Prolonged Reversible Vasospasm in Cyclosporin A–Induced Encephalopathy

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Summary: The mechanisms responsible for cyclosporin-induced encephalopathy remain controversial. Herein we present a case of cyclosporin-induced encephalopathy with unusually prolonged vasospasm, which might have contributed to the slow recovery of the patient.

Vasospasm has been proposed as one of the mechanisms of cyclosporine A (CsA)–induced encephalopathy (1), although the first case of demonstrated vasospasm was reported only recently (2). T2-weighted MR images typically show hyperintense lesions involving the subcortical regions of the occipital, posterior temporal, and parietal lobes (1, 3, 4); these sometimes involve the cortical regions (2). Herein, we report a further case of CsA-induced encephalopathy with vasospasm of a protracted course.

Case Report

A 35-year-old woman receiving CsA for 1 month for myelodysplastic syndrome associated with autoimmune hemolytic anemia experienced a sudden onset of blurred vision, headache, and loss of balance. Two days later, she had left-sided limb weakness and sensory impairment; therefore, the CsA was discontinued. Upon her admission to the hospital 4 days after symptom onset, her blood pressure was 130/80 mm Hg. The neurologic examinations revealed left hemianopia, a limitation of the leftward gaze, quadriparesis (muscle power grade 2 of 5 in the left limbs and grade 4 of 5 in the right limbs), generalized hyperreflexia, and bilateral Babinski signs. The whole-blood CsA level was 189 ng/mL (range, 75–350 ng/mL). The hemo-

globin level was 11.1 g/dL, and the platelet count was 23

cases of cyclosporin-induced encephalopathy are similar to those of eclampsia and hypertensive encephalopathy (3, 4, 6, 7). We report a patient with CsA-induced encephalopathy with both cortical and subcortical involvement. A multiple segmental arterial vasospasm was a possible underlying cause and responsible for the lesions at the bilateral parieto-occipital regions. The low intralesional ADCs were consistent with cytotoxic edema (5).

Discussion

The underlying mechanism and pathophysiologic features of CsA-induced encephalopathy have not been well defined, and they might be multifactorial (3, 4, 6, 7). We report a patient with CsA-induced encephalopathy with both cortical and subcortical involvement. A multiple segmental arterial vasospasm was a possible underlying cause and responsible for the lesions at the bilateral parieto-occipital regions. The low intralesional ADCs were consistent with cytotoxic edema (5).

The clinical and neuroimaging findings of CsA-induced encephalopathy are similar to those of eclampsia and hypertensive encephalopathy (8). Because vasospasm is an important mechanism for eclampsia and hypertensive encephalopathy (9, 10), it is also proposed as a mechanism responsible for CsA-induced encephalopathy (1). However, the first patient with possible vasospasm was reported by Bartynski et al only recently (2). Their patient had bilateral MCAs and PCAs with a reduced caliber, as assessed by using MRA, and vasospasm or vasculitis was suggested. The patient died 5 days after imaging, and the pathologic examination showed both cortical ischemic necrosis and subcortical vasogenic edema with no disease of the involved vessels. Therefore, vasospasm rather than vasculitis was responsible for the reduced caliber of the involved arteries. Our patient was similar to that of Bartynski et al, except that ADC mapping showed only cytotoxic edema. The possibility of vasculitis was less likely in our patient because of negative ANA results and a normal complement titer. Hypertension was not found in our

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Therefore, the reversible, protracted vasospasm (at least 14 days) in our patient might have been one of the possible mechanisms of CsA-induced encephalopathy, and it might have accounted for the long clinical course of recovery in our case. This observation is in contrast to what is seen in patients with CsA-induced encephalopathy and vasogenic edema; these patients usually have a reversible course and a better outcome (3). MRAs and diffusion-weighted images are helpful in understanding the pathophysiology of CsA-induced encephalopathy. Prolonged vasospasm might be rare, but it is nevertheless an existent mechanism responsible for the cytotoxic edema of both the gray matter and the white matter in certain patients.

References

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