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Eclamptic encephalopathy: MRI, including diffusion-weighted images

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Abstract Eclampsia is a rare condition peculiar to pregnant and puerperal women. We analyse imaging features in five patients with eclampsia, and determine whether diffusion-weighted imaging (DWI) could differentiate cytotoxic and vasogenic oedema in four of them. All were imaged within 4 days of the onset of symptoms. We found lesions with a prolonged T2 in the brain of all five patients, in the basal ganglia in four, pons in three and posterior cerebral white matter in two. Isotropic DWI revealed variable intensity in these regions. The ADC was decreased in one, and increased in all the others. The lesion with reduced ADC progressed to infarction.

Keywords Eclampsia · Magnetic resonance imaging · Diffusion-weighted imaging

Introduction

Pre-eclampsia is defined as the development of hypertension, proteinuria, and oedema after the 24th week of gestation; it occurs in 4–5% of pregnant women [1, 2]. Eclampsia is defined as seizures before, during or after delivery in patients with pre-eclampsia. Pre-eclamptic and eclamptic patients develop many neurological problems, such as headache, visual changes, confusion, and seizures [3], and neuroimaging studies are abnormal [4, 5, 6, 7].

The reported MRI features of eclampsia are reversible lesions with prolonged T2, predominantly in the posterior cerebral white matter [6, 7, 8]. Recent case reports indicate that diffusion-weighted MRI (DWI) can discriminate cytotoxic and vasogenic oedema in eclamptic patients [9, 10, 11]. We analysed the distribution of lesions and DWI features in eclamptic patients.

Materials and methods

We reviewed MRI of five eclamptic patients (Table 1), aged 26-33 years (mean 29.2 years). All patients were primigravida and developed seizures before, during or after delivery. All were examined by MRI within 4 days of onset.

All underwent MRI at 1.5 tesla. We acquired fast spin-echo (FSE) and fluid-attenuated inversion-recovery (FLAIR) T2-weighted images with the following parameters: FSE: TR 3903 TE 100 ms, echo train length (ETL) 10, FLAIR TR 10000 TE 120 TI 2725 ms, ETL 17. DWI was obtained in four patients using a diffusion gradient in three orthogonal directions, with a maximum b of 1000 s/mm^2. We used single-shot, multisection, SE echo-planar imaging: TR 4000 TE 100 ms, field of view 240 mm, matrix 128×96, obtaining 19 sections 6 mm thick, 1 mm intersection gap with all sequences.

We generated apparent diffusion coefficient (ADC) using an image analysis system, and we assessed the isotropic (b 1000 s/mm^2) DWI and ADC maps. ADC were measured from regions of interest within areas of apparently normal white matter and within high-signal lesions on FLAIR images.

Table 1 Clinical features of patients with eclampsia

Patient	Age, years	Pregnant, weeks	Onset of seizures	Delivery	Highest blood pressure, mm Hg	MRI after onset, days	Other tests
1	26	40	just after delivery	natural	150/80	2 day	
2	27	38	during delivery	vacuum	156/90	3 day	
3	28	33	before delivery	cesarean	140/100	4 day	HELLP[a] syndrome
4	32	39	during delivery	natural	208/130	2 day	
5	33	39	1 hour after delivery	natural	200/100	4 day	HELLP syndrome

Table 2 Abnormal signal on T2-weighted images

Patient	Brain stem	Basal ganglia			Capsules	Subcortical white matter
		Putamen	Caudate nucleus	Globus pallidus		
1		Left	Left			
2[a]	Pons	Bilateral	Bilateral	Bilateral	Bilateral	
3						Bilateral occipital, parietal, frontal
4	Pons	Lt	Left		Left	Right parietal
5	Pons	Bilateral	Right		Bilateral	–

[a]intraventricular haemorrhage in addition

Results

All patients had systemic hypertension, but only patients 4 and 5 had severe hypertension, with blood pressures >180/110 mm Hg. Two patients were diagnosed as having the HELLP syndrome (Table 1). We saw lesions with prolonged T2 in all patients (Table 2). The most affected regions were the basal ganglia, in which two patients had bilateral and two unilateral abnormalities. The pons was abnormal in three patients, and the subcortical white matter in two patients. Patient 1 had a very faint long-T2 lesion in left basal ganglia, which were more prominent on FLAIR images (Fig. 1). Patient 2 had an intraventricular haemorrhage, and we carried out ventricular drainage (Fig. 2). Patient 3 had bilateral multifocal lesions predominantly in the posterior subcortical cerebral white matter (Fig. 3).

Table 3 summarises the isotropic DWI and ADC findings. Isotropic DWI revealed variable intensity in regions which appeared abnormal on T2-weighted images. The ADC was decreased in one lesion, and increased in all the others that showed low, high or isointensity on isotropic DWI. The lesion with reduced ADC, in patient 2, had progressed to cerebral infarction on the subsequent, but the patient had no neurological deficit on discharge.

Discussion

Neuroimaging studies in eclampsia have shown reversible lesions of low density on CT and high signal on T2-weighted MRI in the subcortical white matter and basal ganglia [4, 5, 6, 7, 8], occasional infarcts [11, 12, 13] and intracerebral haemorrhage [14, 15]. Digre et al. [8] reported that nine of 10 eclamptic patients had abnormal

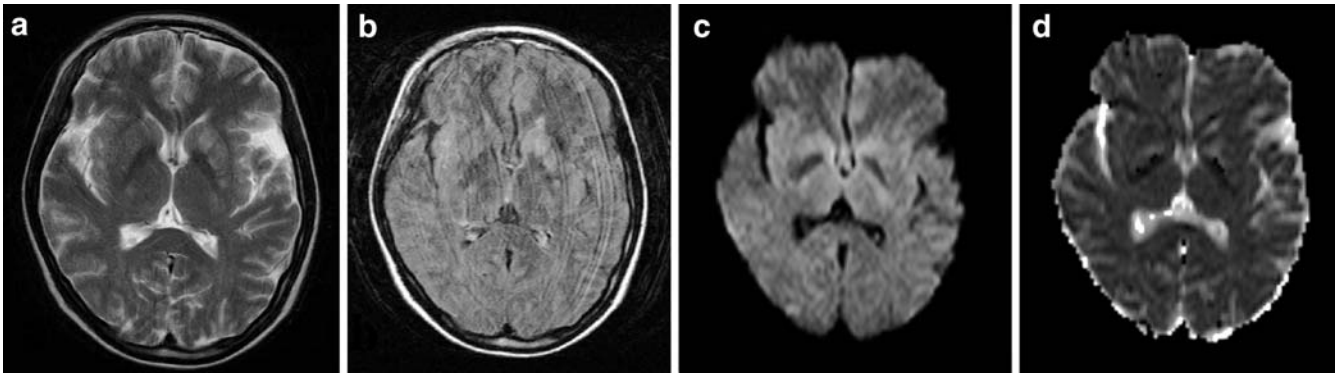

Fig. 1a–d Axial MRI through one level in patient 1. T2 weighted **a** fast spin-echo (FSE) **b** FLAIR images show slightly high signal in the left putamen and the head of the caudate nucleus. **c** Isotropic diffusion-weighted imaging (DWI) and **d** an apparent diffusion coefficient (ADC) map showed no difference from the right side

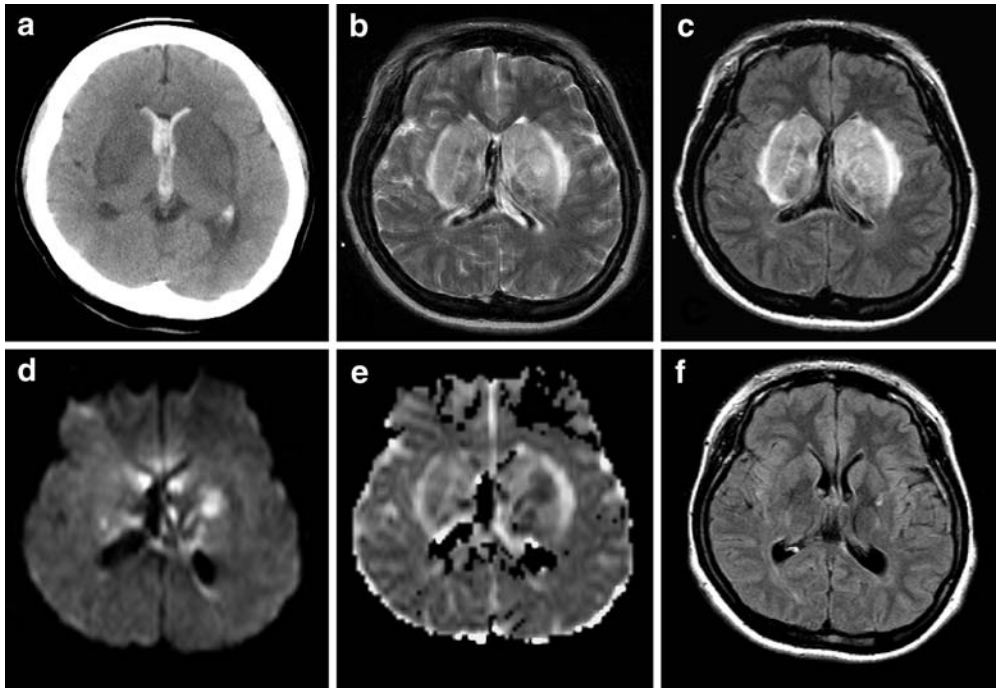

Fig. 2a–f Patient 2. **a** CT shows intraventricular haemorrhage and low density bilaterally in the basal ganglia and external capsule. T2-weighted **b** FSE and **c** FLAIR images show multiple high lesions in these areas. **d** DWI shows the very high signal in the left putamen. **e** ADC map showing low ADC in the left putamen, and increased ADC bilaterally in the external capsule. **f** FLAIR image 3 months later shows high signal in the left putamen, suggesting an infarct

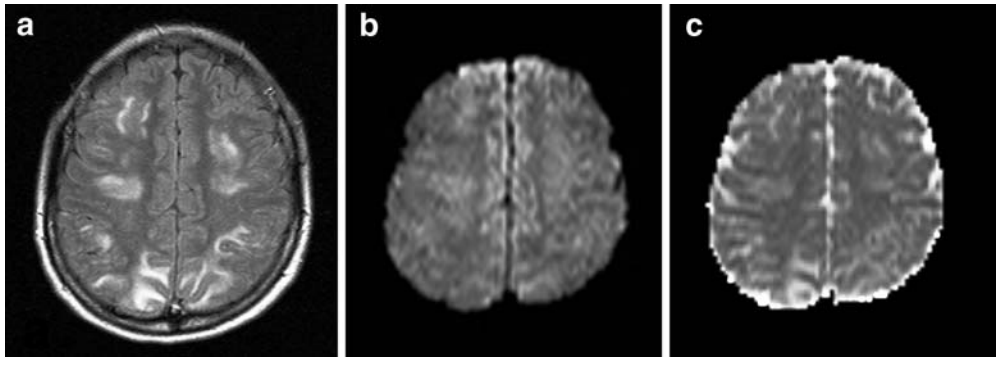

Fig. 3a–c Axial MRI in patient 3. **a** FLAIR image shows multiple high-signal lesions bilaterally in frontal and parietal subcortical white matter. **b** On DWI the frontal lesions give slightly high and the parietal lesions slightly low signal **c** ADC map: all lesions show slight increase in ADC

Table 3 Diffusion-weighted imaging (DWI) and apparent diffusion coefficients (ADC) in lesions with long T2

Patient	Site	Signal on DWI[a]	ADC map	ADC ($\times 10^{-3}$mm^2/s)	ADC normal white matter ($\times 10^{-3}$mm^2/s)	Follow-up MRI
1	Left putamen	Isointense	Bright	0.963	0.805	None
2	Left putamen	High	Dark	0.681	0.779	Infarct
	Right putamen	High	Bright	1.079	0.779	Normal
	Left external capsule	Low	Bright	1.612	0.779	Normal
3	Right parietal	Isointense	Bright	1.340	0.795	None
4	Left putamen	High	Bright	0.960	0.837	Normal

[a]compared with normal white matter

signal on MRI; the one patient with normal MRI was examined 5 days postpartum. All our patients showed abnormalities and patient 1 had a very faint lesion on the day after onset (Fig. 1). This may have disappeared after few days, because other obvious lesions had returned to normal on follow-up in patients 4 and 5. Thus, MRI was abnormal in almost all patients with eclampsia during the acute phase.

We found abnormalities predominantly in the basal ganglia, and none was evident in the subcortical white matter of three patients. One report [8] described that the basal ganglia was involved in three of nine patients,

and summarised previous reports in which the basal ganglia were abnormal in five of 19 patients. Basal ganglia involvement was also associated with lesions in the white matter. The reason for these discrepancies with our results is unknown. In eclampsia, the lesions were mainly in the basal ganglia and subcortical white matter, bilaterally or unilaterally, and a number of patients have abnormalities only in the basal ganglia.

The basal ganglia are involved in uraemic encephalopathy [16, 17, 18], including glomerulonephritis, haemolytic-uraemic syndrome (HUS) and thrombotic thrombocytopenic purpura. Two hypotheses have been proposed to explain uraemic encephalopathy [19]. One is that the blood-brain barrier is penetrated due to endothelial injury by uraemic toxins, which increases the concentration of water in the brain parenchyma, causing oedema and neurological disturbances. Endothelial dysfunction has been considered one of the key causative mechanisms of pre-eclampsia [20, 21], and increased permeability of endothelial cells was demonstrated in pre-eclamptic patients [22]. Abnormal red blood cell morphology and elevated lactic dehydrogenase, which indicate microangiopathic haemolysis and endothelial damage, are associated with pre-eclampsia and eclampsia accompanied by brain oedema [23]. The other hypothesis is that intravascular microthrombi occlude cerebral arterioles or capillaries, causing focal hypoxia and associated oedema. In both theories, the basal ganglia are likely to be most affected because they are nourished by small, fragile end-arteries susceptible to insult from toxins or microemboli [19]. We postulate that the pathophysiology of eclampsia includes both increased permeability of small vessels due to endothelial injury and a breakdown in cerebrovascular autoregulation caused by hypertension.

Most eclamptic patients have reversible lesions with long T2 [6, 8], but some progress to infarction [11, 12, 13]. Therefore, it may be helpful to differentiate cytotoxic and vasogenic oedema in the acute phase. We found that areas giving abnormal signal on T2-weighted images varied on isotropic DWI, causing difficulties in assessment, because of T2 shine-through [24]. To assess such lesions, it was necessary to look at them quantitatively.

Three case reports have described eclamptic encephalopathy and ADC changes [9, 10, 11]. Two [9, 10] showed extensive bilateral lesions on T2-weighted images, in which an ADC map revealed an increased in ADC, suggesting vasogenic oedema; this returned to normal on follow-up. Another [11] described similar lesions in the brain, but an ADC map showed, while that many had an increased ADC, indicating, vasogenic oedema, some had small areas within them in which the ADC was decreased, suggesting cytotoxic oedema. These progressed to infarction on follow-up. Our findings were consistent with these. Quantitative diffusion measurements predicted irreversible changes in patients with other types of hypertensive encephalopathy [25, 26].

References

1. Lindheimer MD, Katz AI (1989) Preeclampsia: pathophysiology, diagnosis, and management. Annu Rev Med 40: 233–250
2. Cunningham FG, Lindheimer MD (1992) Hypertension in pregnancy. N Engl J Med 326: 927–932
3. Thomas SV (1998) Neurological aspects of eclampsia. J Neurol Sci 155: 37–43
4. Fredriksson K, Lindvall O, Ingemarsson I, Astedt B, Cronqvist S, Holtås S (1989) Repeated cranial computed tomographic and magnetic resonance imaging scans in two cases of eclampsia. Stroke 20: 547–553
5. Duncan R, Hadley D, Bone I, Symonds EM, Worthington BS, Rubin PC (1989) Blindness in eclampsia: CT and MR imaging. J Neurol Neurosurg Psychiatry 52: 899–902
6. Sanders TG, Clayman DA, Sanchez-Ramos L, Vines FS, Russo L (1991) Brain in eclampsia: MR imaging with clinical correlation. Radiology 180: 475–478
7. Manfredi M, Beltramello A, Bongiovanni LG, Polo A, Pistoia L, Rizzuto N (1997) Eclamptic encephalopathy: imaging and pathogenetic considerations. Acta Neurol Scand 96: 277–282
8. Digre KB, Varner MW, Osborn AG, Crawford S (1993) Cranial magnetic resonance imaging in severe preeclampsia vs eclampsia. Arch Neurol 50: 399–406
9. Schaefer PW, Buonanno FS, Gonzalez RG, Schwamm LH (1997) Diffusion-weighted imaging discriminates between cytotoxic and vasogenic edema in a patient with eclampsia. Stroke 28: 1082–1085
10. Engelter ST, Provenzale JM, Petrella JR (2000) Assessment of vasogenic edema in eclampsia using diffusion imaging. Neuroradiology 42: 818–820
11. Koch S, Rabinstein A, Falcone S, Forteza A (2001) Diffusion-weighted imaging shows cytotoxic and vasogenic edema in eclampsia. AJNR 22: 1068–1070
12. Lewis LK, Hinshaw DB, Jr., Will AD, Hasso AN, Thompson JR (1988) CT and angiographic correlation of severe neurological disease in toxemia of pregnancy. Neuroradiology 30: 59–64
13. Raps EC, Galetta SL, Broderick M, Atlas SW. (1993) Delayed peripartum vasculopathy: cerebral eclampsia revisited. Ann Neurol 33: 222–225
14. Sharshar T, Lamy C, Mas JL (1995) Incidence and causes of strokes associated with pregnancy and puerperium. A study in public hospitals of Ile de France. Stroke in Pregnancy Study Group. Stroke 26: 930-936
15. Drislane FW, Wang AM (1997) Multifocal cerebral hemorrhage in eclampsia and severe pre-eclampsia. J Neurol 244: 194–198
16. Jeong YK, Kim IO, Kim WS, Hwang YS, Choi Y, Yeon KM (1994) Hemolytic uremic syndrome: MR findings of CNS complications. Pediatr Radiol 24: 585–586

17. D'Aprile P, Farchi G, Pagliarulo R, Carella A (1994) Thrombotic thrombocytopenic purpura: MR demonstration of reversible brain abnormalities. AJNR 15: 19–20
18. Barnett ND, Kaplan AM, Bernes SM, Cohen ML (1995) Hemolytic uremic syndrome with particular involvement of basal ganglia and favorable outcome. Pediatr Neurol 12: 155–158
19. Port JD, Beauchamp NJ (1998) Reversible intracerebral pathologic entities mediated by vascular autoregulatory dysfunction. Radiographics 18: 353–367
20. VanWijk MJ, Kublickiene K, Boer K, VanBavel E (2000) Vascular function in preeclampsia. Cardiovasc Res 47: 38–48
21. Granger JP, Alexander BT, Llinas MT, Bennett WA, Khalil RA (2001) Pathophysiology of hypertension during preeclampsia linking placental ischemia with endothelial dysfunction. Hypertension 38: 718–722
22. Haller H, Hempel A, Homuth V, et al (1998) Endothelial-cell permeability and protein kinase C in pre-eclampsia. Lancet 351: 945–949
23. Schwartz RB, Feske SK, Polak JF, et al (2000) Preeclampsia-eclampsia: clinical and neuroradiographic correlates and insights into the pathogenesis of hypertensive encephalopathy. Radiology 217: 371–376
24. Burdette JH, Elster AD, Ricci PE (1999) Acute cerebral infarction: quantification of spin-density and T2 shine-through phenomena on diffusion-weighted MR images. Radiology 212: 333–339
25. Ay H, Buonanno FS, Schaefer PW, Le DA, Wang B, Gonzalez RG, Koroshetz WJ (1998) Posterior leukoencephalopathy without severe hypertension: utility of diffusion-weighted MRI. Neurology 51: 1369–1376
26. Mukherjee P, McKinstry RC (2001) Reversible posterior leukoencephalopathy syndrome: evaluation with diffusion-tensor MR imaging. Radiology 219: 756–765