Reversible Cerebral Vasocostriction Syndrome and Subarachnoid Hemorrhage; Which Occurs First?

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To the Editor Noda and colleagues described a case with reversible cerebral vasocostriction syndrome (RCVS) and subarachnoid hemorrhage (SAH), as well as posterior reversible encephalopathy syndrome (PRES) and cerebral infarction (1). The case is interesting; however, we would argue against their hypothesized scenario with regard to the pathogenic course of the disease.

In this case, SAH, localized in the right superior frontal lobe, was noted by computed tomography (CT) at the very early stage. Although digital subtraction angiography (DSA) on the same day demonstrated an unruptured aneurysm in the left internal carotid artery, no typical segmental vasocostriction in the anterior or posterior circulation was identified. DSA performed later revealed multiple segments of irregularity consisting of narrowed areas of multiple branches of internal carotid arteries, indicative of RCVS. It is thus more plausible that RCVS and generalized seizures arise from SAH per se. The unruptured aneurysm, along with a history of hypertension, supports an aneurysmal etiology of SAH, despite the findings that a minimal amount of subarachnoid blood overlied the cortical surface and that the vasocostriction was widespread. However, we cannot rule out the possibility that RCVS may in turn aggravate SAH manifestations.

PRES was initially described by Hinchey et al (2) in 1996 as a clinico-radiological syndrome. Different conditions may attribute to the pathogenesis of PRES, including eclampsia, hypertensive encephalopathy, renal diseases and the use of immunosuppressive drugs (3). The pathophysiological mechanism of PRES is vasogenic edema (4), which requires apparent diffusion coefficient (ADC) maps elaborated from magnetic resonance diffusion-weighted imaging (DWI) for identification (5). Since an acknowledged set of diagnostic criteria for PRES is lacking, we are concerned about the diagnosis of PRES in this case. As discussed by the authors, some of the right parietooccipital hypertensive lesions on FLAIR images were hypointense, and the left frontal hyperintense lesion on FLAIR images was isointense on DWI, which are indicative of vasogenic edema. We are strongly against this assumption, because ADC maps are well acknowledged to be more sensitive and specific for vasogenic edema than DWI.

In summary, the clinical course of the above-mentioned case can be explained by the scenario that hypertension-associated SAH followed by vasospasm leads to RCVS and cerebral infarction.

The authors state that they have no Conflict of Interest (COI).

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