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# A characteristic feature of acute haematomas in the brain on echo-planar diffusion-weighted imaging

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**Abstract** Diffusion-weighted MRI (DWI) is used in the diagnosis of acute ischaemic disease of the brain, but it is not clear whether or not it can be used to differentiate an acute haematoma from an infarct. Our purpose was to identify any characteristic feature of acute haematomas which can be recognised on DWI and to evaluate the usefulness of DWI in acute cerebral stroke. We examined nine patients with acute haemorrhage using CT and MRI including DWI. We measured the volume and apparent diffusion coefficient (ADC) of the haematomas. All showed heterogeneous signal on DWI, and the centre of the large ( > 20 ml) haematomas especially a mixed pattern with high and low signal. The characteristic feature of acute haematomas was a peripheral low-signal region, found in all subjects regardless of the size of the haematoma; acute infarcts did not show this. This low-signal rim on DWI may be useful for differentiating an acute haematoma from an infarct.

**Keywords** Acute cerebral haemorrhage · Diffusion weighted images · Susceptibility effects

## Introduction

In the diagnosis of acute cerebral haemorrhage, CT is thought to have higher sensitivity than MRI, but Schellinger et al. [1] reported that T2- and T2*-weighted images were as sensitive as CT. Signal loss has been reported gradient-echo images in in vitro studies, animal models and clinical practice [2, 3, 4, 5, 6], but the potential of DWI in detection of acute haemorrhage is not clear; there have been few papers describing the DWI features. Linfante et al. [7] examined five patients with acute haematomas within 2 h of the onset of symptoms and reported the usefulness of echo-planar imaging (EPI). We examined nine patients with acute cerebral haematomas by EPI DWI and conventional MRI, and looked at the signal pattern of the haematoma depending on its size. Our purpose was to identify any characteristic feature and to evaluate the usefulness of DWI for differential diagnosis between infarcts and haemorrhage in patients with acute cerebral stroke.

## Materials and methods

We studied nine patients with acute cerebral haemorrhage initially thought to have had a cerebral infarct, who underwent MRI and

**Table 1.** Patients with acute intracerebral haematoma

| Patient | Age (years), sex | Time of MRI (h:min) | Site | Volume (ml) |
|---|---|---|---|---|
| 1 | 28, F | 0:40 | Basal ganglia | 62.4 |
| 2 | 88, F | 1:00 | Thalamus | 5.8 |
| 3 | 46, F | 1:20 | Thalamus | 6.4 |
| 4 | 80, M | 1:30 | Basal ganglia | 9.4 |
| 5 | 51, M | 1:30 | Subcortical | 63.6 |
| 6 | 49, M | 1:30 | Basal ganglia | 70.6 |
| 7 | 88, M | 2:00 | Thalamus | 22.7 |
| 8 | 43, M | 4:30 | Subcortical | 3.7 |
| 9 | 80, M | 13:30 | Basal ganglia | 188.5 |

**Table 2.** Patients with acute cerebral infarcts

| Patient | Age (years), sex | Time of MRI (h:min) | Site | Volume (ml) |
|---|---|---|---|---|
| I | 54, M | 1:00 | Subcortical | 157.1 |
| II | 51, M | 1:00 | Subcortical | 115.9 |
| III | 64, F | 1:30 | Basal ganglia | 16.9 |
| IV | 53, M | 2:00 | Subcortical | 21.1 |
| V | 79, F | 3:00 | Subcortical | 6.8 |
| VI | 60, F | 3:20 | Basal ganglia | 11.1 |
| VII | 73, M | 3:30 | Subcortical | 28.7 |
| VIII | 73, F | 3:30 | Subcortical | 82.4 |
| IX | 78, M | 4:30 | Basal ganglia | 15.9 |

subsequent CT; we obtained informed consent from each patient or permission from their family. We also studied nine patients with infarcts of similar duration and size (Tables 1, 2).

MRI was performed at 1.5 tesla. For DWI, we used a single-shot EPI image: TR 1000 TE 99.2 ms, field of view 24 cm, slice thickness 5 mm, slice gap 1.5 mm, matrix 128×128, and b (motion probing gradient) 1000 and 0 $mm^2/s$ applied along three axes. We also obtained T2-weighted spin-echo images: TR 4000 TE 105 ms, slice thickness 5 mm, slice gap 1.5 mm, field of view 24 cm, matrix 256×224.

The signal pattern was noted by two specialised radiologists and its intensity classed by eye as lower, isointense with or higher than normal white matter. For quantitative evaluation, we obtained an apparent diffusion coefficient (ADC) map from two images with the different values of b and a region of interest (ROI) was placed on each lesion. The mean ADC in the ROI was obtained and the decrease (rADC) from that of normal white matter was calculated as follows:

$$\mathrm{rADC}(\%) = \{(\text{ADC value of lesion}) - (\text{ADC value of the opposite white matter})\} / (\text{ADC value of the opposite white matter})$$

The variation in ADC in the lesion was assessed by the coefficient of variance [CV(%)] of ADC in the ROI. The haematoma was hand-traced on each of the T2-weighted images and its total volume calculated after compensation for the interslice gap. Statistical analysis was by analysis of variance (ANOVA) to compare the values between the haematomas and infarcts.

## Results

Typical MRI of large and small haematomas is shown in Figs. 1 and 2. Fig. 1 shows a patient 28 years of age who lost consciousness 15 min after delivery of a baby. DWI, T2-weighted images and CT 40 min after the onset of symptoms showed a 62.4 ml putaminal haematoma. On DWI its centre gave mainly high signal, but small low-signal areas gave a heterogeneous pattern. A peripheral low-signal rim was clearly seen and there was high signal surrounding the haematoma. The ADC map showed

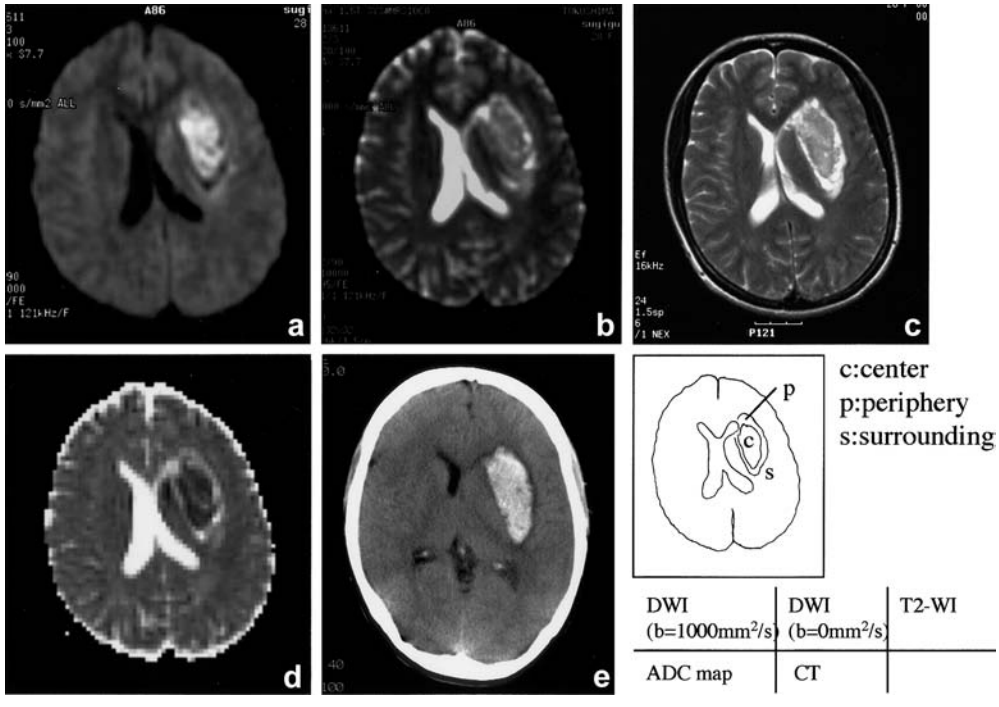


**Fig. 1a–e.** Patient 1. MRI at 40 min after the onset of symptoms shows a 62.4 ml left putaminal haematoma. **a, b** On diffusion-weighted imaging (DWI) the centre of the haematoma gives mainly high signal, but includes low-signal areas, giving a heterogeneous pattern. **d** The apparent diffusion coefficient (ADC) map showed mainly a low ADC, but an increase in the surrounding region. **e** CT shows a left putaminal haematoma surrounded by low-density areas representing vasogenic oedema

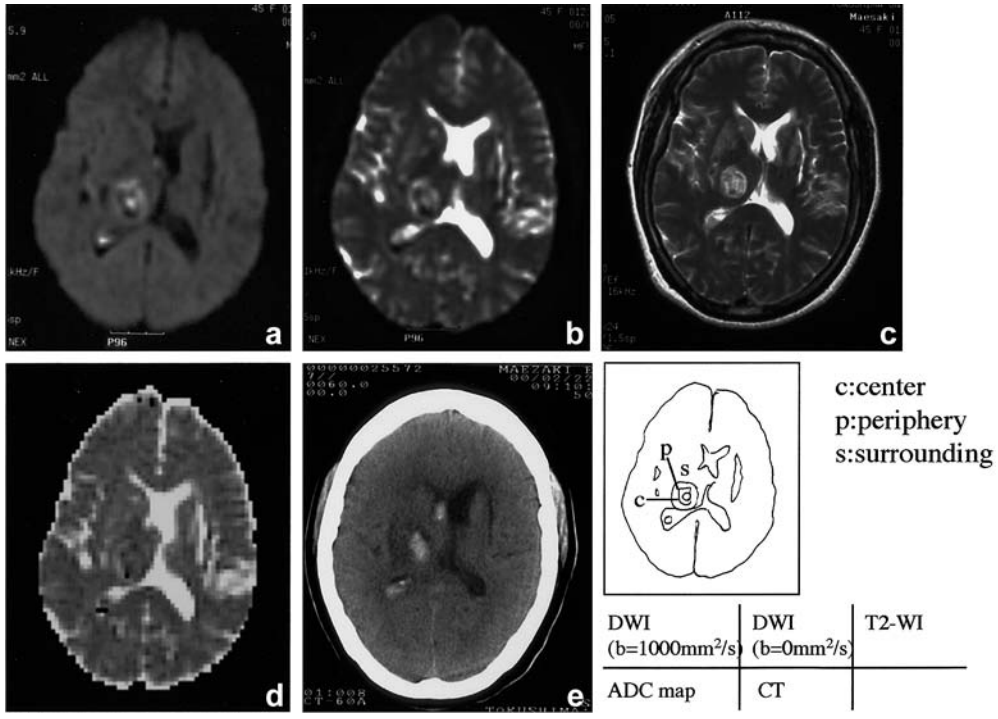


**Fig. 2a–e.** Patient 3. MRI 80 min after the onset of symptoms shows a 6.4 ml right thalamic haematoma. **a, b** On DWI its centre shows homogeneous high signal and a low-signal rim. The surrounding brain gave high signal. **d** The ADC map shows a similar pattern. **e** CT shows a right thalamic haematoma

that the centre and the outer high-signal area had slightly lower and much higher ADC, respectively, than normal white matter. On T2-weighted images, the haematoma showed heterogeneous high signal, with a higher-signal rim.

The patient shown in Fig. 2 was a 46-year-old woman with sudden speech disturbance and left arm weakness. MRI 80 min later revealed a small 6.4 ml haematoma in the right thalamus. Its signal pattern on DWI also consisted of three layers. The centre was homogeneous high-signal spot, the middle layer was a low-signal rim and the outermost region a high-signal rim. The centre had a slightly high ADC, as did the outer rim, indicating high water diffusivity. T2-weighted images showed almost the same pattern as the DWI.

Fig. 3 shows an acute infarct examined 4.5 h after the onset, which showed relatively homogeneous high signal on DWI and ADC. There was no notable low-signal periphery.

The MRI findings are summarised in Table 3. The signal from the centre of the haematomas on DWI was homogeneous or heterogeneous. Large haematomas, measuring $>20$ ml showed the heterogeneous pattern, and small haematomas, $<20$ ml, relatively homogeneous central high signal. Regardless of their volume, the low-signal rim was found in all haematomas on DWI, but some large haematomas did not show peripheral low signal on the T2-weighted images. The centre of all haematomas on T2-weighted images was isointense or gave high signal, indicating oxyhaemoglobin as the main component.

Table 4 shows the rADC and CV of haematoma and infarct. The rADC of the haematomas was higher and their CV much larger than those of the infarcts, and this was statistically significant ($r < 0.01$).

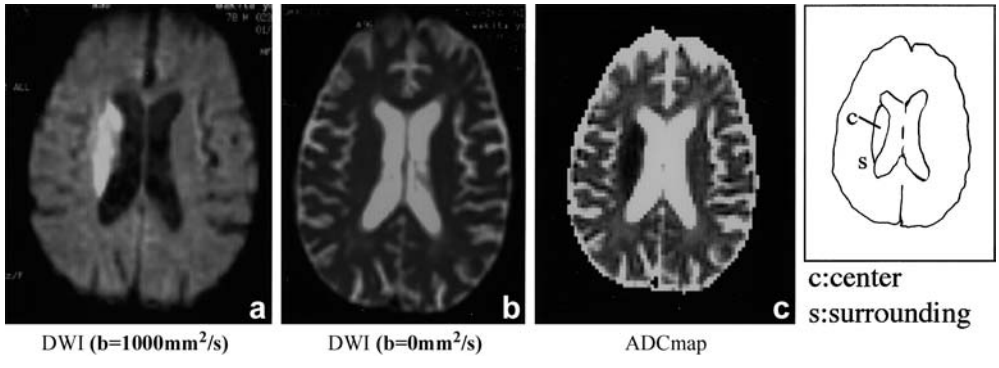


**Fig. 3a–c.** Patient 8. MRI 4.5 h after the onset of symptoms shows a 15.9 ml right cerebral infarct. **a** On DWI its centre shows homogeneous high signal, but with $b = 0$ it appears isointense. **c** The ADC map shows a low ADC

**Table 3.** Signal on MRI: acute haematomas and infarcts

| Patient | Centre | | Periphery | | Surrounding brain | |
|---|---|---|---|---|---|---|
| | Diffusion-weighted images | T2-weighted images | Diffusion-weighted images | T2-weighted images | Diffusion-weighted images | T2-weighted images |
| Haematomas | | | | | | |
| 1 | Isointense-high | Isointense-high | Low | Isointense | High | High |
| 2 | High | High | Low | Low | High | High |
| 3 | High | High | Low | Low | High | High |
| 4 | High | High | Low | High | High | High |
| 5 | Low-high | Isointense-high | Low | Isointense | High | High |
| 6 | Low-high | Isointense-high | Low | Low | High | High |
| 7 | Low-high | Low-high | Low | Isointense | High | High |
| 8 | High | High | Low | Isointense | High | High |
| 9 | Low-high | Low-high | Low | High | High | High |
| Infarcts | | | | | | |
| I | High | Isointense | – | – | Isointense | Isointense |
| II | High | Isointense | – | – | Isointense | Isointense |
| III | High | Isointense | – | – | Isointense | Isointense |
| IV | High | Isointense | – | – | Isointense | Isointense |
| V | High | Isointense | – | – | Isointense | Isointense |
| VI | High | Isointense | – | – | Isointense | Isointense |
| VII | High | Isointense | – | – | Isointense | Isointense |
| VIII | High | Isointense | – | – | Isointense | Isointense |
| IX | High | Isointense | – | – | Isointense | Isointense |

**Table 4.** Apparent diffusion coefficients (ADC)

| Patient | Coefficient of variation of ADC of normal white matter | Change in ADC in centre lesion (%) | Coefficient of variation of ADC in centre of lesion |
|---|---|---|---|
| Haematomas | | | |
| 1 | 11.9 | −7 | 48.2 |
| 2 | 5.2 | 30 | 41.4 |
| 3 | 10.6 | 8 | 30.1 |
| 4 | 7.8 | 112 | 50.3 |
| 5 | 9.1 | 28 | 49.5 |
| 6 | 8.1 | 46 | 47.1 |
| 7 | 8.8 | −2 | 46.6 |
| 8 | 9.8 | 15 | 49.6 |
| 9 | 5.7 | 42 | 37.4 |
| Mean | 8.6 ± 2.2 | 30.2 ± 35.8 | 44.5 ± 6.9 |
| Infarcts | | | |
| | 8.6 | −45 | 27.7 |
| | 12.4 | −39 | 11.5 |
| | 8.7 | −38 | 15.2 |
| | 10.3 | −34 | 13.1 |
| | 4.1 | −59 | 6.3 |
| | 13.8 | −49 | 19.6 |
| | 5.6 | −32 | 11.5 |
| | 13.7 | −73 | 63.0 |
| | 7.6 | −57 | 20.4 |
| | 9.4 ± 3.4 | −47 ± 14.0 | 20.9 ± 17.0 |

## Discussion

The signal from a haematoma on MRI is attributable to the state of oxygenation of the haemoglobin and the status of the membrane of the red blood cells (RBCs) [8, 9, 10]. In the acute stage, a cerebral haematoma may still contain intact biconcave RBCs with oxygenated haemoglobin, but the RBCs gradually change, shrinking and forming echinocytes, and intracellular oxyhaemoglobin loses oxygen to form deoxyhaemoglobin. Atlas et al. [11] reported that lysed RBCs had higher ADC and intact RBCs a lower ADC than with white matter. In our study, the high rADC and high signal on T2-weighted images of the centre of haematoma may reflect an increased fluid component and the high diffusivity of serum which has leaked out. The heterogeneity of the centre of the haematoma may be caused by a mixture of different stages of oxygenation (early deoxygenation) in intact RBCs, possibly including fibrin and various proteins.

Although its size was variable, the low-signal rim was the most common finding in the haematomas. It was more prominent on DWI than on conventional T2-weighted images and we think it may have been due to the susceptibility effects of deoxyhaemoglobin in the periphery of the haematoma, related to early deoxygenation of intact RBCs. Maldjian et al. [12] reported that measurement of ADC of haematomas which give low signal on T2-weighted images may be problematic; the ADC may be inaccurate due to variable, unpredictable susceptibility effects within the haematoma. The signal pattern on DWI could therefore be governed more by T2- and susceptibility effects than by diffusivity. Nevertheless, the low-signal rim appears characteristic of haematomas and this suggests that acute haemorrhage can be diagnosed if such a rim is found on DWI

and that the lesion can be differentiated from an infarct. MRI might thus be the imaging modality of first choice in acute stroke.

We found a high-ADC region surrounding the haematoma in some cases, indicating high diffusivity possibly due to vasogenic oedema. DWI can provide pathophysiological information about the adjacent tissue, and may be useful for understanding the influence of haematomas on the brain and possibly in assessing prognosis.

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