Hypertensive Encephalopathy: Isolated Pons Involvement Mimicking Central Pontine Myelinolysis

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Sir,

We are presenting here a case of an elderly, 60-year-old man who presented to the emergency department in an unconscious state. He had no previous history of diabetes mellitus or hypertension, and there was no history of trauma. On examination he was found to have bilateral papilloedema. His blood pressure at the time of presentation was 220/150 mmHg. The routine laboratory investigations were within normal limits. The serum potassium and sodium levels were normal, the renal function tests were normal and the blood sugar level was normal.

MRI of the brain was performed, and it demonstrated an isolated high signal on the T2 weighted and fluid attenuated inversion recovery sequences that involved only the central pons with sparing the periphery (Figs. 1A, B). There was no restricted diffusion on diffusion weighted imaging. The differential diagnosis included posterior reversible syndrome and central pontine myelinolysis; however, the blood sodium on admission was normal. Treatment was then initiated with 10 mg nifedipine and the blood pressure gradually returned to normal. The patient regained his consciousness a day after the start of administering antihypertensive agents. The clinical follow-up at 2 months revealed a blood pressure of 140/90 mmHg. Follow-up by MRI was performed at 2 months, and it showed significant resolution of the radiological abnormalities (Figs. 2A, B). The clinical features and resolution with administering appropriate antihypertensive treatment favor the diagnosis of RPLS secondary to hypertensive encephalopathy (HE). This case is unusual in that the distribution of was entirely confined to the pons in a man with no previous history of hypertension.

Hypertensive encepahlopathy is a medical emergency, and the patients may present with headache, altered alertness and behavior ranging from drowsiness to stupor, seizures, vomiting and mental abnormalities, including confusion and diminished spontaneity and speech, along with abnormalities of visual perception that are due to the accelerated hypertension.

The imaging findings depend upon the severity of the hypertension. In mild cases of hypertensive encephalopathy, the imaging features are those of edema, usually within the cortex and the subcortical white matter of the parietal, occipital and temporal lobes, and to a lesser degree, the posterior frontal lobes, typically with bilaterality, although not always with perfect symmetry. In more severe cases, there will be involvement of the subcortical white matter and this may extend to the frontal, posterior temporal, cingulate and central sylvian regions, as well as to the cerebellar white matter. There is a varying degree of thalamic, insular and pontine involvement in the more severe cases (1).

The pathogenesis of HE is that the auto-regulatory mechanisms that control the cerebral blood flow are exceeded, resulting in hyper-perfusion. The consequent over-distension of the cerebral vessels, the breakdown of the blood brain barrier and ultimately, the extravasation of fluid into the interstitium all cause vasogenic edema (2).

In most cases, the changes of hypertensive encephalopathy represent reversible vasogenic edema, which can be seen on T2-weighted images, and restricted diffusion is not seen on the diffusion-weighted imaging (DWI) and the apparent diffusion coefficient (ADC) maps. Hypertensive encephalopathy that manifests as a reversible increased signal isolated to the pons on T2-weighted images is extremely uncommon (3−5).

The differential diagnosis for such pontine T2 hyperintensity includes pontine glioma, ischemic and radiation changes (generally irreversible conditions), as well as central pontine myelinolysis (CPM) and demyelinating disorders such as multiple sclerosis, acute disseminated

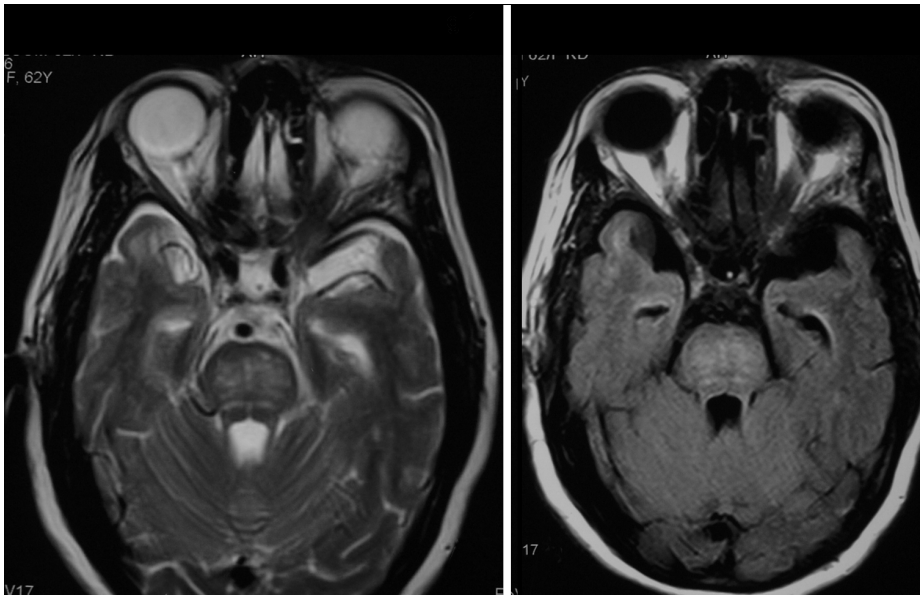

Fig. 1. MRI at the time of admission showing hyperintensity involving the central pons with sparing of the periphery on the T2WI (**A**) and FLAIR sequence (**B**).

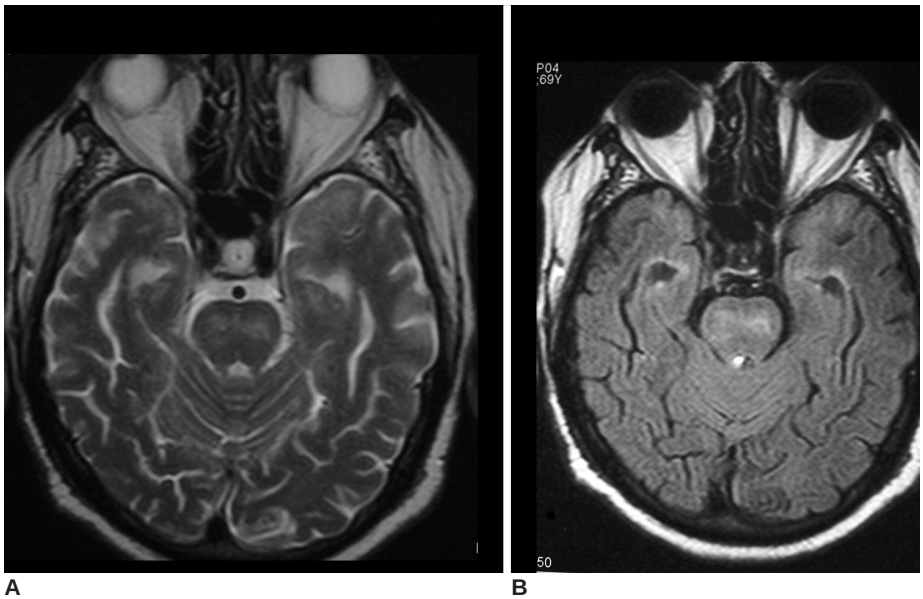

Fig. 2. Follow up MRI after two months showing significant resolution of hyperintensity within the pons on the T2WI (**A**) and FLAIR sequence (**B**).

encephalomyelitis and rhomb-encephalitis. In CPM electrolyte imbalances provide a clue for the diagnosis, where as for glioma, there will be an expansion and mass effect.

In conclusion, clinical recognition of brainstem HE may be difficult. The features of a lack of correlation between the severity of the radiological abnormality and the clinical status, combined with the rapid resolution following antihypertensive treatment, should suggest the diagnosis. It is important for the radiologist to be familiar with the imaging abnormalities of this life-threatening, but treatable condition.

References

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