

Cranial MRI: findings and diagnosis

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MRI findings and diagnosis

Please refer to page 105 of the December 2009 issue of the *SAJR* (<http://www.sajr.org.za/index.php/sajr/article/view/395/310>) for the clinical details and images. We congratulate Dr N Maharaj, who is a radiology registrar at King Edward VIII Hospital, Durban, for the precise and winning diagnosis, for which she receives an award of R1 000 from the RSSA. Dr Misser elaborates below on the causative condition.

Diagnosis

T2-weighted (Figs 1a and 1b) and FLAIR (Fig. 1c) sequences demonstrate bilateral paramedian thalamic and parieto-occipital subcortical hyper-intensity. Bilateral superior parietal gyral swelling is shown on the T1-weighted axial image (Fig. 2a). Nodular lepto-meningeal and perivascular post-gadolinium enhancement is seen in Figs 2b and 2c. In a patient with known colonic carcinoma, on chemotherapy, the most likely cause for abnormalities in such a distribution is chemotherapy-related posterior reversible encephalopathy syndrome (PRES).

PRES, as described in 1996 by Hinchey *et al.*,¹ is a clinico-radiological syndrome characterised by reversible symmetrical subcortical vasogenic oedema with a predilection for posterior cerebral arterial territories and watershed zones. In addition to the parietal lobes, the basal

ganglia, thalami, brain-stem and cerebellum may be involved.¹ There is classically absence of restricted diffusion in these affected areas, and patchy nodular pial enhancement is occasionally seen (as in the patient reported). Causes include hypertensive encephalopathy, eclampsia, combination chemotherapy, renal failure and septic shock.

Despite several theories, the pathogenesis of vasogenic oedema in PRES is not clear.² Relative paucity of sympathetic innervation of the vertebrobasilar circulation when compared with the internal carotid territories has been suggested as an explanation for the distribution frequently observed. Chemotherapeutic and immunosuppressive agents are known to increase porosity of the blood-brain barrier as well as to elevate intravascular pressure. P-glycoprotein expression in the blood-brain barrier is also affected in patients with brain metastases. Hypomagnesaemia, a consequence of some platinum-containing chemotherapeutic agents and eclampsia, has also been implicated in increasing blood-brain barrier permeability.

1. Hinchey J, Chaves C, Appignani B, *et al.* A reversible posterior leukoencephalopathy syndrome. *N Engl J Med* 1996; 334: 494-500.
2. Bartynski WS. Posterior reversible encephalopathy syndrome, part 2: Controversies surrounding pathophysiology of vasogenic edema. *Am J Neuroradiol* 2008; 29: 1043-1049.