Reversible Pontine MRI Lesion in Acute Thalamic Infarct: Reversible Encephalopathy due to Hypertension?

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Posterior reversible encephalopathy syndrome associated with hypertension rarely presents with predominant involvement of the brainstem and is relative sparing of the supratentorial regions. A relative paucity of brainstem signs and symptoms, despite extensive neuroimaging abnormalities therein, support the diagnosis. Although elevation of blood pressure is common in acute cerebral infarction, concomitant brainstem edema has not been reported. We describe here the clinical and neuroimaging features of an unusual brainstem hyperintensity associated with acute ischemic stroke. The neuroimaging abnormalities improved after stabilization of blood pressure, distinguishing this syndrome from brainstem infarction.

Key Words: Posterior reversible hypertensive encephalopathy, Reversible pontine edema, Acute ischemic stroke
CASE REPORT

A 40-year-old man presented with blurred vision, dysarthria, and right-sided limb weakness. The medical history revealed hypertension, diabetes mellitus, and transient dysarthria that had first appeared 2 months previously. He had not taken any antihypertensive drugs. His blood pressure was 220/150 mmHg on admission, and an ophthalmologic examination showed bilateral retinal hemorrhages but no evidence of papilledema. He was alert and had moderate right-sided hemiparesis. His EKG was normal, and complete blood counts were unremarkable. Serum electrolytes, BUN, and creatinine were within the normal ranges, but the serum glucose level was 229 mg/dl. Random urine glucose was 4+, but protein was admission revealed an acute lacunar infarction in the left lateral thalamus and an increased T2-weighted signal in negative, An MRI scan performed 4 days after the the pons (Fig. 1). There also were multiple small infarcts in the bilateral basal ganglia. The hypertension was initially managed by intravenous labetalol. The blood pressure remained above 180 mmHg in systole or 110 mmHg in diastole for the first week, and then reduced to 140/85 mmHg by the end of the second week. A follow-up MRI scan performed 2 weeks later showed persistent thalamic infarction but improved signal intensities of the pons (Fig. 2). FLAIR images showed the same findings as T2-weighted images, but we were unable to obtain diffusion-weighted images (DWI) because of the absence of equipment.

DISCUSSION

The pathophysiologic changes in hypertensive encephalopathy are still unclear, being currently described as edema associated with marked vasodilatation or microinfarcts in the arterial territories. Previous studies have found that the causes of PRES include hypertension, eclampsia, renal failure, and the use of immunosuppressive drugs (e.g., cyclosporine A neurotoxicity). The most common abnormality on neuroimaging is presumed edema involving the white matter in the posterior regions of the cerebral hemispheres, especially in the parieto-occipital areas. Vasogenic edema in PRES involves predominantly the posterior circulation territories, but anterior circulation structures are also frequently involved. FLAIR, T2-weighted imaging, and DWI can successfully differentiate acute whole brainstem vascular diseases, and thus play a pivotal role in patient management. Vasogenic edema syndromes that may mimic acute infarction clinically and on conventional imaging include hypertensive encephalopathy, eclampsia, human immunodeficiency virus encephalopathy, hyperperfusion syndrome following carotid endarterectomy, venous sinus thrombosis, acute central pontine myelinolysis, neoplasm, and mitochondrial encephalopathy, lactic acidosis, and stroke.
Although previous investigations of PRES have detected brainstem abnormalities in MRI, these have been observed usually in association with predominantly supratentorial white matter changes. In only a few case reports has the presence of brainstem abnormalities on neuroimaging been the principal feature of PRES. Hypertensive brainstem encephalopathy must be differentiated from brainstem ischemia or infarction, pontine glioma, infectious encephalitis, as well as central pontine myelinolysis. The lack of significant neurologic symptoms and signs referable to the brainstem, combined with the rapid clinical improvement upon treatment of hypertension, is correlated with the pattern of brainstem involvement on MRI.

The present case of reversible pontine MRI lesion in acute ischemic stroke may be an atypical presentation of PRES, although we were unable to determine whether the lesion was edema because we could not obtain the DWI. Further studies are needed to evaluate the incidence of hypertensive encephalopathy—especially hypertensive brainstem encephalopathy—in acute stroke patients.

REFERENCES