Posterior reversible encephalopathy syndrome

A Shrivastava, N Yousaf, R Vaidhyanath

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a recently proposed distinct clinicoradiological entity. It is characterized by headache, visual disturbance, altered mental function, seizures and stupor in the clinical setting of acute elevation of blood pressure, eclampsia of pregnancy or following treatment with immunosuppressants such as cyclosporin and cisplatin. Magnetic resonance imaging (MRI) findings include bilateral cortical and subcortical oedema in the brain with a predominantly posterior distribution.

DISCUSSION

The clinical signs and neuroimaging findings in patients with PRES are consistent enough for this entity to be readily recognized (Hinchey et al, 1996). Patients often have non-localizing neurological symptoms such as headache, confusion, mental abnormalities, seizures and stupor. Visual abnormalities including cortical blindness have also been reported (Hinchey et al, 1996).

The pathophysiology of PRES is incompletely understood. One theory suggests that the lesions result primar-

Figure 1. Axial T2-weighted image of brain showing confluent areas of hyperintensity in the posterior parietal and occipital regions.

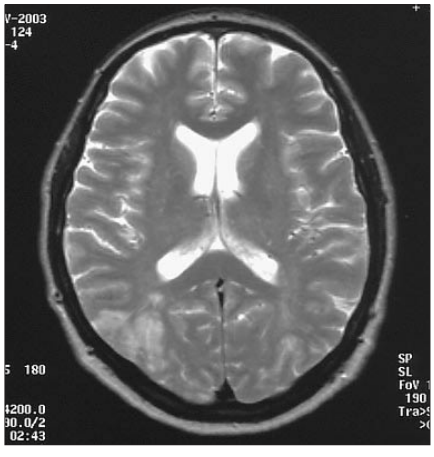
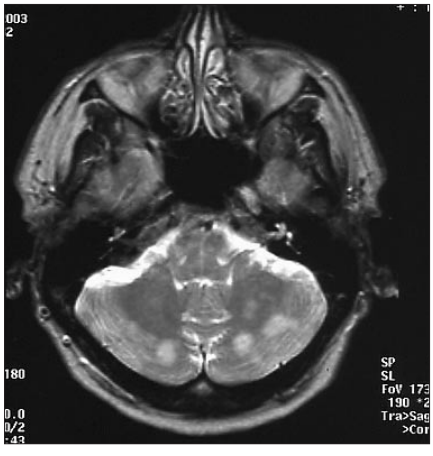


Figure 2. Axial T2-weighted image of brain showing hyperintense areas in both cerebellar hemispheres.



CASE REPORT

A 57-year-old man presented to the medical admissions unit of the authors' hospital with headache and blurred vision. His blood pressure recorded at the time of admission was 234/124 mmHg. Fundoscopy revealed early papilloedema and laboratory examination revealed raised levels of serum creatinine and urinary proteins. The patient reported a similar episode of headache and blurred vision 6 weeks before admission.

The patient underwent a magnetic resonance imaging (MRI) examination of the brain (*Figures 1–3*). This showed multiple bilateral cortical and subcortical areas of hyperintensity on both T2 and fluid attenuated inversion recovery (FLAIR) images in the posterior parietotemporal and occipital regions as well as in the cerebellum. The MRI findings were attributed to hypertensive encephalopathy and a diagnosis of posterior reversible encephalopathy syndrome (PRES) was made.

The patient was started on antihypertensive therapy and was discharged from hospital a few days later with a blood pressure of 120/75 mmHg. A month later, at his next outpatient appointment, his clinical symptoms had resolved and his blood pressure was recorded as being normal. A follow-up MRI of the brain (*Figure 4*) at the same time showed complete resolution of the signal abnormalities seen on the initial scan. This helped confirm the diagnosis of PRES.

Figure 3. Sagittal fluid attenuated inversion recovery (FLAIR) image of brain showing multiple areas of hyperintensity in the parieto-occipital region and cerebellum.

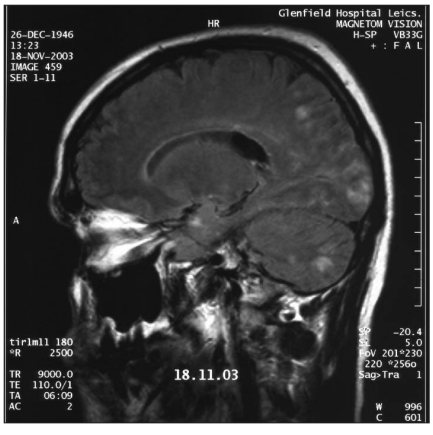
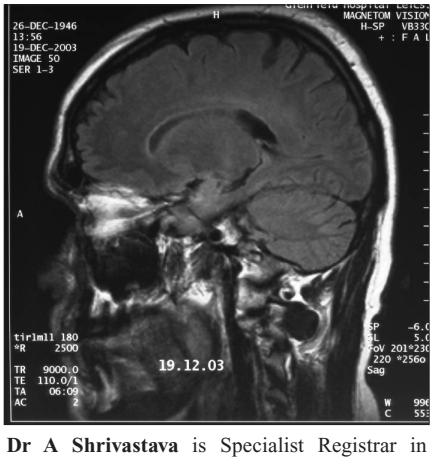


Figure 4. Follow-up magnetic resonance imaging scan showing complete resolution of the hyperintense areas seen on the initial scan.



Dr A Shrivastava is Specialist Registrar in Radiology in the Department of Radiology, **Dr N Yousaf** is Senior House Officer in the Department of Medicine and **Dr R Vaidhyanath** is Consultant Radiologist in the Department of Radiology, University Hospitals of Leicester, Leicester LE3 9QP

Correspondence to: Dr R Vaidhyanath

ily from vasogenic oedema which occurs when sudden elevations of blood pressure exceed the autoregulatory capability of brain vasculature. This results in breakdown of the blood–brain barrier and focal transudation of fluid from the arterioles into the extravascular compartment (Johansson, 1983). Another theory suggests that the lesions are characterized by cytotoxic oedema secondary to potentially reversible ischaemia (Ito et al, 1995). Current evidence indicates that both mechanisms could be operative in PRES. However, most investigators favour loss of autoregulation to be the underlying common pathogenetic mechanism in a number of causes of PRES including hypertensive encephalopathy and eclampsia (Casey et al, 2000). Diffusion-weighted imaging performed in this patient did not show evidence of cytotoxic oedema.

The preponderance of lesions in the posterior white matter in PRES has been attributed to vertebrobasilar circulation being sparsely innervated by autonomic nerve fibres which play a role in cerebral autoregulation (Edvinsson et al, 1976).

Neuroimaging is best performed by MRI using T2 and fluid attenuated inversion recovery (FLAIR) sequences (Casey et al, 2000). The typical findings on MRI are of bilateral hyperintense foci in the cortex and subcortical white matter of parieto-occipital regions on both T2 and FLAIR images. Involvement of cerebellum, brainstem and basal ganglia has also been reported.

The clinical symptoms and signs of PRES should be promptly recognized since the condition is readily treated and reversible. Imaging forms an essential component of the diagnosis and follow up of PRES. **HM**

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