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Posterior reversible encephalopathy syndrome associated with nephrotic syndrome

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Kabicek and colleagues described a case of nephritic-syndrome-associated posterior reversible encephalopathy syndrome (PRES) (Kabicek, *et al.* 2010). This adds to the accumulating evidence that PRES can be associated with disorders other than hypertension. However, we wonder how the authors would explain the neuroimaging findings unsuggestive of vasogenic oedema.

PRES (also named reversible posterior leukoencephalopathy syndrome, RPLS) represents a clinicoradiological syndrome characterized by vasogenic oedema as revealed by apparent diffusion coefficient (ADC) map of diffusion-weighted imaging (DWI) (Bartynski, 2008). The pathogenesis of PRES has been suggested to be autoregulation failure and endothelial dysfunction (Sharma, *et al.*). Although magnetic resonance (MR) T2 weighed imaging (T2WI) or fluid attenuated inversion recovery images (FLAIR) may show some abnormalities, they are not specific for vasogenic oedema (Fugate, *et al.* 2010). Atypical cases are not rare (Sharma, et al), in which PRES may be neither posterior, reversible, nor confined to white matter, whereas they should be diagnosed after prudent exclusion of confounding disorders, including metabolic encephalopathy, inflammatory demyelinating diseases, vasculitis, etc. In this context, reversible clinical manifestations as well as radiological vasogenic oedema might contribute to a certain diagnosis of typical PRES. However, in this case, the increased signal intensity as shown in DWI along with the normal signal intensity in ADC map, failed to support posterior lesions to be vasogenic oedema. Cytotoxic oedema, which may result from ischema, inflammation, toxicosis, etc was not fully excluded from oedema. Although clinical manifestations and radiological findings were reversible, the above pathological process as well as demyelination may also present with reversible changes in neuroimaging. Furthermore, the pathological foci involved more gray than white matter, which did also not support the diagnosis of typical PRES. In terms of cortex involvement, electroencephalography is indispensable to exclude epileptic unconsciousness, which may lead to cytotoxic oedema in the gray matter. Additionally, although uncommon, cerebral embolism with spontaneous recanalization can cause reversible DWI changes in posterior brain. In light of this consideration, electrocardiogram might be of help to exclude cardiogenic emboli.

In summary, PRES represents a clinical and radiological syndrome arising from multiple clinical disorders. The atypical clinical and radiological manifestations do not support this case as a typical PRES.

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