NEFROLOGÍA. Volumen 27. Número 5. 2007

Encefalopatía posterior reversible

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Posterior reversible encephalopathy syndrome (PRES) is a recently described clinical and neuroimaging entity characterized by headaches, vomiting, visual field deficits, conscious changes and seizures, and by typical neuroimaging features corresponding to areas of subcortical edema, occasionally cortical, involving predominantly the occipital and parietal lobes of both hemispheres[1]. Hypertension, uraemia, cyclosporine A neurotoxicity and eclampsia are the most comon etiologies of PRES[1]. Although its pathophysiology is currently unknown, two main theories are considered: cerebral hyperperfusion, which considers the existence of vasogenic edema; and great vessels vasospasm, which points the presence of citotoxic edema[2]; these two mechanisms may occur sequentially[2,3]. Tomodensitometric features are variable and unspecific; thus MRI is the gold-standard method to establish the precise diagnosis of PRES, which permits a timely therapeutic intervention essential to reverse clinical manifestations[4]. Typically, lesions occur predominantly in the posterior white matter of subcortical region, and some involvement of the cortex can also exist. Generally, these lesions are hyperintense on T2 and hypointense on T1-weighted images or isointense on diffusion-weighted images traducing vasogenic edema[4]; thus MRI shows cerebral edema in tipical location and allows distinguishing between vasogenic and citotoxic edema, contributing to the knowledge of pathophysiology of PRES[2-4]. The reversibility of this syndrome depends on timely diagnosis and therapy and therefore it should be kept in mind in the differential diagnosis of seizures or coma on chronic kidney disease patients.

An example was a young hypertensive chronic kidney disease patient on peritoneal dialysis, brought to the emergency room comatous with generalized tonic-clonic seizures; at admission his blood pressure was 194/112 mmHg; the cerebral computorized tomography scan showed a diffuse pattern of brain edema, not conclusive, and cerebral magnetic resonance imaging (MRI) (fig. 1) was performed and evidenced subcortical edema of the parietal, occipital and temporal lobes. These changes were suggestive of hypertensive or metabolic encephalopathy. Anti-hypertensive therapy and hemodialysis allowed complete recovery, including restarting peritoneal dialysis as self-carer.

In this case, we consider that PRES was associated with hypertensive crisis following anti-hypertensive therapy (minoxidil) interruption since peritoneal dialysis provided adequate small molecules clearance and ultrafiltration (weekly urea Kt/V of 2, 4 and ultrafiltration of 600 mL after 4 hours dwell of 3.86% glucose). We highlight the impressive neuroimaging features and stress the importance of considering this syndrome in the differential diagnosis of seizures or coma in chronic kidney disease patients.

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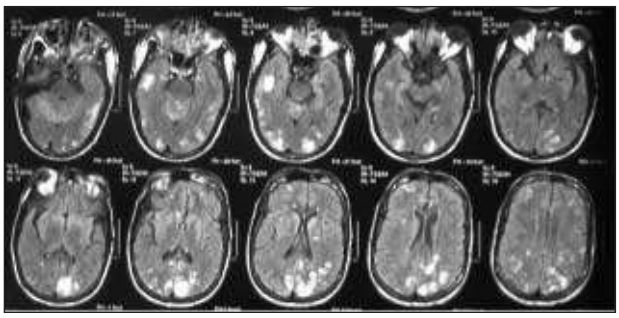

Fig. 1.—Cerebral Magnetic Resonance Imaging features: subcortical areas with hypersignal in FLAIR and hyposignal in T1, predominantly in the temporal and parieto-occipital lobes, suggesting edema. Brain cortex is relatively spared.

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