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LETTER TO THE EDITOR

Ataxic hemiparesis in a patient with posterior cortical borderzone infarction

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Dear Sir,

A 76-year-old woman was admitted to our institution with weakness and lack of coordination along the left side of her body. Review of her medical history revealed that she had hypertension and diabetes mellitus, had undergone stent-assisted coil embolization for a saccular aneurysm on the right distal internal carotid artery, and had taken aspirin, antihypertensive drugs, and glucose-lowering medication. Physical examination revealed a blood pressure of 150/90 mmHg, but no other specific findings were noted. Neurological examination indicated a slight impairment of motor power in the patient's left arm (Medical Research Council grade 4+) and leg (grade 4+). With regard to cerebellar function, finger-to-nose and heel-to-shin tests showed observable dysmetria of the arm and leg, respectively. All aspects of the sensory system including proprioception were normal. Tandem gait was affected; the patient tended to tilt slightly towards the left. These findings suggested ataxic hemiparesis (AH), and hence, lacunar stroke was strongly suspected. However, the results of magnetic resonance imaging performed on admission and on the fourth day after admission showed distinct right posterior cortical borderzone infarction between the middle and posterior cerebral arterial territories (Fig. 1a). A wedge-shaped lesion was seen on the right posterior parietal area (Fig. 1b) alongside a lesion resembling a 'string of beads' on the right posterior centrum semiovale. The previously undertaken stent-assisted coil embolization was evident from the initial computerized tomographic angiography results (Fig. 2a); thereafter, irregular stenosis in the stent was observed on magnetic resonance angiography performed on the fourth day after admission (Fig. 2b). We carried out cardiac evaluation procedures, including electrocardiography and transthoracic echocardiography, but there was no indication of cardioembolism. Finally, we concluded that the posterior cortical borderzone infarction was caused by thromboembolism from the stenotic lesion in the stent. The patient fully recovered and was discharged on the seventh day after admission. Dual antiplatelets with aspirin and clopidogrel were prescribed for secondary prevention.

This is the first reported case of AH caused by cortical borderzone infarction; a thorough review of literature indicated no reports of borderzone infarction causing AH [1]. In this study, we deduced that hemiparesis is associated with the lesion on the centrum semiovale, which involves the pyramidal tract. Ataxia was assumed to be associated with a wedge-shaped posterior parietal lesion. This is a common site for the occurrence of cortical borderzone infarctions [2]. Previously, kinetic ataxia has been reported in a patient with an acute posterior parietal infarction [3]. This association is very rare, but the interruption of cerebellar projections on the cortex was postulated as a possible mechanism [3]. Prior to this study, an autopsy report showed parietal ataxia related to the right postcentral gyrus in the arm [4]. In this article, however, proprioceptive impairment rather than dysmetria was regarded as the neurological basis of ataxia [4]. The neurologic deficits in our patient suggest pyramidal involvement near the centrum semiovale and kinetic ataxia due to a posterior parietal lesion, both of which represent AH. This association of AH with posterior cortical borderzone infarction is novel; AH is traditionally

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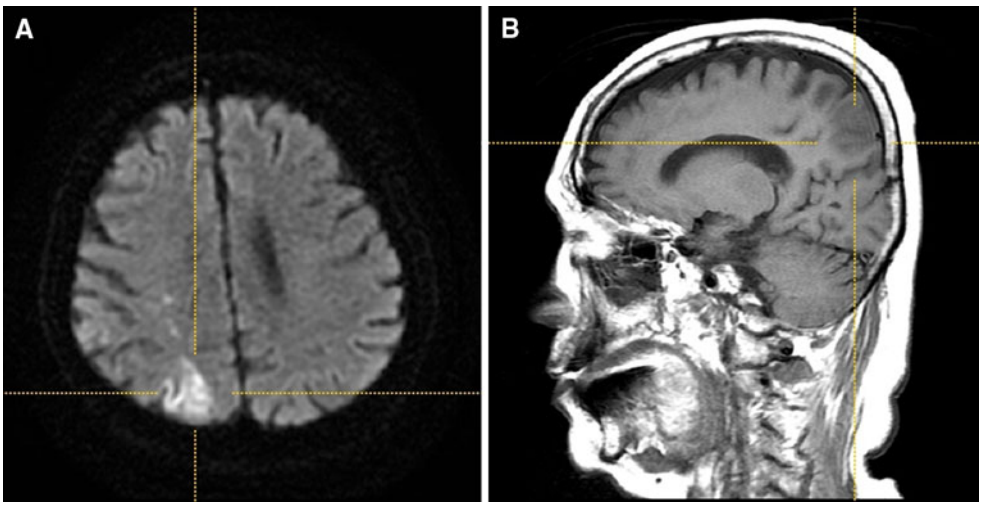

Fig. 1 **a** Findings of diffusion-weighted imaging revealed right posterior cortical borderzone infarction, while neurological examination indicated that the patient had ataxic hemiparesis, a lacunar syndrome. **b** A T1-weighted sagittal image taken on the fourth day after admission shows a right posterior parietal lobe lesion, which is thought to be associated with kinetic ataxia

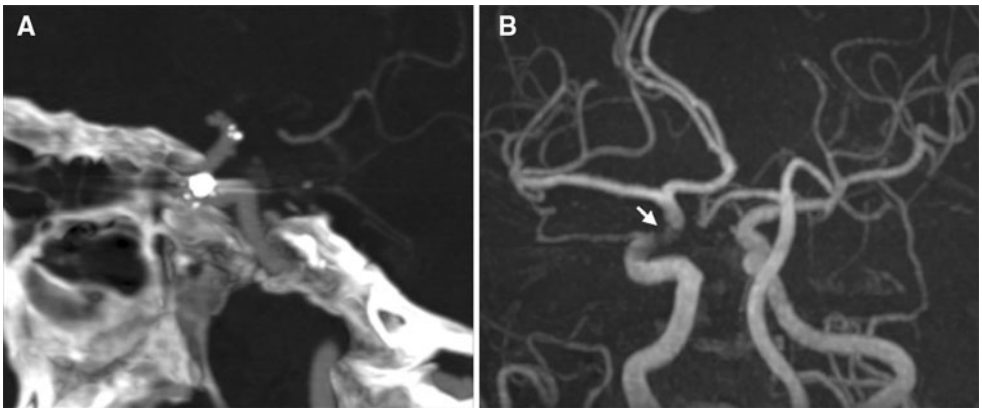

Fig. 2 **a** A sagittal maximum-intensity projection image of computed tomographic angiography performed on admission shows the status of the previous stent-assisted coil embolization of an aneurysm on the right distal internal carotid artery. **b** Three-dimensional time-of-flight angiography performed on the fourth day after admission revealed stenosis in the stent (*arrow*), which might induce artery-to-artery embolism and cause the cortical borderzone infarction

associated with lesions in the pons, the internal capsule, or the corona radiata [5–7].

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