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Cortical laminar necrosis in brain infarcts: chronological changes on MRI

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Abstract We studied the MRI characteristics of cortical laminar necrosis in ischaemic stroke. We reviewed 13 patients with cortical laminar high signal on T1-weighted images to analyse the chronological changes in signal intensity and contrast enhancement. High-density cortical lesions began to appear on T1-weighted images about 2 weeks after the ictus. At 1–2 months they were prominent. They began to fade from 3 months but could be seen up to 11 months. These cortical lesions showed isointensity or high intensity on T2-weighted images and did not show low intensity at any stage. Contrast enhancement of the laminar lesions was prominent at 1–2 months and became less apparent from 3 months, but could be seen up to 8 months.

Key words Brain infarcts · Cortical laminar necrosis · Magnetic resonance imaging

Introduction

Cortical laminar necrosis occurs as a consequence of oxygen or glucose depletion, as in anoxia, hypoglycaemia, status epilepticus, and ischaemic stroke [1]. MRI is useful for detection and characterisation of brain infarcts [2–4], but there have been few reports on MRI of cortical laminar necrosis caused by ischaemic stroke [5, 6]. We investigated patients with cortical laminar necrosis with special reference to chronological changes of signal intensity and contrast enhancement of the lesions.

Patients and methods

Among 50 patients diagnosed as having brain infarcts on clinical features and CT and/or MRI, we found 13 with laminar high-intensity lesions on MRI. Patients with calcification or haemorrhagic infarcts on CT were excluded. All patients underwent MRI with T1- and T2-weighted images using a 1.0- or 1.5-T imager. There were 2 women and 11 men, aged 55–77 years, mean 66.7 years. We analysed the MR images of these patients retrospectively with regard to chronological changes of signal intensity and contrast enhancement. MRI was carried out on 33 occasions in total, with a mean of 2.5 per patient, from 1 day to 11 months after the ictus; the day of the ictus was termed day 0.

In 8 patients contrast medium was given on 13 occasions: Gadolinium diethylenetriamine-pentaacetic acid (Gd-DTPA) 0.1 mmol/kg. We examined 6 patients with proton-density images on 8 occasions. Cerebral angiography was carried out in 9 of the younger patients, from day 0 (patient 2) to 3 months.

MRI parameters for T1-weighted spin-echo images were repetition time (TR) 510–582 ms, echo time (TE) 14–15 ms, 2 excitations, and for T2-weighted images TR 2015–2200, TE 80 ms, 1 excitation (conventional spin-echo) or TR 5000, TE 90 ms, echo train 7, 1 excitation (fast spin-echo) at 1 T and TR 3830, TE 110 ms, echo train 13, 1 excitation (fast spin-echo) at 1.5 T. For proton-density images we used TR 2200–2500, TE 20 ms, 1 excitation (spin-echo). Slice thickness was 6–7 mm, field of view 23 cm; axial images with a 192–256 × 256 matrix were obtained. Signal intensity was assessed as: low, isointense, slightly high, high, and very high compared to the relevant normal brain structures.

Results

Clinical and MRI data are summarised in Table 1.

Figure 1 illustrates the chronological changes in signal intensity of the cortical lesions on T1-weighted images. On day 1, the cortical lesion gave slightly low signal on T1-weighted and high signal on T2-weighted images (Fig. 2a, b), which subsequently became high in-

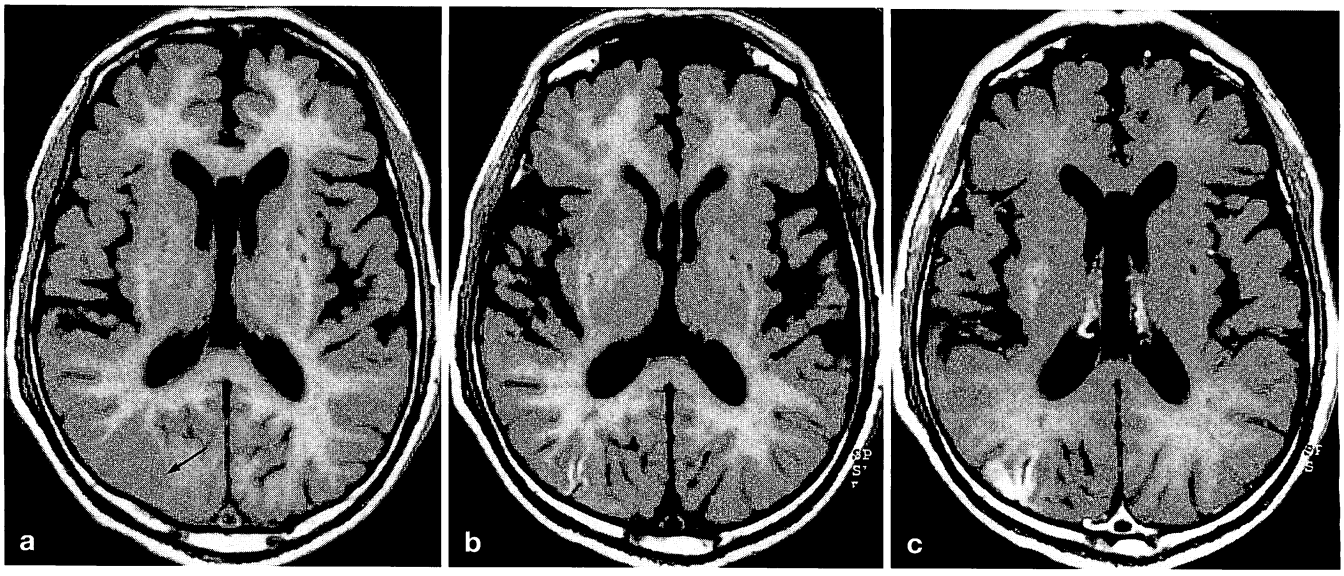

Fig. 3a–c Patient 7. A 69-year-old man with hemianopia. **a** On a T1-weighted image at 2 weeks from ictus, there is no high-intensity lesion, but effacement of a cortical sulcus is noted *(arrow)*. **b** At 2 months, a T1-weighted image does demonstrate a high-intensity laminar lesion, which shows contrast enhancement (**c**)

Figure 6 illustrates the chronological changes in contrast enhancement of the cortical lesions. Contrast enhancement was prominent 1–2 months (Figs. 3c, 4b) (patients 4, 7), and was observed up to 8 months (patient 8); however, it was not observed at 3 months in patients 4 and 9. In patient 4, contrast enhancement disappeared at 3 months, when T1-weighted images disclosed a slightly high intensity laminar lesion (Fig. 4b). In patient 8, T1-weighted images did not show high intensity at 6 or 8 months but there was contrast enhancement. High-intensity lesions on T1-weighted images at 1–5 months showed contrast enhancement in patients 1, 4 and 7 (Figs. 3c, 4b).

Discussion

Chronic brain infarcts are typically seen as low-intensity lesions on T1-weighted and high-intensity lesions on T2-weighted images due to prolonged T1 and T2 values [3, 4]. In some infarcts, high-intensity lesions are observed on T1-weighted images. Haemorrhagic infarcts show characteristic changes of the signal intensity, similar to those of haemorrhage, due to deoxyhaemoglobin, methohaemoglobin and haemosiderin [7, 8]. Petechial haemorrhage may occur in cortical infarcts but cannot explain the high-intensity laminar lesions in all patients [9]. High intensity on T1-weighted images (T1 shortening) can be due to methaemoglobin, mucin, high protein concentration, lipid or cholesterol, calcification and cortical laminar necrosis [6]. In ischaemic stroke, high-intensity laminar lesions can be cortical laminar necrosis, haemorrhagic infarcts (microhaemorrhage), or a combination of the two.

The grey matter has six layers. The third is the most vulnerable to depletion of oxygen and glucose. When a relatively mild ischaemic or hypoglycaemic insult occurs, the vulnerable layers are selectively injured (selective neuronal vulnerability) [1].

The cortical laminar necrosis, seen as a laminar high-signal lesion on T1-weighted images, was first described by Sawada et al. [10] in a patient with anoxic encephalopathy. The cortical lesion is usually in the watershed region in this condition [9]. Cortical laminar necrosis is also reported in ischaemic stroke [5, 6]. Although Nabatame et al. [5] thought the high intensity on T1-weighted images was due to methohaemoglobin in haemorrhagic tissue, pathological study revealed no haemorrhage [6, 11]. We think many of their patients had cortical laminar necrosis with haemorrhagic infarcts or simply haemorrhagic infarcts in the subacute stage, as they speculated.

Takahashi et al. [9] reported a patient with a cortical laminar lesion showing low intensity on T2-weighted images in the chronic stage, which they thought due to haemosiderin. We believe there were no cases of haemorrhagic infarct in our series because we excluded patients with CT-documented haemorrhagic infarcts, because T2-weighted images, even at high magnetic field (1.0–1.5 T), did not demonstrate low-intensity lesions due to haemosiderin in the chronic stage, and because high-intensity laminar lesions were demonstrated for up to 6–11 months which were not consistent with haemorrhagic infarcts [8]. We used both conventional and fast spin-echo T2-weighted images. The latter is reported to be slightly less sensitive to magnetic susceptibility effect, and thus slightly less sensitive to haemosiderin [12].

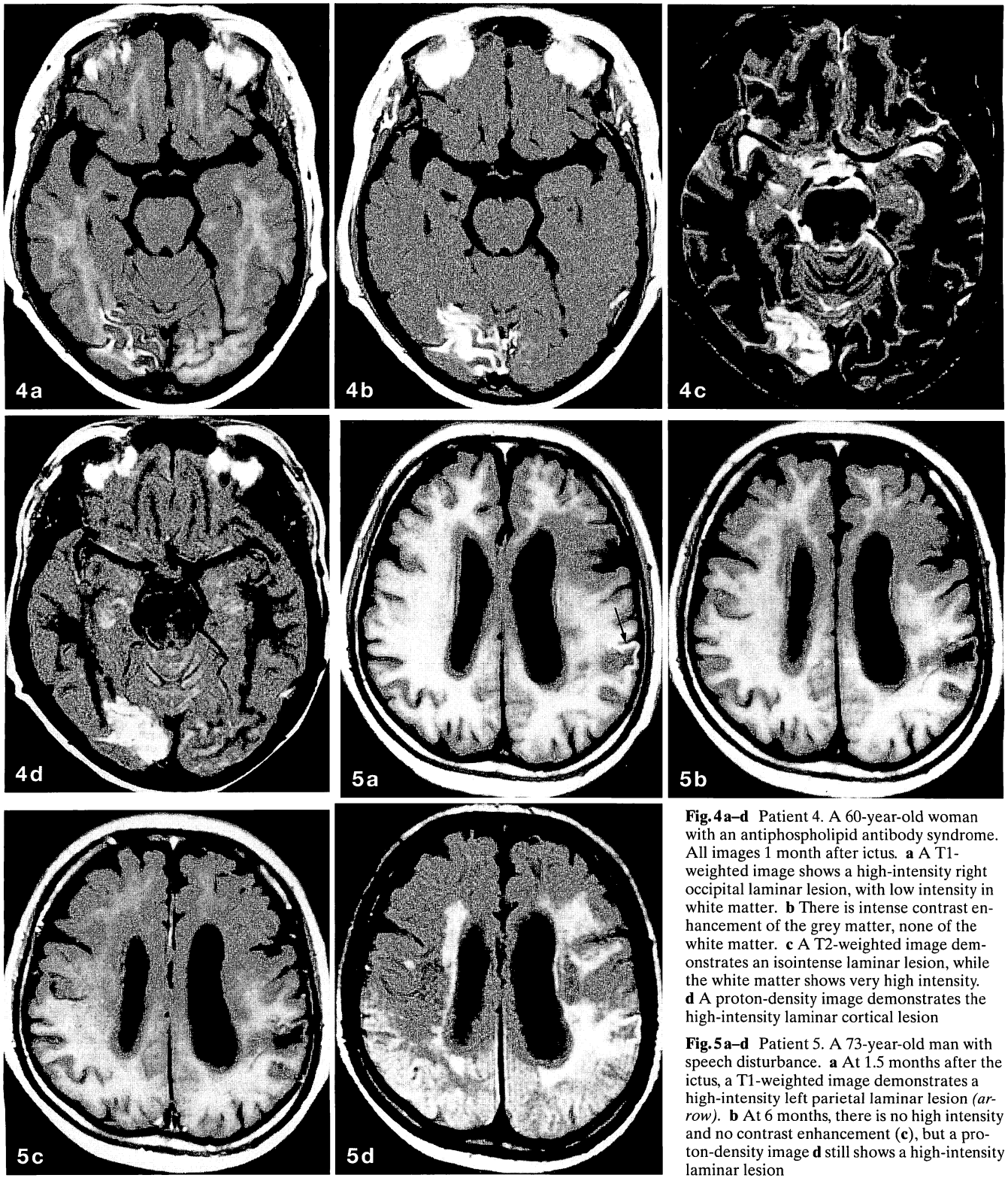

Fig. 4a–d Patient 4. A 60-year-old woman with an antiphospholipid antibody syndrome. All images 1 month after ictus. **a** A T1-weighted image shows a high-intensity right occipital laminar lesion, with low intensity in white matter. **b** There is intense contrast enhancement of the grey matter, none of the white matter. **c** A T2-weighted image demonstrates an isointense laminar lesion, while the white matter shows very high intensity. **d** A proton-density image demonstrates the high-intensity laminar cortical lesion

Fig. 5a–d Patient 5. A 73-year-old man with speech disturbance. **a** At 1.5 months after the ictus, a T1-weighted image demonstrates a high-intensity left parietal laminar lesion *(arrow)*. **b** At 6 months, there is no high intensity and no contrast enhancement (**c**), but a proton-density image **d** still shows a high-intensity laminar lesion

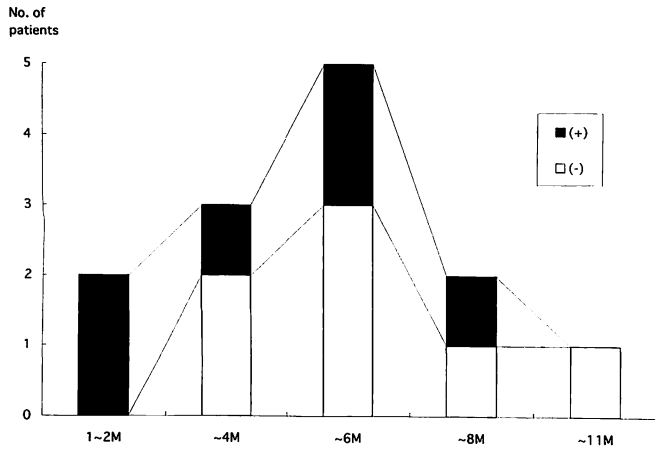

Fig. 6 Chronological changes in contrast enhancement of cortical laminar necrosis. *(+)* contrast enhancement *(–)* no enhancement

However, we found no low-intensity haemosiderin in any patient, even on conventional spin-echo T2-weighted images.

Boyko et al. [6] described cortical laminar necrosis in brain infarcts as linear high signal based on the cortical surface and becoming less intense over time (months). In hypoxic brain damage, T1-weighted images revealed a laminar high-intensity lesion in the cortex in the late subacute stage (21–28 days) which tended to fade after 2 months but persisted up to 6 months [9]. We found that cortical high intensity on T1-weighted images generally began to appear about 2 weeks after the ictus, became prominent at 1–2 months and began to fade at 3 months, although it could persist up to 11 months. Proton-density images were more sensitive than T1-weighted images to the cortical laminar lesions. Early cortical changes on day 1 showed high intensity on T2-weighted images and low intensity on T1-weighted images, which later became high intensity. This early change was due to prolonged T1 and T2 values caused by acute ischaemic change (tissue oedema).

In cortical laminar necrosis, CT does not demonstrate haemorrhage or calcification, and MRI also fails to demonstrate haemorrhage. Although the mechanism of T1 shortening in the cortical laminar necrosis remains unclear, high cortical intensity on T1-weighted image is believed to occur by neuronal damage and reactive tissue change, that is, reactive change of glia and deposition of fat-laden macrophages [9, 10]. On our proton-density images, cortical laminar necrosis was demonstrated high intensity due to increased mobile protons in the reactive tissue. A less likely explanation is that microhaemorrhage in the cortical laminar lesion can occur in some patients, which may contribute to T1 shortening.

On CT cortical laminar necrosis shows contrast enhancement due to disruption of the blood-brain barrier, where loss of neurons and vascular proliferation occur [11]. On MRI contrast enhancement occurs in the cortical lesion with disruption of the blood-brain barrier. Parenchymal enhancement in brain infarcts is common and is generally maximal between 3 days and 3 weeks; it disappears by 3 months [13–15]. The greater sensitivity of MRI to changes in the blood-brain barrier results in prolonged detection of blood-brain barrier disruption in the cortical lesion, at up to 8 months in our series.

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