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Posterior encephalopathy with vasospasm: MRI and angiography

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Abstract Posterior encephalopathy is characterised by headache, impairment of consciousness, seizures and progressive visual loss. MRI shows bilateral, predominantly posterior, cortical and subcortical lesions with a distribution. Our aim was to analyse the MRI lesion pattern and angiographic findings because the pathophysiology of posterior encephalopathy is incompletely understood. We report three patients with clinical and imaging findings consistent with posterior encephalopathy who underwent serial MRI including diffusion-weighted imaging (DWI) and construction of apparent diffusion coefficient (ADC) maps, and four-vessel digital subtraction angiography (DSA). DWI revealed symmetrical subcortical and cortical parieto-occipital high signal. High

and also low ADCs indicated probable vasogenic and cytotoxic oedema. On follow-up there was focal cortical laminar necrosis, while the white-matter lesions resolved almost completely, except in the arterial border zones. DSA revealed diffuse arterial narrowing, slightly more marked in the posterior circulation. These findings suggest that posterior encephalopathy may in some cases be due to diffuse, severe vasospasm affecting especially in the parieto-occipital grey matter, with its higher vulnerability to ischemia. Cerebral vasospasm due to digitoxin intoxication, resulting in posterior encephalopathy, has not yet been described previously.

Keywords Posterior encephalopathy · Magnetic resonance imaging · Arterial spasm · Angiography

Introduction

In posterior encephalopathy white matter and the cortex, especially in the posterior parietal and occipital lobes are typically affected [1, 2]. The condition was first described as posterior reversible leukoencephalopathy by Hinckley et al. [3], but several studies have shown occipital cortical involvement, sometimes with slight hemorrhagic transformation [1, 2, 4, 5]. Whereas in most cases the condition is reversible, it may cause irreversible neurological deficits due to cytotoxic oedema, particularly in the parieto-occipital cortex [6, 7, 8]. There is therefore disagreement over the proper name for this

syndrome [1, 5]. The most common causes are hypertensive encephalopathy, preeclampsia, eclampsia [1, 2, 3, 4, 5, 6, 7, 8, 9], or cytotoxic substances, notably immunosuppressants such as cyclosporine A (CSA) or tacrolimus (FK 506) [3, 10, 11, 12, 13]. Less common causes are severe hypercalcaemia [14], porphyria [15, 16], and chemotherapeutic drugs such as gemcitabine [17], cisplatin, and L-asparaginase [11, 12, 18]. Clinically, the condition is characterised by headache, nausea, vomiting, altered mental state, seizures and progressive visual loss [1, 2]. Its pathophysiology is incompletely understood. One suggestion is reversible vasospasm [9, 19, 20, 21, 22, 23]; another is cerebral hyperperfusion [1, 2, 24,

25, 26]. Due to limited cerebral autoregulation, hyperperfusion may cause breakdown of the blood-brain barrier and vasogenic oedema. One reason for the characteristic anatomical distribution might be a lower number of adrenoreceptors in the posterior circulation

Fig. 1a–f Patient 1. **a, b** Axial T2-weighted spin-echo and fluid-attenuated inversion-recovery (FLAIR) images showing bilateral parieto-occipital high signal in white and grey matter, sparing the calcarine region, an old lacune in the left thalamus and small incidental small foci of vascular aetiology in the right basal ganglia and external capsule. **c** Axial diffusion-weighted image, showing high signal. **d–f** Digital subtraction angiography (DSA) showing multiple areas of narrowing of **d** both posterior cerebral arteries (PCA), particularly the left P2 segment (*arrow*) and **e** the A1 and M1–M2 segments (*arrows*), with **f** segmental dilatation of the pericallosal artery (*arrow*)

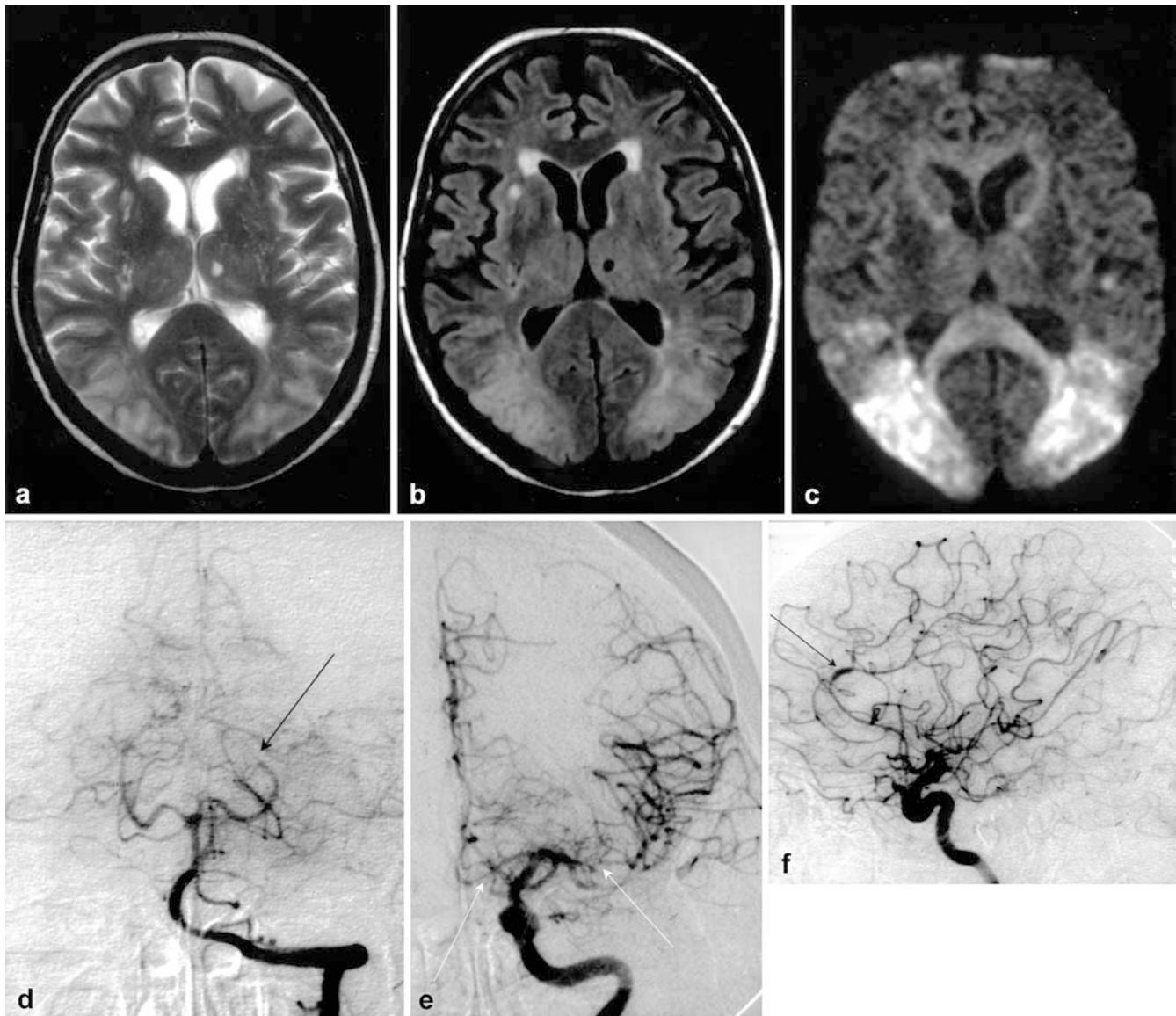
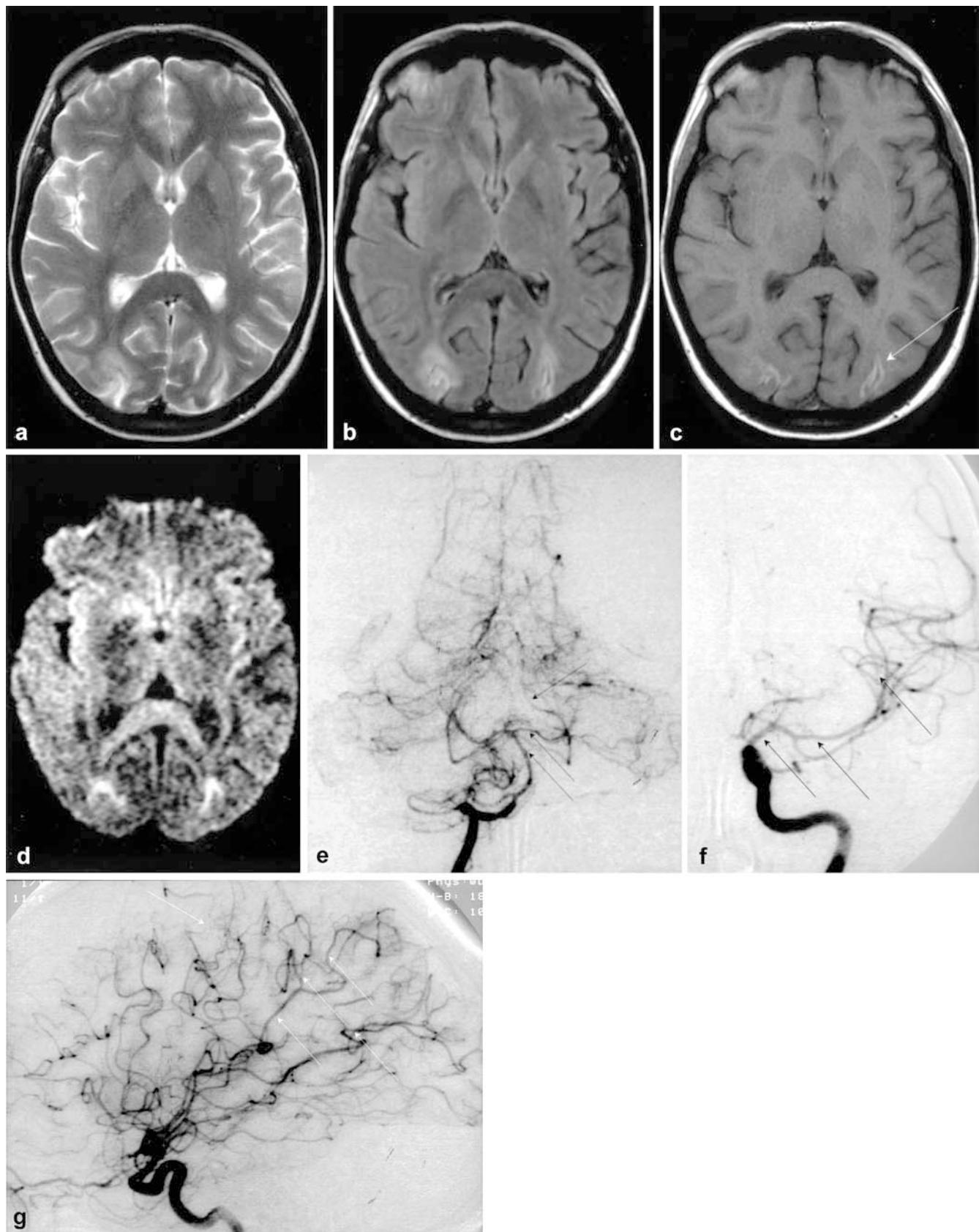


Fig. 2a–g Patient 2. **a, b, d** Axial T2-weighted image, FLAIR and DWI with an axial diffusion gradient, showing small bilateral high signal areas in the cortex and subcortical white matter of the occipital poles. **c** A T1-weighted image showed gyriform high signal, characteristic of cortical laminar necrosis. **e–g** DSA, demonstrating diffuse **e** narrowing of the middle and distal segments of the basilar artery and both PCA, consistent with severe spasm (*arrows*) and **f, g** constriction of the C1, M1 and M2 segments and of the distal middle cerebral artery (MCA) branches, particularly the posterior parietal and angular arteries (*arrows*)

than in the internal carotid artery territory [13, 25, 26, 27, 28]. MRI reveals relatively symmetrical lesions in the subcortical and cortical structures of the occipital and parietal lobes giving high signal on T2-weighted spin-echo and fluid-attenuated inversion-recovery (FLAIR) images [1, 2, 25, 26]. These lesions less commonly occur



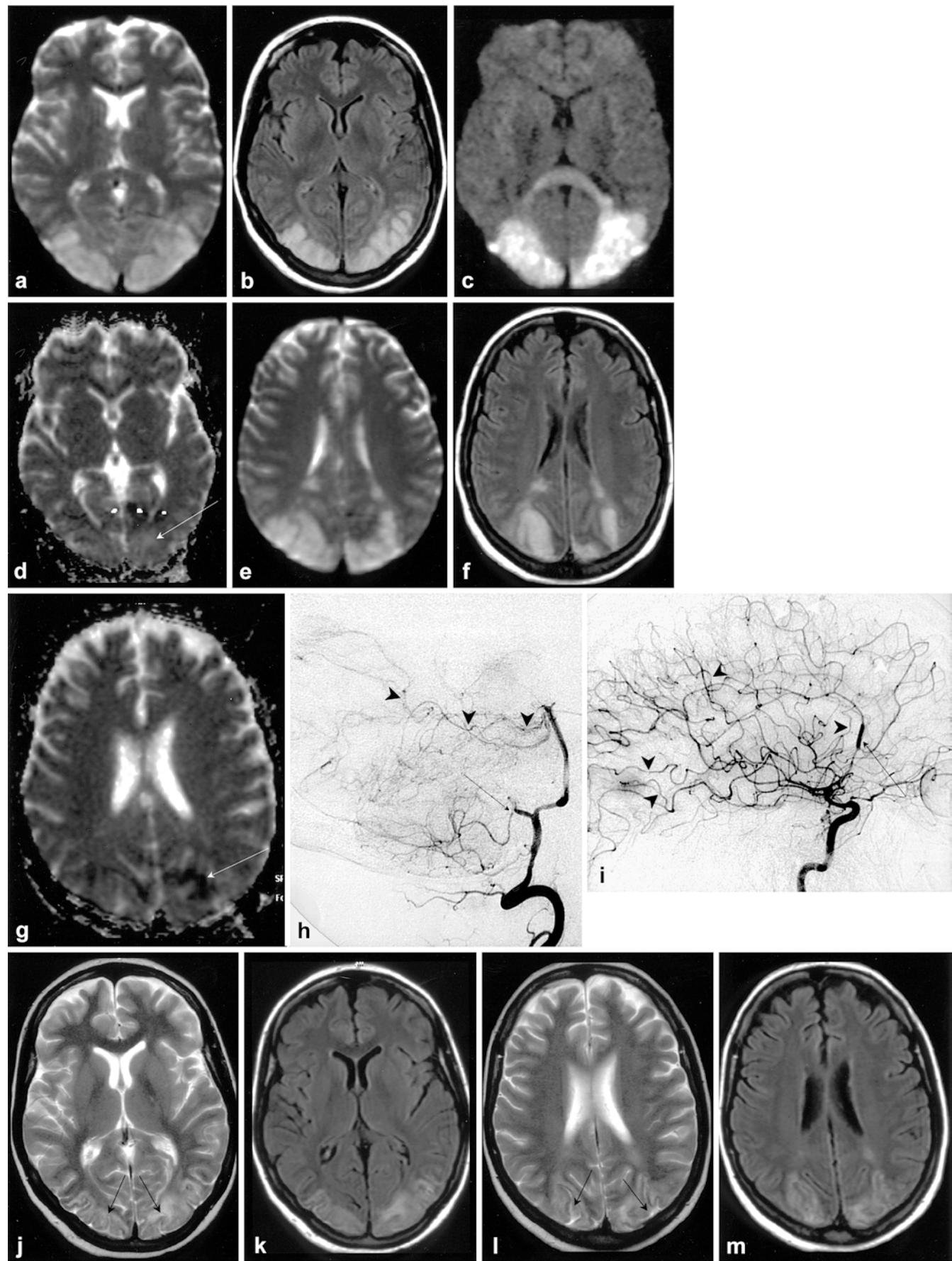


Fig. 3a–m Patient 3. **a, b** Axial T2-weighted spin-echo and FLAIR images through the basal ganglia demonstrating symmetrical, inhomogeneous grey and white matter high signal. **c** Axial DWI and **d** apparent diffusion coefficient (ADC) map showing high diffusion (ADC $1.27 \times 10^{-3} \text{ mm}^2/\text{s}$), presumably due to vasogenic oedema. **e, f** Axial T2-weighted images through the upper part of the lateral ventricles showing bilateral cortical and subcortical parieto-occipital high signal; there is also high signal in deep white matter, more evident on the right side. **g** The ADC, $0.54 \times 10^{-3} \text{ mm}^2/\text{s}$, suggests that there is also focal cytotoxic oedema. **h** Vertebral angiography (lateral view) shows diffuse spasm of the posterior inferior cerebellar artery, involving hemispheric and inferior vermian branches, sparing the proximal segment (*arrow*). There is also severe reduction in diameter of the P1–3 segments bilaterally (*arrowheads*). **i** Carotid angiogram (lateral view) showing diffuse irregularity due to spasm in the MCA branches (operculofrontal, posterior parietal and temporo-occipital, *black arrowheads*) and in the anterior cerebral artery branches (*white arrowheads*), with focal dilatation of the pericallosal artery (*arrow*). **j–m** MRI 14 days later showing moderate resolution of the signal change. However, occipital subcortical white matter and adjacent cortex still give high signal (*arrows*)

in the posterior frontal or temporal lobes, pons or cerebellum [1, 2, 3, 26]. Because investigation of serial MRI patterns and angiographic features may give insights into the pathogenesis, we report on three patients with posterior encephalopathy, in whom DSA showed diffuse arterial spasm.

Case reports

Case 1

A 73-year-old woman presented with progressive visual loss, disorientation and vomiting 72 h after digitoxin intoxication, with a maximum serum level of $124 \mu\text{g/l}$. Examination showed cortical blindness, with Anton's syndrome, but pupillary reflexes and fundoscopy were normal. The patient was disorientated, with reduced concentration and mild deficits of short-term memory. Blood pressure, routine blood tests, autoimmune antibody screening and cerebrospinal fluid (CSF) analysis were normal. MRI revealed bilateral symmetrical high signal in the parieto-occipital subcortical white matter and cortex on T2-weighted spin-echo and FLAIR images and on DWI (Fig. 1a–c). DSA showed diffuse, irregular narrowing of the major and distal arteries in the anterior, middle and posterior cerebral artery territories (Fig. 1d, e). The greatest reduction in diameter was in the left M1 and proximal M2, and P2 and P3 segments. Over the next 4 months the patient's cognitive deficits and visual acuity slowly improved.

Case 2

A 36-year-old woman was admitted 16 days after the normal delivery of two healthy children with headache and mildly blurred vision for 24 h. By the following morning she had almost complete loss of vision and impaired consciousness, a mild left hemiparesis with brisk reflexes and extensor plantar responses. She had no signs of eclampsia or preeclampsia nor any history of hypertension. Her blood pressure on admission was $126/73 \text{ mm Hg}$. Lumbar puncture revealed normal opening pressure and normal CSF. MRI 24 h after the onset of neurological disturbance

showed bilateral abnormalities in the occipital grey and white matter, with high signal on T2-weighted images and DWI (Fig. 2a, b, d). DSA revealed diffuse arterial narrowing in the anterior and posterior circulation (Fig. 2e–g), with severe spasm of the middle and distal thirds of the basilar artery and of the P1–3 segments, slightly more marked on the left (Fig. 2e). On MRI 12 days later the high signal areas had partly resolved, but T1-weighted images disclosed gyriform high signal, characteristic of cortical laminar necrosis complicating infarcts of the occipital cortex, sparing the calcarine region (Fig. 2c). Follow-up over 31 months showed complete regression of the neurological disorder, with the exception of slight visual field defects. The patient could read and had unrestricted daily activities.

Case 3

A 29-year-old woman with a 8-month history of Crohn's disease underwent jejunal and ileocaecal resection because of a severe fistula and abdominal adhesions. She had been treated with corticosteroids for 7 months, most recently 40 mg prednisone/day. Four days later she had progressive visual loss, leading to cortical blindness and generalised seizures, and T2-weighted images (Fig. 3a, b, e, f) and DWI (Fig. 3c) showed marked symmetrical parieto-occipital high signal in grey and subcortical white matter. The dorsal part of centrum semiovale and the right cerebellar hemisphere also gave high signal. Apparent diffusion coefficients (ADC) were low in focal subcortical regions and the adjacent grey matter (Fig. 3g), whereas they were high in large areas of the white matter (Fig. 3d), probably indicating combined cytotoxic and vasogenic oedema. DSA revealed diffuse spasm of medium-size and small arteries, more marked on the posterior cerebral arteries (Fig. 3h, i). MRI 2 weeks later showed moderate resolution of the high signal abnormalities on T2-weighted images (Fig. 3j–m); there had also been gradual improvement of vision. CSF analysis was normal, and extensive laboratory investigations showed no evidence of vasculitis.

Discussion

Hinchey et al. [3] reported on 15 patients with reversible white-matter lesions with high parieto-occipital signal on T2-weighted images. The main clinical features were cortical blindness and other visual defects, confusion and seizures [2, 3]. Probable causes were immunosuppressive therapy [29, 30] especially with CSA or FK 506 [3, 10, 11, 12, 13, 17, 18], acute hypertensive encephalopathy associated with renal disease [3, 6], eclampsia [24, 25, 27], and interferon therapy [3]. All 15 patients' neurological deficits resolved within 2 weeks. Further reports showed both grey and white matter involvement [1, 2, 4, 5]. Follow-up MRI reported from Ay et al. [6] and Antunez et al. [7] revealed partially irreversible lesions, especially in the subcortical occipital white matter and high signal in the adjacent cortex on T1-weighted images, presumably cortical laminar necrosis. Thus, posterior encephalopathy may not be reversible. Associated with a wider variety of conditions even without severe hypertension, other presumed causes include treatment with gemcitabine [17], cisplatin [18] or L-asparaginase [18, 29], hypercalcaemia [14], porphyria

[15, 16], late-onset postpartum eclampsia [19] and postpartum cerebral angiopathy [23].

Although not all lesions are parieto-occipital, the most common neuroradiological findings are in the posterior cerebral artery territory [1, 3, 25, 26]. Three different pathophysiological explanations for the MRI appearances have been pursued.

The first is that breakdown of cerebrovascular autoregulation because of increased systemic blood pressure may result in dilatation with hyperperfusion and vasogenic oedema [1, 3, 5, 8, 25, 26]. Predominantly parieto-occipital involvement of regions could be explained by sparser sympathetic innervation in this vascular territory [2, 27, 28], so that protective sympathetic activity may be less effective in acute hypertension [2, 25]. Fluid extravasation into the intercellular space, vasogenic oedema, causes high signal on T2-weighted images and ADC maps, whereas DWI appears normal because of the counteracting effects of T2 prolongation and increased diffusion [25, 26, 31, 32]. There could be also small bright regions on DWI, with lower ADC consistent with cytotoxic oedema, although there is evidence that this relationship is not constant [1, 4, 6]. However, our angiographic findings were of diffuse vasoconstriction. MRI revealed probable cytotoxic oedema and cortical necrosis (Figs. 2c, 3g), probably a consequence of vasospasm-induced ischaemia [33, 34].

The second theory suggests spasm of cerebral arteries in response to acute hypertension [19, 23, 32, 35]. Decreased cerebral blood flow and possible intra-arterial thrombosis as a consequence of over-reaction may result in ischaemia and cytotoxic oedema, especially in the border zones between arterial territories [1, 16, 22, 32, 35]. Sundgren et al. [32] showed markedly decreased perfusion with reduced regional cerebral blood flow (rCBF) in both occipital lobes, with areas of increased signal on T2-weighted images. However, there are few reports of diffuse narrowing on DSA [2, 20, 21, 22, 36], and in the four women with acute postpartum neurological problems reported by Raps et al. [19], there was no clinical evidence for eclampsia, especially no hypertension, DSA revealing diffuse vasospasm in two. It was suggested that they had delayed peripartum vasculopathy or postpartum cerebral angiopathy without hypertension [23].

Finally, in CSA-induced deficits, posterior encephalopathy being the most serious, there are other pathophysiological considerations. It is suggested that CSA causes cerebral ischaemia by direct endothelial damage [10, 11, 12], and that, with release of vasoactive substances such as endothelin 1, spasm in cerebral arteries would be likely [11, 13]. However, the signal changes are usually reversible, which probably indicates vasogenic, not cytotoxic oedema [37].

Our findings support the second theory, because DSA showed diffuse vascular narrowing with slight preference

for the posterior circulation (Figs. 1d, 2e, 3h). None of the patients had a history of hypertension. Serial MRI revealed a uniform pattern of occipital and parieto-occipital lesions. ADC maps (Fig. 3g) showed reduced cortical and subcortical diffusion, probably indicating cytotoxic oedema [24, 26, 31], and increased diffusion, suggesting vasogenic oedema, in the deep white matter (Fig. 3d) [2, 4, 24, 25, 31, 38]. Thus, high signal on T2-weighted images was likely to be due to cytotoxic and vasogenic oedema, with a predominance of cytotoxic changes in the cortex [1, 25, 26]. The changes in the white matter were largely reversible, whereas adjacent occipital grey matter showed necrosis [1, 6, 22] (Fig. 2c). Although Sundgren et al. [32] described severely reduced perfusion with vasogenic oedema in posterior encephalopathy, we demonstrated cytotoxic oedema with infarction. Ay et al. [6] demonstrated a similar phenomenon, with decreased microcirculation in the regions of cytotoxic oedema. Permanent neurological deficits and infarcts at the site of decreased rCBF were reported on follow-up. Thus, cytotoxic oedema and reversible tissue damage may depend on the severity of vasospasm-induced ischaemia [6, 26, 32, 33, 34]. Grey matter particularly at risk, with a greater vulnerability to ischaemia [1, 6, 20, 22]. Ay et al. [6] and Sundgren et al. [32] showed severely reduced perfusion on MRI, while we found arterial constriction on DSA. We therefore hypothesise that vasospasm is responsible for hypoperfusion and cerebral oedema.

Although angiography demonstrated diffuse severe vasospasm in the posterior, middle and anterior cerebral artery territories, the tendency is to involvement of the parieto-occipital regions supplied by the posterior circulation [1, 3, 26]. Contrast-medium side effects after coronary angiography also favour the occipital region [39, 40]. The clinical features are quite similar to those in posterior encephalopathy, with blurred vision or cortical blindness, impaired consciousness, generalised seizures and headache [39, 40]. There are few pathophysiological data about preferential parieto-occipital involvement; transient cortical blindness after coronary angiography might be due to higher contrast medium levels in the occipital region because of the patient's position [39].

A review of the literature might suggest that posterior encephalopathy is a relatively nonspecific reaction to different vascular disturbances [28]. Our patients, like those in some previous reports [19, 22, 23, 32, 33, 34] may represent a pathophysiological subgroup with severe vasospasm as a probable cause. In addition, diffuse cerebral vasospasm due to digitoxin intoxication has not been reported previously, and may be added to the probable causes of posterior encephalopathy.

The signal characteristics and the site of the lesions are not specific [1, 15]. Especially when grey-matter involvement predominates on MRI, the differential diagnosis should include mitochondrial cytopathy [41]

and reversible bilateral cortical signal abnormality due to seizures [42]. T2-weighted images may show increased parieto-occipital signal due to impairment of venous outflow in superior sagittal sinus thrombosis [43] and in occlusion of the basilar artery, the “top of the basilar” syndrome, with abrupt onset of neurological disorders [44, 45].

Although spasm was striking, two of our patients had segmental dilatation of the pericallosal arteries, possibly due to focal vasoparalysis or partial destruction of the vessel wall [20, 46]. The angiographic differential diagnosis includes infectious and noninfectious vasculitides [46, 47], so that extensive neurological and laboratory

investigations, including CSF analysis are necessary to exclude vasculitis or meningoencephalitis [48]. Other causes of multiple intracranial narrowings are arteriosclerosis, migraine, radiation, recreational drugs such as methamphetamine and cocaine, pharmaceutical agents, including ergot derivates and oral contraceptives, arterial spasm after subarachnoid haemorrhage, fibromuscular dysplasia, lymphoma and severe pituitary disturbances [46, 49, 50].

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