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High signal in cerebrospinal fluid mimicking subarachnoid haemorrhage on FLAIR following acute stroke and intravenous contrast medium

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Abstract We describe five cases of high signal in the cerebrospinal fluid (CSF) on fast-FLAIR images 24–48 h after onset of stroke. All the patients had undergone perfusion-weighted MRI within 6 h of the onset of the symptoms. The CSF was far brighter than the cortical gyri. The high signal was diffusely around both cerebral hemispheres in two cases and around one hemisphere in two others; it was focal, around the acute ischaemic lesion, in one. CT was normal in all cases. The CSF high signal was transient, decreasing in extent and intensity with time and resolving completely within 3–6 days. It was not associated with worsening of the clinical state or poor outcome. Our explanation of this phenomena is hypothetical: we speculate that it could be due to disruption of the blood-brain barrier resulting in leakage of protein, gadolinium chelates, or both in to the subarachnoid space. It should not be confused with subarachnoid haemorrhage.

Key words Magnetic resonance imaging · Pulse sequences · Stroke · Fluid, cerebrospinal

Introduction

Fluid-attenuated inversion recovery (FLAIR) imaging is currently used in a wide range of cerebral pathology and said to be of particular value in imaging stroke [1, 2]. On a FLAIR sequence, the cerebrospinal fluid (CSF) signal is nulled, allowing better analysis of adjacent tissue. High-signal CSF on FLAIR has been demonstrated in cases of subarachnoid haemorrhage (SAH) [3, 4], acute meningitis [5] and superior sagittal sinus thrombosis [6].

We report five cases of high-signal CSF on fast FLAIR on the 1 st or 2 nd days after a stroke in patients who had undergone perfusion-weighted MRI.

Materials and methods

We reviewed five patients in whom high signal had been observed in the CSF on fast FLAIR images 24–48 h after the onset of stroke. All had been investigated within the first 6 h of their neurological episode using an MRI protocol including, fast FLAIR and echo-planar (EPI) perfusion-weighted imaging (PWI). PWI was performed with a bolus injection of gadopentetate dimeglumine (0.1 mmol/kg, 10 ml/s). All examinations were performed on the same 1.5-T unit. The final diagnosis was ischaemic brain damage in all cases. All the ischaemic brain lesions were seen on diffusion and perfusion-weighted images in the acute phase (Table 1).

The fast FLAIR parameters were TR 10,002, TE 148, TI, 2,200 ms; echo train length, 16; one excitation; field of view 24 cm; matrix 256 × 160; slice thickness 5.0 mm, gap 0.5 mm; readout bandwidth ± 31.2 kHz. Nonselective inversion (3 slices width) was used. There was no metallic susceptibility artefact in any patient.

The intensity, site and extent of the CSF high signal were recorded, as were the clinical status and outcome. The CSF signal was compared with a contemporaneous CT in all cases, T1-weighted (TR 640, TE 10 ms) images in three and analysis of CSF obtained by lumbar puncture in two. CSF signal intensity over time was studied in four patients by repeating the fast FLAIR sequence on day 3 in four cases and on day 6 in two.

Table 1 Acute ischaemic lesions and CSF high signal on day 1 fast-FLAIR images (24–48 h after onset of symptoms)

Case	Age (years)	Sex	Ischaemic arterial territory	Volume of ischaemic lesion on day 1 (cc)	High signal in subarachnoid space on day 1
1	83	M	Left middle cerebral	1	Left convexity
2	78	F	Left middle cerebral	1	Focal next to ischaemic lesion
3	77	F	Right middle cerebral	12	Right convexity
4	60	M	Left anterior cerebral	26	Both convexities
5	81	M	Left posterior cerebral	72	Both convexities

Results

In all five patients, the CSF signal intensity was normal on the fast FLAIR images obtained within the first 6 h of the stroke (Fig. 1 a, 2 a). CSF signal intensity on the fast FLAIR images 24–48 h after stroke onset was far higher than that of cerebral cortex in all cases (Fig. 1 b, 2 b). In two patients, the high signal was diffuse, over both cerebral hemispheres (Fig. 2 b). In another two it was limited to the side of the acute ischaemic lesion, but was far more extensive than the brain lesion (Fig. 1 b). In one case focal, next to the ischaemic lesion. The cerebral lesions were cortical in all patients.

No abnormality suggestive of haemorrhage in the subarachnoid space was seen on a CT study performed at the same time as the fast FLAIR in any patient (Fig. 1 c, 2 c). The T1-weighted spin echo images in two cases did not show any CSF abnormality. In one case a slight increase in signal intensity was seen in the subarachnoid space in the same position as on fast FLAIR. CSF samples in two cases did not show blood cells; in case 4, there was a slightly increased protein concentration (69 mg/dl; normal range 15–45 mg/dl), but in case 3 protein was normal (26 mg/dl). No patient had suggestive symptoms (headache, meningism, etc.) or worsening of clinical status. High signal in the CSF intensity decreased progressively in extent and intensity with time and disappeared completely within 3 days in cases 2 and 4 and 6 days in cases 1 and 3.

Discussion

On FLAIR images, high signal from the CSF may result from artefacts. Ghosting artefacts are due to inflow of non-nulled CSF into a section with a high CSF flow-rate [1, 7] and are seen mainly in the basal cisterns. They are unusual over the convexities, where we observed high signal. These artefacts are reduced with non section-selective inversion pulses, as used in our sequence.

Artefacts can also be caused by metal, which creates field heterogeneity within the slice, resulting in incomplete nulling of CSF by the slice-selection inversion pulse [1]. In our patients, EPI spin-echo DWI and gradient-echo PWI sequences very sensitive to the presence of metal had been performed and helped us to exclude metallic artefacts.

Moreover, artefacts were highly unlikely in our patients since the signal abnormalities decreased over time and resolved.

FLAIR is known to be sensitive to detect acute SAH [3, 4], which can appear in stroke, especially with ischaemic cortical lesions [8]. In our patients no evidence of SAH was found on CT or in the CSF samples available.

Our first hypothesis stems from the fact that FLAIR is sensitive to increase in protein concentration in the CSF. A high protein level decreases the T1 relaxation time of the CSF and causes an offset of the null inversion time, so that CSF appears of high intensity. Protein concentration thresholds depending on the effective echo time of the FLAIR sequence have been demonstrated experimentally [5]. Brain ischaemia alters the blood-brain barrier, causing an increase in vascular permeability to protein. Elevation of CSF protein concentration has been reported in about half of patients with stroke [9–11]. Although one of our patients showed a slight increase in protein concentration, CSF protein was normal in the other. Increase in the CSF signal intensity on FLAIR images, associated with an elevation of protein concentration in CSF samples, has been reported in a case of superior sagittal sinus thrombosis [6]; the patient had received an intravenous injection of gadopentetate dimeglumine 8 h before the FLAIR sequence.

The second potential explanation takes into account the intravenous administration gadolinium chelate in the first 6 h of the stroke. This paramagnetic agent could be responsible for a delayed change in the signal intensity of the meninges or subarachnoid space on the 24–48 h FLAIR images. Contrast enhancement of the meninges adjacent to an early cerebral infarct has been described from days 2 to 6 after the onset [12]. It has been observed on T1-weighted images with or without magnetisation transfer immediately after intravenous injection of contrast medium [12, 13]. To the best of our knowledge, however, there has been no report of such meningeal enhancement seen with FLAIR. No data are available on delayed FLAIR studies in stroke performed several hours or days after injection. Rupture of

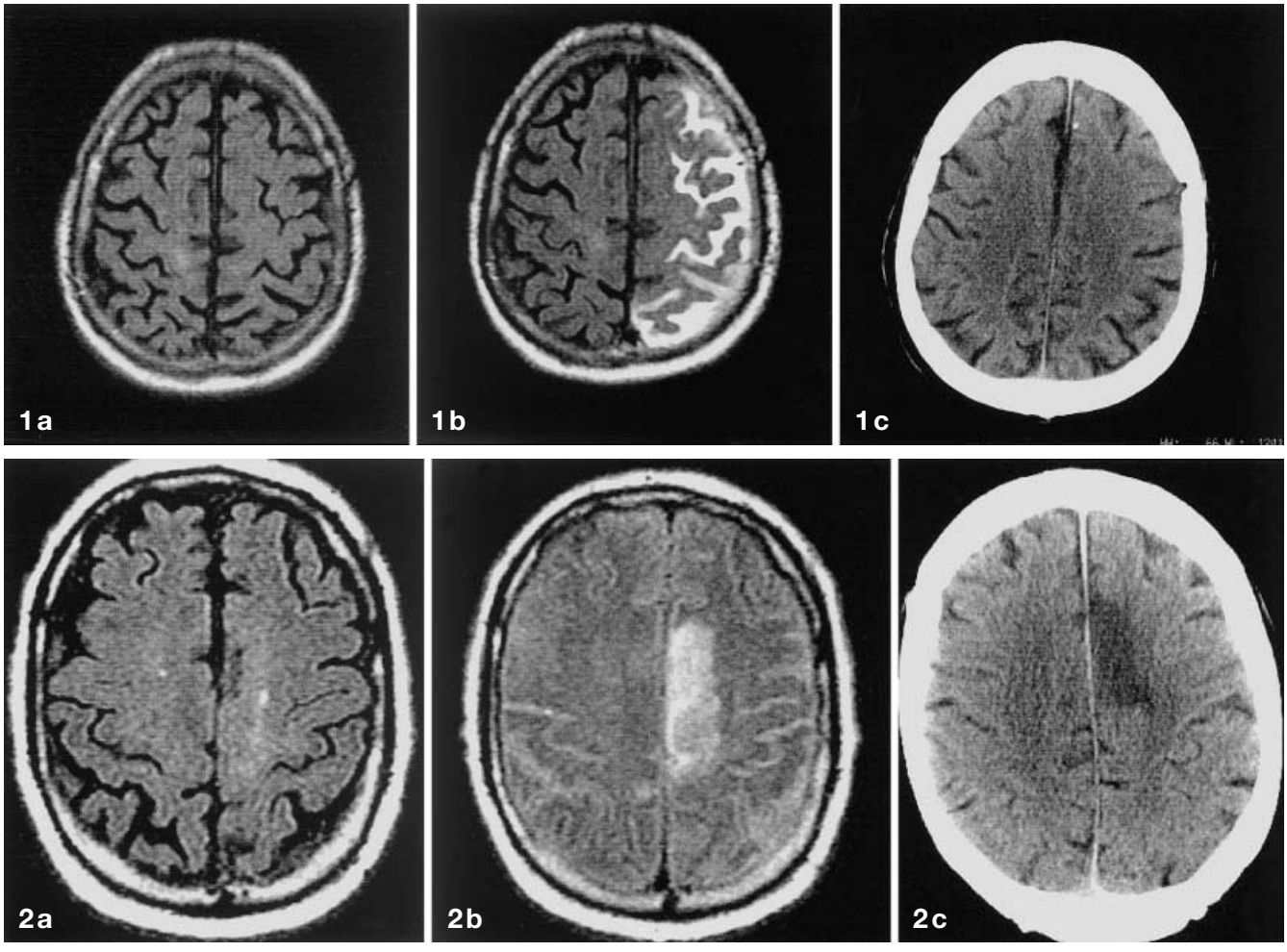

Fig. 1 a–c *Case 1. Acute ischaemic lesion (1 cc) in the left middle cerebral artery territory.* MRI on day 0 **a** and day 2 **b** axial fast FLAIR images. On day 0, 2 h after the onset of stroke, the subarachnoid space (SAS) gives normal signal. High signal is seen in the subarachnoid space over the left convexity on day 2. **c** No evidence of subarachnoid haemorrhage (SAH) is seen on CT on day 2. Microcalcification is seen in the left frontal lobe

Fig. 2 a–c *Case 4. Acute ischaemic lesion (20 cc) in the left anterior cerebral artery territory.* MRI on day 0 **a** and day 1 **b,** axial fast FLAIR images. On day 0, 2 h after the onset of stroke, the subarachnoid space shows normal low signal; only focal chronic lesions were shown in the centrum ovale. On day 1, an ischaemic area is demonstrated in the left anterior cerebral artery territory and high signal is diffusely present in the SAS. On the contemporaraneous CT **c,** the ischaemic brain appears as low density and there is no evidence of SAH

the blood-brain barrier caused by infarction might lead to accumulation of gadolinium chelates in the subarachnoid space in sufficient concentration to induce increase signal enhancement on FLAIR images. Fast FLAIR has recently been demonstrated to be more sensitive than T1-weighted imaging to low concentrations of gadolinium and to be more efficient in detecting superficial abnormalities such as meningeal disease [14].

Further investigation is warranted to determine the role of protein levels and gadolinium chelates in this phenomenon.

References

1. Adams JG, Melhem ER (1999) Clinical usefulness of T2-weighted fluid-attenuated inversion recovery MR imaging of the CNS. AJR 172: 529–536
2. Alexander JA, Sheppard S, Davis PC, Salverde P (1996) Adult cerebrovascular disease: role of modified rapid fluid-attenuated inversion recovery sequences. AJNR 17: 1507–1513
3. Noguchi K, Ogawa T, Inugami A, et al (1995) Acute subarachnoid hemorrhage: diagnosis with fluid-attenuated inversion-recovery MR imaging. Radiology 196: 773–777
4. Noguchi K, Ogawa T, Hikaru S, et al (1997) Subacute and chronic subarachnoid hemorrhage: MR imaging with fluid-attenuated inversion-recovery pulse sequences. Radiology 203: 257–262
5. Melhem ER, Jara H, Eustace S (1997) Fluid-attenuated inversion-recovery MR imaging: identification of protein concentration thresholds for CSF hyperintensity. AJR 169: 859–862
6. Bozzao A, Bastianello S, Bozzao L (1997) Superior sagittal sinus thrombosis with high-signal-intensity CSF mimicking subarachnoid hemorrhage on MR FLAIR images. AJR 169: 1183–1184
7. De Coene B, Hajnal JV, Gatehouse P, et al (1992) MR of the brain using fluid-attenuated inversion recovery pulse sequences. AJNR 13: 1555–1564
8. Lee MC, Heaney LM, Jacobson RL, Klassen AC (1975) Cerebrospinal fluid in cerebral hemorrhage and infarction. Stroke 6: 638–641
9. Al-Kassab S, Skyhoj Olsen T, Skriver EB (1981) Blood-brain barrier integrity in patients with cerebral infarction investigated by computed tomography and serum-CF-albumin. Acta Neurol Scand 64: 438–445
10. Hornig CR, Busse O, Dorndorf W, Kaps M (1983) Changes in CSF blood-brain barrier parameters in ischemic cerebral infarction. J Neurol 229: 11–16
11. Niebroj-Dbosz I, Mariam AG, Lakasiuk M, Rafalowska J (1992) Blood-cerebrospinal fluid barrier integrity in cerebral infarction. Neuropathol Pol 30: 29–33
12. Elster AD, Moody DM (1990) Early cerebral infarction: gadopentetate dimeglumine enhancement. Radiology 177: 627–632
13. Mathews VP, King JC, Elster AD, Hemilton CA (1994) Cerebral infarction: effects of dose and magnetization transfer saturation at gadolinium-enhanced MR imaging. Radiology 190: 547–552
14. Mathews VP, Caldemeyer KS, Lowe MJ, Greenspan SL, Weber DM, Ulmer JL (1999) Brain: gadolinium-enhanced fast fluid attenuated inversion-recovery MR imaging. Radiology 211: 257–263