Symptomatic Lacunar Infarct Accompanied with Posterior Reversible Encephalopathy Syndrome: A Case Report

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We present a 48-year-old man with a history of hypertension, who suddenly noticed dysarthria and right hemiparesis. Diffusion-weighted MRI at 1 day after the onset showed a small high-intensity region in the left corona radiata, indicating the acute phase of lacunar infarction. Fluid attenuation inversion recovery images showed extensive hyperintense lesions predominantly in the white matter of the fronto-temporoparietal lobes and pons, indicating posterior reversible encephalopathy syndrome (PRES). In addition, T2*-weighted gradient-echo images showed multiple small round hypointense lesions in white matter and basal ganglia, indicating cerebral microbleeds. This is a rare case of symptomatic lacunar infarction accompanied with both PRES and microbleeds, which may suggest that the pathophysiology of PRES is related to hypertension.

Key words: Cerebral microbleeds, hypertension, Lacunar infarct, posterior reversible encephalopathy syndrome, MRI

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is usually a reversible radiological and clinical entity that presents with a variety of symptoms ranging from headache, seizures, visual disturbances, and altered mental status to loss of consciousness [1]. The most common causes of the syndrome are severe hypertension, eclampsia, bone marrow or solid organ transplantation, immunosuppressive drug toxicity, renal failure, infection, and various autoimmune diseases [2, 3].

Typical cerebral imaging abnormalities of PRES are often symmetric and appear predominantly in the posterior white matter; they usually do not show enhancement, and exhibit restricted diffusion [2, 3]. On diffusion-weighted images (DWI), PRES lesions are usually isointense or less commonly hyperintense due to a T2 shine-through effect, and they are hyperintense on apparent diffusion coefficient (ADC) maps, suggesting vasogenic edema [4]. Restricted diffusion, representing cytotoxic edema, is also observed occasionally, and is associated with irreversible infarction located within PRES lesions [4].

To our knowledge, there are only few reported cases of symptomatic cerebral infarction accompanied with PRES [5]. Herein, we describe a rare case of symptomatic lacunar infarction accompanied with both PRES and cerebral microbleeds (CMB), which may suggest that the pathophysiology of PRES is related to hypertension.

CASE REPORT

A 48-year-old man suddenly noticed dysarthria and right hemiparesis, and was admitted to Tokai University Hospital. He did not have severe headache, or nausea, or consciousness disturbance. He had a history of hypertension and dyslipidemia 5 years ago, but had received no treatment. He is social drinker and non-smoker, with no remarkable family history. On admission, his temperature and blood pressure were 36.2℃ and 219/153 mmHg, respectively. He was alert. Neurological examination revealed mild right hemiparesis including the face, with no sensory impairment. Tendon reflexes on the right side were exaggerated and a pathological plantar reflex was not elicited.

Laboratory examinations revealed high LDL-cholesterol (171 mg/dl), aldosterone (467 pg/ml), noradrenalin (1543 pg/ml), dopamine (189 pg/ml) and renin (42 ng/ml/hr). On the other hand, renal function (BUN 14 mg/dl, Cr 1.10 mg/dl), HbA1c (5.7%), thyroid functions, autoimmune antibody titers, and tumor markers were within normal ranges. An abdominal echogram revealed no abnormal lesion, including adrenal glands. No specific cause of secondary hypertension was apparent, even to an endocrinologist.

On MRI at 1 day after the onset (lower part in Fig. 1), we detected a small lesion in the left corona radiata (diameter: less than 15 mm) showing hyperintense on DWI and hypointense on ADC, indicating the acute phase of lacunar infarction. MR angiography showed no stenotic lesion in the carotid and intracranial arteries. There were extensive hyperintense lesions predominantly in white matter of the fronto-temporoparietal

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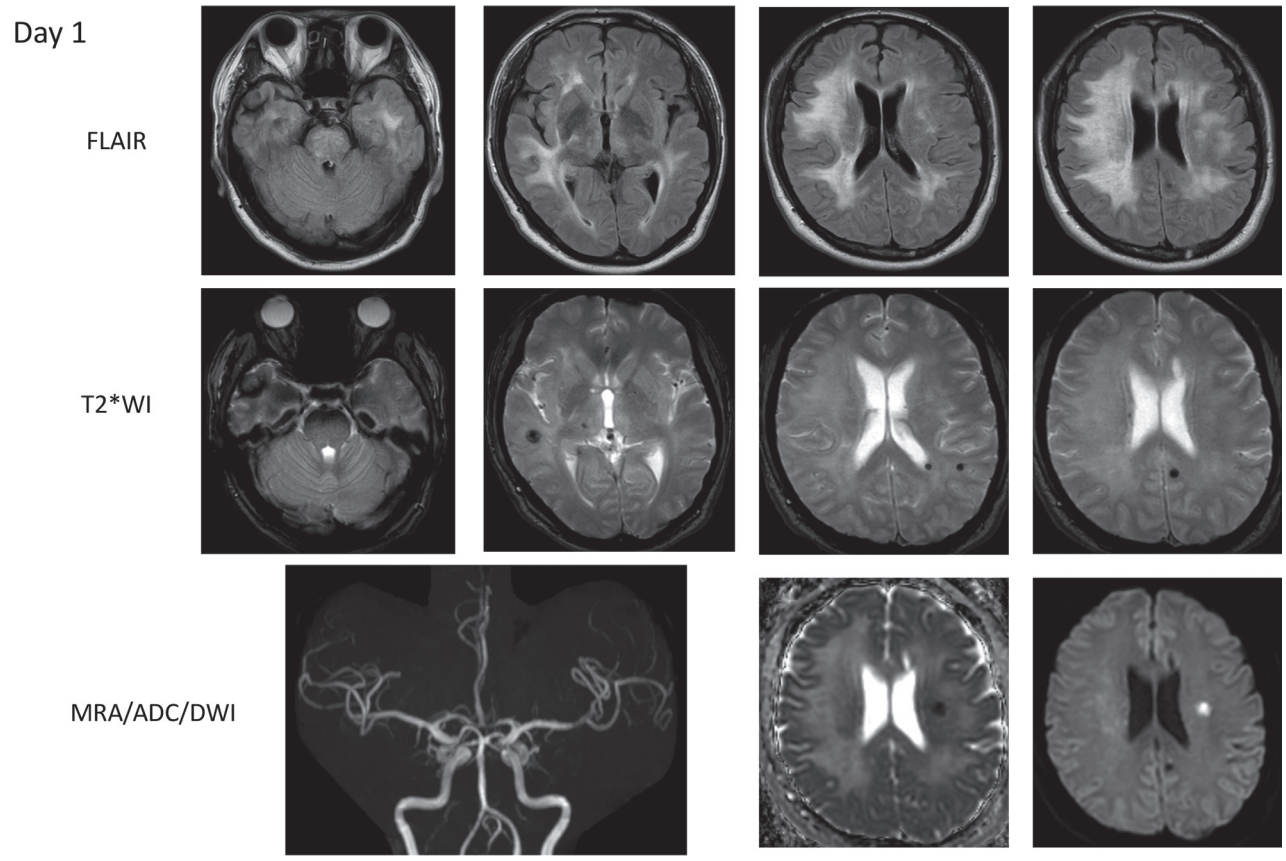

Fig. 1 MRI at 1 day after the onset
Upper part: Extensive hyperintense lesions on FLAIR images were predominantly seen in white matter of the fronto-temporoparietal lobes and pons, indicating PRES.
Middle part: Multiple small round hypointense lesions on T2*WI were seen in white matter and basal ganglia, indicating CMB.
Lower part: Hyperintense lesions on DWI and hypointense lesions on ADC were seen in the left corona radiata, indicating the acute state of lacunar infarction. MR angiography showed no stenotic lesion in the carotid and intracranial arteries.

lobes and pons on fluid attenuation inversion recovery (FLAIR) images (upper part in Fig. 1) and hyperintense lesions on ADC (lower part in Fig. 1), indicating PRES. Furthermore, multiple small round hypointense lesions on T2*-weighted gradient-echo images (T2*WI) were seen in white matter and basal ganglia, indicating CMB (middle part in Fig. 1).

We used an antihypertensive agent (nicardipine) intravenously to lower his blood pressure, and his systolic blood pressure fell to below 140 mmHg within 3 days. Then, we administered oral antiplatelet (aspirin), anti-hypertensive (amlodipine), and lipid-lowering (atorvastatin) agents. At day 20 after the onset, hyperintense lesions in white matter of fronto-temporoparietal lobes and pons on FLAIR images had become less prominent (upper part in Fig. 2). CMB on T2*WI (middle part in Fig. 2) did not change, but hyperintense regions on DWI had become blurred (lower part in Fig. 2), compared with those at Day 1. His weakness gradually improved and he was transferred to the rehabilitation hospital after a month. At 2 months after the onset, he had no deficit and returned to his job.

DISCUSSION

We present a patient with symptomatic lacunar infarction accompanied with both PRES and CMB, which is a rare case with interesting implications for the pathophysiology of PRES. PRES was first described in 1996 in patients with radiological findings suggestive of white matter edema in the posterior parieto-temporo-occipital region [1]. Since then, there have been many reports suggesting a spectrum of causes, mechanisms, and imaging findings of this syndrome [1–6]. Typical MRI findings are symmetric hyperintensity in the bilateral parieto-occipital cortical-subcortical white matter (98%) on FLAIR images [6]. However, the frontal lobe (68%), inferior temporal lobe (40%), cerebellum (30%), basal ganglia (14%), brain stem (13%), and deep white matter (18%) can also be involved [6]. The involvement of brain stem or basal ganglia with sparing of the subcortical regions was named "central-variant" PRES (4%) and involvement of the splenium of the corpus callosum was noted in 10% of the patients [3, 7, 8]. On DWI, lesions are usually isointense or less commonly hyperintense due to a T2 shine-through effect and hyperintense on ADC maps, suggesting vasogenic edema [6–8].

An unusual case of unilateral or asymmetrical PRES mimicking a middle cerebral artery infarction has been reported [5]. Restricted diffusion representing cytotoxic edema is also observed occasionally, and is associated with irreversible infarction [4]. However, these irreversible ischemic changes are observed inside the PRES area, and are not symptomatic. In the pres-

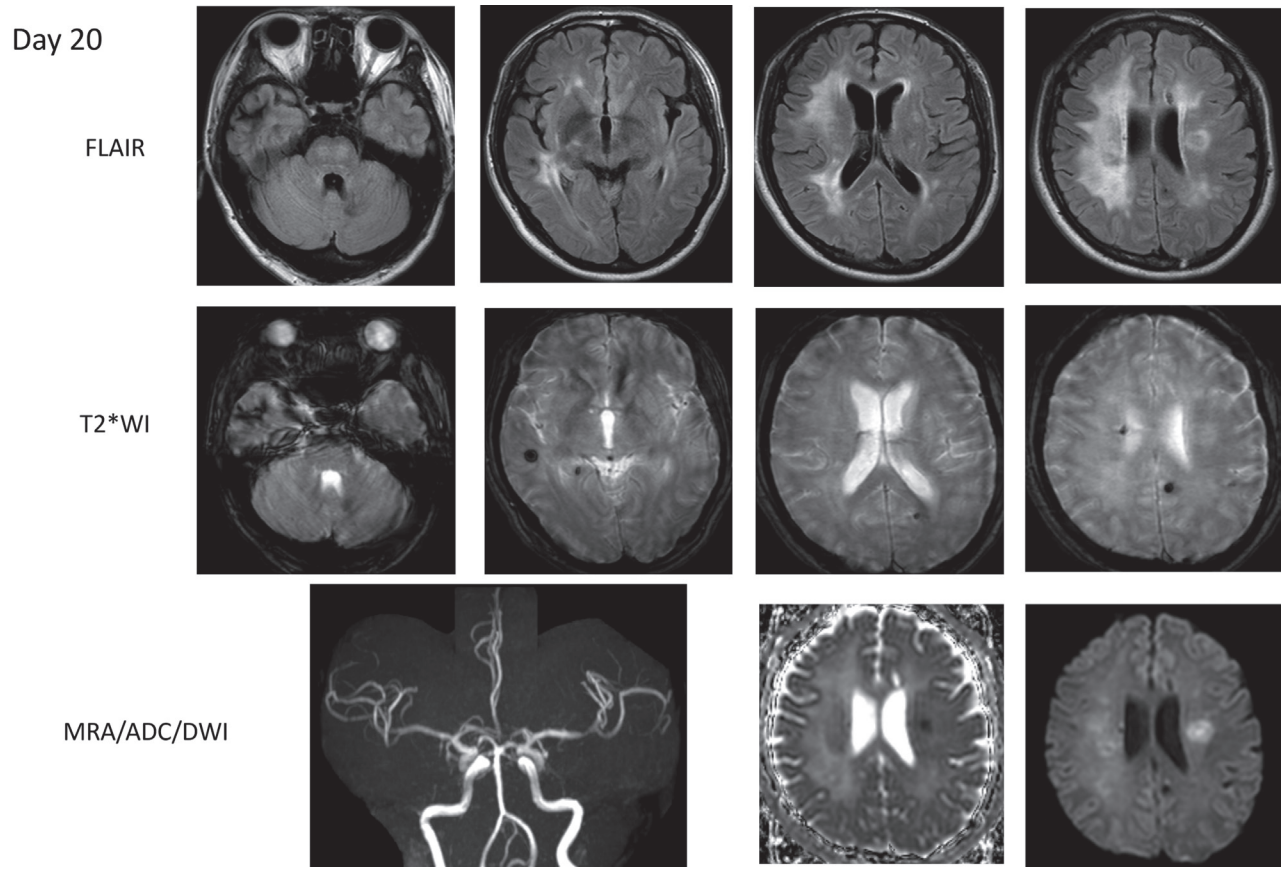

Fig. 2 MRI at 20 day after the onset
Upper part: Hyperintense lesions on FLAIR image were less prominent, compared with those at Day 1.
Middle part: CMB on T2*WI was unchanged.
Lower part: MR angiographical findings were unchanged, but hyperintense lesions on DWI became blurred, compared with those at Day 1.

ent patient, right hemiparesis was the chief complaint, without severe headache or consciousness disturbance, and MRI indicated the acute phase of typical lacunar stroke. Symptomatic acute stroke located outside of the PRES area has not been reported in the literature.

Regarding the pathogenesis of PRES, autoregulatory failure and endothelial damage have been proposed. Autoregulation is the maintenance of a constant blood flow in the brain by arteriolar constriction and dilatation, regulated by sympathetic, cholinergic, and myogenic mechanisms [9]. Sudden elevation of systemic blood pressure could exceed the capacity of autoregulation, causing arterioles to dilate, and resulting in brain hyperperfusion, blood brain barrier breakdown, and extravasation of fluid and blood products. This could also explain the posterior predilection of the syndrome, as the arteries of the posterior circulation have relatively poorer sympathetic innervation [1]. Hypertension may have been the central pathophysiologic phenomenon in our patient, and the onset of PRES may have coincided with his blood pressure exceeding the limits of autoregulation. Endothelial damage may then have led to the occurrence of CMB and acute lacunar stroke.

CMB is defined as small, rounded, homogeneous, hypointense foci on T2*WI or susceptibility-weighted imaging [10]. Histopathological correlation studies suggest that radiologically defined CMBs generally correlate well with small collections of blood-breakdown products, that have presumably leaked from damaged small vessels into the brain parenchyma [11]. Detection of CMB has clinical implications with respect to the diagnosis of underlying small vessel disease, the safety of antithrombotic use, and the risk of symptomatic intracerebral haemorrhage, cognitive impairment and dementia [11]. In PRES, the overall incidence of cerebral hemorrhage was 15.2% on CT and conventional MRI, and the incidence of minute hemorrhages was 7.9% [12]. In lacunar infarction, the incidence of CMB was 62.1% [13]. Thus, although the coexistence of CMB, lacunar infarct, and PRES in a hypertensive patient is clinically rare, it could result from underlying endothelial damage. A recent report that a patient with symptomatic lacunar infarction developed white matter hyperintensity [14] may also support this fundamental mechanism.

In conclusion, we present a rare case of symptomatic lacunar infarction accompanied with both PRES and CMB, which may suggest that the pathophysiology of PRES is related to hypertension.

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