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# Fast FLAIR sequence for detecting major vascular abnormalities during the hyperacute phase of stroke: a comparison with MR angiography

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A. Peeters Department of Neurology, Cliniques Universitaires Saint-Luc, Brussels, Belgium enhancement in maximum-intensity projection (MIP) MR angiography (MRA). To compare these techniques, we examined 53 patients within 6 h of a stroke, using a standardised MRI protocol including fast-FLAIR and 3D time-of-flight TOF MR to detect vessel occlusion or reduced flow corresponding to the suspected ischaemic territory. Brain infarcts were confirmed on MRI after 1–5 days in 41 cases (77 %). The overall accuracy of 3D-TOF

**Abstract** In the hyperacute phase

of stroke, occluded vessels can be

seen as high signal on fast-FLAIR

images or as absence of flow-related

MRA was 68% and sensitivity, specificity, positive and negative predictive values were 67%, 71%, 87%, and 43% respectively. Values for the fast-FLAIR sequence were: 65%, 85%, 93% and 44%, with an overall accuracy of 70%. The fast-FLAIR sequence was thus able to show occluded vessels or reduced flow with about the same accuracy as 3D-TOF MRA and enabled better prediction of the ischaemic area.

Key words Brain ischaemia · Brain, infarct · Magnetic resonance imaging · Magnetic resonance angiography · Pulse sequences

### Introduction

Early identification of stroke, which allows early treatment, requires an imaging technique. Diffusion and perfusion-weighted images have shown early parenchymal signal change but are not widely available [1]. More widely available conventional sequences such as FLAIR also reveal signal change in ischaemic areas. Conventional MRI sequences also show flow-related phenomena [2–4]; abnormal high signal from vessels in an ischaemic area was reported on FLAIR sequences by Noguchi et al. [5], due to flow-related changes. Arterial occlusion is usually the major cause of stroke, and preliminary reports have shown that widely-available MR angiography (MRA) techniques can show major vascular occlusions [6–7]. Our aim was to compare the accuracy of fast-FLAIR sequences and MRA for identifying occlusion or reduced flow within major vessels and to determine if MRA is useful in the initial investigation of hyperacute stroke.

## **Materials and methods**

We looked at patients admitted to our stroke centre with a history and examination indicating stroke, on whom it was possible to carry out imaging within 6 h of the onset of symptoms. Exclusion criteria were haemorrhage on CT, MRI or contraindications to MRI. During a period of 20 months, 53 patients (33 women and 20 men, mean age 69 years, range 26–90 years) were prospectively studied. One patient was examined within the first h after the onset of symptoms, 13 during the second, 14 during the third, 15 during the fourth, 8 during the fifth and 2 during the sixth. Sedation was required in one third of patients.

All examinations were performed on the same 1.5-T system equipped with echo-planar capabilities. The protocol included a fast-FLAIR sequence and 3D time-of-fight (TOF) MRA. Two slightly different protocols were used, since the patients were included in two different prospective studies on perfusion-weighted imaging. The major difference in the fast-FLAIR sequence concerned the slice thickness and the number of slices: 36 slices for the first protocol, slice thickness 3.6 mm, no gap and the following parameters: TR 10002 TE 159ef TI 2200 ms, matrix  $256 \times 160,1$ acquisition, acquisition time 4.31 min; and for the second 24 slices,



**Fig.1** 51-year-old man presenting with right hemiparesis and aphasia, examined 2 h after onset. **a** MRA: the distal left middle cerebral arteries are poorly seen compared to the normal right side. **b** Initial fast-FLAIR axial image: high signal from the vessels is visible in the lateral fissure *(arrows)*. **c** Fast-FLAIR 1 day later, high signal is clearly seen within the infarct in the insula, putamen and posterior internal capsule

slice thickness 5 mm, gap 0.5 mm, TR 10002 TE 148ef TI 2200 ms acquisition time 4.31 min.

The 3D-TOF MRA protocol was: TR 36 TE 6.9 ms, flip angle  $15^{\circ}$ , field of view  $24 \times 18$  cm, matrix  $256 \times 224$ , with zero-filling interpolation (ZIP 512), 1 acquisition, 96 slices in three slabs, thickness 2 mm with 1 mm overlapping, flow compensation, superior saturation, magnetisation transfer, acquisition time 6.56 min.

The fast-FLAIR protocol was repeated 1–5 days after the onset of the stroke to determine whether a brain infarct, defined as high signal in the parenchyma from the infarcted area, had actually occurred.

Each MRI study was assessed independently by two neuroradiologists (GC, TD) who had been informed of the history. The initial fast-FLAIR images were analysed by each neuroradiologist to detect any abnormal high signal from the vessels in the suspected territory and any high abnormal parenchymal signal within the suspected ischaemic area. The MRA was also analysed by each neuroradiologist on a film printed at the time of the initial MRI study, containing six or eight views of the acquired volume in the three orthogonal planes and several oblique views; loss of high signal from the vessels of the suspected ischaemic area was considered a positive finding. At the end of the study, a consensus was reached between the two neuroradiologists.

#### Results

Of the 53 patients with suspected brain ischaemia, 12 patients did not show infarcts on the delayed MRI study. In 10 cases, the initial MRA and fast-FLAIR

images did not show any abnormal vessels. In one case MRA revealed known bilateral chronic internal carotid artery (ICA) occlusion; the fast-FLAIR images were normal. In another, both MRA and the initial fast-FLAIR images showed abnormal signal from vessels; there was also abnormal high signal from the parenchyma. This case was considered as a transient ischaemic attack (TIA) with complete anatomical and functional recovery.

In 42 patients a brain infarct was confirmed on the images obtained after 1–5 days. These cases were subdivided into five subgroups:

1. Both the initial fast-FLAIR images and MRA were normal in nine cases. The mean delay to MRI was 3.8 h, range 2–6 h. The mean volume of the infarct was 3 cc, with a 1–9 cc range. The arterial territories affected were right watershed anterior cerebral artery (ACA), middle cerebral artery (MCA) (1), ACA (1), vertebrobasilar (VBA) (5) and deep MCA (2). Abnormal parenchymal signal was detected on the initial fast-FLAIR images in 6 of 9 cases. In one case, MRA showed a left MCA aneurysm probably responsible for the ischaemic lesion.

2. The initial fast-FLAIR images and MRA both showed abnormal vessels in 21 cases (Fig. 1, 2, 3). The mean delay was 3.3 h, range 2–6 h. The mean infarct volume was 48 cc, with a 2–150 cc range. Abnormal parenchymal signal was seen initially in 8. The arterial territories affected were left MCA (8), right MCA (6), left ACA (1), left posterior cerebral artery (PCA) (2), left ICA (1) and VBA (3).

3. The fast-FLAIR images showed high signal from the vessels while MRA was normal in five cases. The mean delay was 3.8 h, range 3–4 h. The mean infarct volume was 24 cc, range 1–60 cc. The arterial territory affected was the left MCA in all 5 cases. In one patient with an insular infarct on follow-up, fast-FLAIR



**Fig.2** A 63-year-old man presenting with right hemiparesis and hemianopia, examined 2 h after onset. **a** MRA: no flow is visible in the distal posterior cerebral artery (*arrow*), only the P1 segment showing high signal. **b** Initial axial fast-FLAIR high signal from the left posterior cerebral artery is visible (*arrow*). **c** Fast-FLAIR 1 day later shows the high signal of a temporo-occipital infarct

showed high signal from the left MCA branches and MRA an abnormal right PCA. The fast-FLAIR images showed abnormal parenchymal high signal in two cases.

4. The initial fast-FLAIR images did not show abnormal high signal from the vessels, but MRA was ab-

**Fig. 3** A 64-year-old man presenting with right predominantly leg weakness, examined 2 h after onset. **a** MRA show right A1 hypoplasia or aplasia (*thin arrow*) and interruption of the left anterior cerebral artery (*thick arrow*). **b** Initial axial fast-FLAIR: high signal is visible in an anterior cerebral artery (*arrow*). **c** Fast-FLAIR 1 day later, at a higher level, showing high signal in a frontal greymatter infarct

normal in five cases. The mean delay was 2.8 h, range 2–4 h. The mean infarct volume was 6 cc, range 1–15 cc. The affected arterial territories were left MCA (3), left ICA (1), right PCA (1). Abnormal parenchymal signal was seen on initial fast-FLAIR images in two cases.

5. In one case, MRA showed abnormalities in the right PCA and left MCA territories and fast-FLAIR high signal from the vertebral and left PC arteries. The infarct volume was 50 cc, in the deep right MCA territory. The delay was 2 h. Abnormal high parenchymal signal was seen on the initial fast-FLAIR images.

Our results, in terms of sensitivity, specificity, positive and negative predictive value and overall accuracy of MRA and fast-FLAIR are summarised in Table 1. The accuracy of the fast-FLAIR sequence was assessed for each sign in three subgroups: parenchymal signal positive, vessel signal positive and both parenchymal and vessel signal positive.



 Table 1 Comparison of fast-FLAIR and 3D-TOF MRA in 53 cases

	Fast-FLAIR sequence Abnormal signal			MR angio-
	Paren- chyma	Vessels	Paren- chyma + vessel	graphy s
True positive	19	26	35	26
False positive	1	2	1	4
True negative	11	11	11	10
False negative	22	14	6	13
Sensitivity (%)	46	65	85	67
Specificity (%)	92	85	92	71
Positive predictive value (%)	95	93	97	87
Negative predictive value (%)	33	44	65	43
Overall accuracy (%)	56	70	87	68

#### Discussion

In this study of hyperacute stroke, fast-FLAIR imaging showed high signal from the parenchyma in 46% of cases (Figs.1c, 2c, 3c), a lower proportion than reported by Noguchi et al. [5]. The case in which a high parenchymal signal on the initial fast-FLAIR examination and positive diffusion weighted imaging within a deep right MCA territory were observed, and the final fast-FLAIR examination was normal, probably representing a TIA, was the only false positive fast-FLAIR result.

In the 41 patients in whom an infarct was demonstrated on delayed MRI, high signal was detected from the vessels of the VBA or ICA systems on the initial fast-FLAIR sequence in 26 cases (63%) (Figs.1b, 2b, 3b).

Because of the "time-of-flight" phenomenon, fastflowing blood is seen as a flow void. Normal vessels with fast flow therefore usually appear dark on FLAIR images as well as on conventional or fast-spin-echo T2weighted images. In stroke, the high signal from the vessels on FLAIR images is probably due to slowly moving or stationary blood. In our study, it was remarkable but not surprising that high signal from the vessels was more often detected on fast-FLAIR images when the infarct was large. High signal from stationary blood on FLAIR images was already described in subarachnoid haemorrhage [8-10] and acute subdural or parenchymal haematomas [11]. When arterial occlusion occurs, flow is reduced and signal from the vessel increases. Theoretically, high signal from a vessel on a FLAIR image could also be due to a thrombus, which could contain methaemoglobin. However, in hyperacute phase, transformation into thrombus has not yet occurred and there is therefore no methaemoglobin in the distal vessels at this stage. Nevertheless, maethemoglobin may be present in chronic thrombus in major arterial trunks, i.e., the ICA, M1 segment or VBA. Because methaemoglobin gives high signal, an occluded vessel may appear as high signal on a TOF image, mimicking blood flow and may thus cause a discrepancy between positive FLAIR images and negative MRA. In our study, a discrepancy was observed in five cases of reduced flow in the MCA without ICA occlusion. Even though methaemoglobin could theoretically cause high signal from the vessel, it is probably very rare in the hyperacute phase. This was also indicated by the fact that, the high signal from the vessels almost always disappeared during the next 1–5 day.

Since arterial occlusion is a major cause of stroke, angiography would logically be the main imaging technique employed. Early conventional angiography is usually performed with a view to intra-arterial thrombolysis and rarely for diagnosis. Given noninvasive character of MRA its use in the diagnosis of vascular occlusion was reported early [6-7]. On 3D-TOF MRA, vessels with normal, fast flow give high signal and those with reduced flow or thrombosis give with none (Figs. 1a, 2a, 3a). In our study, when a infarct was demonstrated on delayed MRI, MRA showed abnormal vessels in 63% of cases. Previous reports from Warach et al. [6], and Gillard et al. [7] indicated 67% and 50%, respectively. With conventional angiography, occluded arteries were demonstrated in 40–60% of cases [6]. It therefore seems that MRA can be used to identify maior vascular abnormalities.

The fast-FLAIR sequence proved to be more useful than MRA for predicting ischaemic areas. The overall accuracy for detection of abnormal flow of 3D-TOF MRA was lower than that of the FLAIR sequence when signal from the vessels was the only criterion. Except for sensitivity (67% vs 65%), all results were better with the fast-FLAIR sequence. Fast-FLAIR sequence and MRA both showed abnormal flow in the vessels of the ischaemic territory in 21 cases (50%), including ICA and VBA territories. In these cases, the mean infarct volume was 50 cc, but it varied from 20 cc to 150 cc. Both techniques were more often negative in cases of VBA, deep MCA or watershed ischaemia.

Discrepancies were observed. In five cases, the fast-FLAIR images were positive but MRA was negative. In all these cases, the abnormal vessels were in the superficial MCA territory and the infarct volumes were very variable. In one case, the findings of failure MRA to show an abnormal MCA was due to poor positioning of the images, because the symptoms suggested a VBA lesion and the slices did not therefore cover the entire MCA territory. MRA showed an abnormal right PCA (the infarct was in the MCA territory). In another case, MRA retrospectively showed an abnormal MCA but needed further segmentation and comparison between left and right MCA in a sagittal reformat to detect the abnormal vessel. In three cases, high signal from the vessels was clearly present on the FLAIR images and MRA clearly showed normal flow. This discrepancy may be due to different sensitivity to flowing blood.

In five cases MRA was positive and the fast-FLAIR images negative. In two of these, the FLAIR images could retrospectively be considered positive, while in one the MRA could retrospectively be considered negative. In the two remaining cases, the discrepancy was probably due to a chronically occluded artery. In one case, there was a complete ICA occlusion and only a 1 cc final infarct. In the other, there was a distal M1 occlusion with visibility of the distal MCA vessels, which gave intermediate signal.

In addition to being more accurate in showing abnormal flow, fast-FLAIR images also showed abnormal high signal from the ischaemic area and/or mass effect, with much higher accuracy than MRA. The latter appeared useful in only three of 53 cases (6%) where initial fast-FLAIR showed high signal from neither the vessels nor the parenchymal of the ischaemic area. In addition, high signal from abnormal vessels was seen easily on fast-FLAIR images while it was sometimes difficult to detect on MRA and the images often had to be reformated and segmented. The time required to obtain similar results was shorter with the fast-FLAIR sequence than with 3D-TOF MRA, taking in to account acquisition time (4.31/6.56 min), data transfer and reformatting.

We therefore recommended that this MRA technique not be used routinely for initial diagnosis of stroke, and that MRA be performed only be when no high signal from the vessels of the suspected arterial territory on is detected FLAIR images.

## References

- Sorensen AG, Buonanno FS, Gonzalez RG, et al (1996) Hyperacute stroke: evaluation with combined multisection diffusion-weighted and hemodynamically weighted echo-planar MR imaging. Radiology 199: 391–401
- 2. Yuh WTC, Crain MR, Loes DJ, et al (1991) MR imaging of cerebral ischemia. AJNR 12: 621–629
- 3. Biller J, Yuh WTC, Mitchell GW, et al (1988) Early diagnosis of basilar artery occlusion using magnetic resonance imaging. Stroke 19: 297–306
- 4. Kate BH, Quencer RM, Kaplan JO, et al (1989) MR imaging of intracranial carotid occlusion. AJNR 10: 345–350
- Noguchi K, Ogawa T, Inugami A, et al (1997) MRO of acute cerebral infarction: a comparison of FLAIR and T2weighted fast spin-echo imaging. Neuroradiology 39: 406–410
- Warach S, Li W, Ronthal M, Edelman R (1992) Acute cerebral ischemia: evaluation with dynamic contrast-enhanced MR imaging and MR angiography. Radiology 182: 41–47
- 7. Gillard JH, Oliverio PJ, Barker PB, Oppenheimer SM, Bryan N (1997) MR angiography in acute cerebral ischemia of the anterior circulation: a preliminary report. AJNR 18: 343–350
- Noguchi K, Ogawa T, Inugami A, et al (1995) Acute subarachnoid hemorrhage: MR imaging with fluid-attenuated inversion recovery pulse sequences. Radiology 196: 773–777
- Mikami T, Saito K, Okuyama T, Sakamoto Y, Takahashi A, Shibata K (1996) FLAIR images of subarachnoid hemorrhage. No Shinkei Geka 24: 1087–1092
- 10. Tsurushima H, Meguro K, Wada M, et al (1996) FLAIR images of patients with head injuries. No Shinkei Geka 94: 891–895
- Ashikaga R, Araki Y, Ishida O (1997) MRI of head injury using FLAIR. Neuroradiology 39: 239–242