The value of MRI in angiogram-negative intracranial haemorrhage

S. A. Renowden, A. J. Molyneux, P. Anslow, J. V. Byrne

Department of Neuroradiology, Radcliffe Infirmary, Oxford, UK

Received: 9 November 1993/Accepted: 12 November 1993

Abstract. In one year, cerebral angiograms were performed for intracranial haemorrhage (ICH) on 334 patients. No cause for haemorrhage could be identified in 41 (12 %), 30 of whom had predominantly subarachnoid (SAH) and 11 predominantly parenchymal haemorrhage (PH). These patients were prospectively examined by cranial MRI 1-6 weeks after the ictus. The MRI studies were positive in 7 patients (17 %). In the 30 patients examined after SAH, 2 studies were positive, showing an aneurysm in one case and a brain stem lesion of uncertain aetiology in the other. In those examined after PH, cavernous angiomas were shown in 2, a tumour in 1 and a vascular malformation in another; useful diagnostic information was thus obtained in 36 % of this group.

Key words: MRI - Angiogram negative intracranial haemorrhage

Spontaneous subarachnoid haemorrhage (SAH) results from rupture of an intracranial aneurysm in 75 % of cases [1]; other causes include vascular malformations, neoplasms, meningoencephalitis and blood dyscrasias. The reported incidence of SAH for which no cause can be found at angiography ranges from 2 % to 27 % [1-10]; the lower figures come from the earlier series [2, 4]. Angiography is likely to demonstrate the cause were intracranial haemorrhage is secondary to aneurysmal rupture, a bleeding AVM, vasculitis or venous thrombosis. Intracranial haemorrhage for which no cause can be found at angiography is often attributed to ruptured microaneurysms and leakage from small lenticulostrate and perforating vessels. Angiography may also be normal in patients in whom haemorrhage is due to cavernous angiomas. We have investigated the place of MRI in the management of patients with intracranial haemorrhage for which no cause can be found on angiography.

Patients and methods

We studied patients referred for investigation of the cause of spontaneous intracranial haemorrhage. Patients presenting with SAH, diagnosed by lumbar puncture or cranial CT were investigated by selective angiography of all cerebral vessels whereas patients whose CT studies demonstrated predominantly parenchymal haemorrhage (PH) were studied by injection of the appropriate intracranial vessels. Patients in whom angiography failed to show a cause for haemorrhage were further investigated by cranial MRI 1-6 weeks after the ictus.

The MRI examinations were performed on an imager operating at 1.5 Tesla. A dedicated head coil was used to obtain spin-echo T1-weighted (T1W) sagittal images (TR 500: TE 11; matrix 256 x 192; 1 NEX), fast spin-echo T2-weighted (FSE T2W) axial images (TR 5500: effective TE 102; echo train length 16; interecho spacing 20 ms; matrix 256 x 256; 1 NEX), and FSE dual-echo coronal images (TR 3000: effective TE 18 and 108; echo train length 8; interecho spacing 20 ms; matrix 256 x 256; 1 NEX). Gradient-echo (GRE) images were performed only where appropriate.

All examinations were interpreted independently by two neuroradiologists.

Results

In one year, 334 patients were investigated for spontaneous intracranial haemorrhage. In 41 patients (12 %), 24 women and 17 men no structural cause for haemorrhage was demonstrated by angiography, and they were examined by MRI. Patient mortality was zero. No patient declined MRI.

Angiogram negative subarachnoid haemorrhage (ANSAH)

There were 30 patients (17 women and 13 men, aged 22-74 years) with CT evidence of SAH in 17 and negative CT but SAH confirmed by lumbar puncture in 13, who had normal cerebral angiograms. None had a history of hypertension. MRI showed a possibly relevant abnormality in only two. In a 26-year-old woman with CT evidence of generalised SAH, extending into the fourth ventricle and...
Fig. 1. T2W FSE (a) and GRE (b) axial images at the level of the third ventricle demonstrate a midline aneurysm. Oblique projection of left internal carotid arteriogram performed shortly after the haemorrhage demonstrates no abnormality. Corresponding projection after the MRI examination confirms the presence of a left anterior cerebral artery aneurysm.

Fig. 2. a Midline sagittal T1W image demonstrates a cystic vermian tumour exhibiting minimal mass effect. The appearance is readily differentiated from resolving haemorrhage. b Contrast-enhanced axial CT section through the posterior fossa demonstrates an area of low attenuation posterior to the fourth ventricle. This is not space-occupying and shows some irregular contrast enhancement. The appearance could be consistent with a resolving haematoma.

Frontal horns of the lateral ventricles, MRI performed 6 weeks later revealed an interhemispheric aneurysm (Fig. 1a, b). Repeat angiography (Fig. 1d) demonstrated an anterior cerebral artery aneurysm which had not been shown on the first angiogram (Fig. 1c).

In a 44-year-old woman with CT evidence of blood in the pontine, right ambient and chiasmatic cisterns, MRI demonstrated a lesion, without mass effect or contrast enhancement, on the left of the medulla oblongata and pons. The patient is now asymptomatic and without abnormal neurological signs. MRI 3 months later was unchanged. The nature of the lesion is uncertain and the patient remains under observation since biopsy is considered unwarranted.

In the remaining 28 patients with ANSAH, MRI did not demonstrate a causative lesion. Haemoglobin degradation products were demonstrated within the ventricles of one patient and small white matter ischaemic lesions were seen in five. None of the patients developed hydrocephalus.

In 15 patients CT localised subarachnoid haemorrhage to the right Sylvian fissure in 1 and to the perimesencephalic cisterns in 14; in the latter group the haemorrhage extended into the ventricular system in 4, the chiasmatic cistern in 2 and the anterior interhemispheric fissure in 1.

Angiogram negative parenchymal haemorrhage (ANPH)

No structural cause was demonstrated on angiography in 4 women and 7 men aged 16–48 years with predominantly parenchymal haemorrhage on CT.

In 7 of these patients, MRI demonstrated resolving haemorrhage without evidence of a structural lesion; in 3 patients, the clot was in the left temporal lobe and in the left frontal lobe, left thalamus, right-perisylvian area and right basal ganglia in the remaining 4. Two 38-year-old men, who were receiving antihypertensive medication had haematomas in the left temporal lobe and right basal ganglia respectively.

Histologically confirmed cavernous angiomas were diagnosed on the basis of typical MRI appearances at the site of a small haematoma in the right hippocampus in a 35-year-old man and in a 16-year-old female with a haematoma in the left posterior frontal and temporal lobes. In the latter patient MRI demonstrated two small additional cavernous angiomas in the right frontal lobe; her brother had bled some years previously from a cavernous angioma.

In another 16-year-old female, MRI revealed a cystic tumour, later shown histologically to be a pilocytic cerebellar astrocytoma, in association with a posterior fossa midline haematoma (Fig. 2a). On subsequent contrast-enhanced CT we could not differentiate resolving haematoma from the underlying neoplasm (Fig. 2b).

A vascular anomaly was suggested by MRI in a 31-year-old man with a haematoma in the left thalamus. Angiography 3 days after the acute event demonstrated only mass effect from the haematoma. MRI confirmed the presence of a resolving haematoma and demonstrated an abnormally large blood vessel posterior and superior to the haematoma, suggesting an underlying vascular anomaly. The patient refused repeat angiography and remains well.