a transient ischaemic accident in the right hemisphere. Punctiform lesion on diffusion imaging with no reduction in the ADC. The ADC being normal does not mean than an ischaemic lesion can be excluded if the lesion is small. On the follow-up MRI, confirmation of the ischaemic nature of the lesions, the signal from which is increasing.

Figure 16. Hyperintense diffusion signal from the internal capsule. Anisotropy of the corticospinal tract may be responsible for non-pathological, high signal intensity from the posterior limb of the internal capsule: a: three-directional diffusion sequence. High signal intensity from the posterior limb of the right internal capsule (arrow); b: diffusion sequence with 25 diffusion directions allowing an ischaemic lesion to be excluded here by correcting the artifact related to anisotropy.

Figure 17. Optimised diffusion imaging and transient deficit. Diffusion imaging may be normal if the origin of the transient deficit is vascular: a: three-directional diffusion imaging in 5 mm slices showing no high signal intensity; b: multi-directional diffusion imaging (12 directions) in 3 mm slices revealing a small left thalamic ischaemic lesion.
Figure 18. 3D TOF MRA and sudden onset deficit. 3D TOF MRA with segmentation of the anterior circulation (a) showing occlusion of the left middle cerebral artery. This MRA is obtained by positioning a single slab centred on the circle of Willis (b).

Figure 19. MRA and unusual sudden onset headaches: a: MRA showing an aneurysm of the top of the basilar artery (arrowhead). The recently ruptured aneurysm is located at the edge of the field of exploration (arrow); b: catheterisation angiography of the same patient confirming the top of the basilar artery aneurysm and a larger aneurysm at the origin of the postero-inferior cerebellar artery [PICA]; c: in another patient, an aneurysm of the pericallosal artery; d: illustration of the best position for looking for an aneurysm using 3D TOF MRA: two slabs from the foramen magnum to above the corpus callosum, so as not to fail to recognise aneurysms of the PICA. NB: two instances of the two slabs overlapping to avoid signal losses at the edge of the field of acquisition.
a few seconds and can be useful in agitated patients. This sequence is not solely reserved for study of cerebral perfusion or functional activation MRI. Finally, magnetic susceptibility sequences (SWI, duration 4–5 min) [4] are extremely sensitive to the effects of magnetic susceptibility: they detect more microbleeds and more leptomeningeal haemosiderin deposits and will be useful in looking for an amyloid angiopathy [15].

**Diffusion sequence**

The most serious error on the diffusion sequence is not recognising a parenchymal haematoma, which, if recent, appears as a hyperintense signal in diffusion-weighted images, like arterial ischaemia. It is therefore essential to exclude the presence of blood in T2' images before analysing the diffusion-weighted images (Fig. 13).
Subarachnoid haemorrhage will also produce a hyperintense signal in diffusion-weighted images (by a T2 effect). The trap lies in considering these hyperintense signals due to the presence of blood in the subarachnoid space as hyperintense signals from the cortex indicating ischaemia in the context of vasospasm (Fig. 13). The tip is to systematically look at the T2* image before analysing the diffusion image. Similarly, if there is a haemorrhagic lesion, diffusion imaging has little if any diagnostic value.

In order to avoid the diagnostic traps when confronted with a hyperintense T2/FLAIR signal and a hyperintense signal in diffusion imaging, a common situation, the tip is to calculate the apparent diffusion coefficient (ADC) [16] (Fig. 14). The range of diagnoses is indeed different depending whether it is low (ischaemia, tumours rich in cells, a pyogenic abscess, postcritical oedema, Creutzfeldt-Jakob disease) or elevated (perilesional vasogenic oedema, inflammation, gliosis, a necrotic lesion, etc.). The diagnostic range should be discussed depending on the entire MRI examination and the clinical context. Nevertheless, calculation of the ADC can also be a source of error when the lesions are small. If there are small hyperintense signals in diffusion weighted images, and there is no corresponding hyperintense FLAIR signal, a recent ischaemic lesion should not be excluded just because the ADC is normal (Fig. 15). The tip is not to take account of the ADC value if the hyperintense signal in the diffusion weighted image is small. Indeed, the ADC calculated from two images (\(b = 0–1000\text{s/mm}^2\)), in 5 to 6 mm slices, could be wrong due to the partial volume effect, distortion or tiny movements by the patient between acquisition at \(b = 0\) and \(b = 1000\text{s/mm}^2\). A more reliable ADC may be obtained by using intermediate \(b\) values, more diffusion directions or thinner slices. Another classic trap arises from the presence of 'physiological' hyperintense signals in the posterior limb of the internal capsule [16]. They are related to the anisotropic effects of the corticospinal tract, are usually discrete and bilateral (Fig. 16a). The error lies in attributing them to an ischaemic lesion. If there is proportional motor impairment, it is sometimes difficult to distinguish them from an ischaemic lacuna of the posterior limb of the capsule. In this case, the use of optimised diffusion sequences, with more than six diffusion directions, throws off any anisotropic effects and they are easier to interpret (Fig. 16). Finally, the fact that the diffusion images may be normal does not exclude the deficit being of vascular origin. Diffusion can occasionally be normal in the very first hours. If the patient is a candidate for fibrinolysis, therefore, it would be advisable to ensure that there is no occlusion using MRA, or T2* to exclude a thrombus, or no hypoperfusion, before leaning towards a pseudo-ischaemic deficit. Similarly, diffusion imaging is positive in only 40 to 50% of patients whose deficit is transient and regresses in less than 24 hours. Consequently, normal diffusion imaging does not exclude a transient deficit being of vascular origin.

If the MRI schedule is not too busy, an 'optimised' diffusion sequence clearly improves the sensitivity of MRI for detecting these small ischaemic hyperintense signals following transient neurological deficit (Fig. 17). There are several solutions for optimising this technique: increasing the number of diffusion directions, the \(b\) value, the number of excitations, or reducing slice thickness [17].

3D time-of-flight MRA of the circle of Willis

There are relatively few traps if the MRA cover is adapted to the indication. If the deficit is of sudden onset, short, focalised, single-slab MRA will be adequate for looking for an arterial occlusion (approximately 2 min at 1.5 T). On the other hand, this is inadequate for exploring patients with
headaches or for screening for intracranial aneurysm, since the aneurysms could be located at the origin of the posterior inferior cerebellar artery (PICA) or the pericallosal artery. The tip is to cover the length from the foramen magnum to above the corpus callosum with two slabs (Figs. 18 and 19). Finally, remember that signal asymmetry in time-of-flight MRA between the right and left internal carotid arteries should initiate a search for a cervical carotid stenosis, located beyond the field of exploration [18].

Scout T1-weighted images

One of the main traps is omitting to look at the images acquired, for example forgetting to analyse the sagittal scout T1 sequence. The tip is to systematically analyse the midline, paying particular attention to the following three regions: the corpus callosum/pineal gland, the foramen magnum and the pituitary region (Fig. 20). A type 1 Chiari malformation (Fig. 20) could produce vertigo or headaches. Any rounded mass in the pituitary region, regardless of its signal, should initiate checking to exclude an aneurysm, because of proximity to the arteries of the circle of Willis. More frequently, however, T1 hyperintense signals from the median line indicate lipomas, a diagnosis easily confirmed by a sequence with saturation of the fat signal (Fig. 20).

How to cope with an 'agitated' patient

A few tips are useful for dealing with agitated patients: avoid 3D sequences, which in general contain very many artifacts. 3D TOF MRA is still unavoidable but must be as short as possible. Echo-planar sequences (EPI) can be diffusion-, T2*, FLAIR- (Fig. 20) or T2-weighted, and last but a few seconds. Finally, there are sequences on offer not degraded by movements owing to over-sampling of the centre of the Fourier plane (PROPELLER, BLADE, Multivane, etc.) (Fig. 21). These tips will help you perform protocols of less than 5 min (Fig. 22), and produce imaging that can be interpreted from virtually all patients.

Conclusion

To conclude, with an MRI protocol adapted to the reason for the examination, that is to say, including T2* and FLAIR without artifacts (2D or 3D) for headaches, optimised diffusion imaging for transient deficit, single-slab TOF MRA for a suspected stroke, multislab for exploring headaches, a special protocol for ‘agitated’ patients, and systematic analysis of the brain structures, not forgetting the veins and the midline, calculation of the ADC if there is a hyperintense signal in T2-weighted and diffusion images, the main traps of brain MRI in vascular disorders will be avoided.

**TAKE-HOME MESSAGES**

- Protocol recommended if deficit is of sudden onset: sagittal T1, FLAIR, T2*, diffusion, TOF MRA of the circle of Willis with or without perfusion study, although not evoked in this document.
**Clinical case**

A 40-year-old woman with a history of left middle cerebral artery stroke. Sudden onset of aphasia. MRI 6 hours from the onset of the symptoms (Fig. 23).

**Questions**

1. What is the posterior lesion (asterisk)?
2. How should the diffusion signal and the ADC of the anterior lesion (arrowheads) be interpreted?
3. Use the various sequences to determine the nature of this image (arrowheads).
4. What is your final diagnosis?

**Answers**

1. The posterior lesion has a fluid signal in diffusion imaging (a low intensity diffusion signal, elevated ADC value), with peripheral gliosis in FLAIR, is located in the superficial territory of the left middle cerebral artery with attraction of the lateral ventricle. It corresponds to the sequela of the left middle cerebral artery stroke.
2. It is heterogeneous, with a lowered ADC in its centre. In practice, the presence of bleeding must always be sought before analysing the diffusion image. Here, in the presence of blood, the diffusion images may point wrongly towards arterial stroke, an abscess, etc.
3. This is a recent lobar haematoma, indicated by the T\textsuperscript{2} hypointense signal which indicates the presence of deoxy-haemoglobin. The underlying cause must be sought where there is any lobar haematoma.
4. There is a T\textsuperscript{2} hypointense signal from the superior sagittal sinus and the cortical veins (vertex) plus a filling defect of the superior sagittal sinus and the cortical veins following gadolinium injection. The diagnosis is therefore one of cerebral venous thrombosis with secondary lobar haematoma.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

**References**


