Hypertensive Encephalopathy Involving the Brainstem and Deep Structures: A Case Report

Chin-Shih Fong

Abstract- Hypertensive encephalopathy rarely presented with widespread edema in the cerebral white matters, deep structures and whole brainstem. A 80-year-old woman manifested as high arterial blood pressure, visual disturbance, severe headache, nausea, and vomiting. T2-weighted and fluid-attenuated inversion recovery magnetic resonance imaging showed high signal-intensity lesions in the cerebral white matter, cerebellum, basal ganglia, thalamus, and brainstem. Diffusion-weighted brain MRI did not show hyperintense signals in these lesions. These findings suggested the pathological basis of vasogenic edema. After control of hypertension, clinical symptoms and these edematous lesions on MRI gradually reduced.

Key Words: Hypertensive encephalopathy, Reversible posterior leukoencephalopathy syndrome, Magnetic resonance imaging

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INTRODUCTION

Hypertensive encephalopathy is an acute neurological syndrome characterized by an abrupt rise in blood pressure, confusion, headache, vomiting, visual change, seizures and focal neurological signs[1,2]. Hypertensive encephalopathy is usually reversible, if treated promptly. The term, reversible posterior leukoencephalopathy syndrome (RPLS), has been used to describe the characteristic neuroimaging features in patients with acute hypertension resulting from a variety of conditions[3]. The most common abnormality on neuroimaging in RPLS-associated white-matter edema, is predominantly in the posterior parietal-temporal-occipital regions of the brain[4,5]. Extension of the edema into the brainstem, basal ganglia, and cerebellum had rarely been reported[6]. We present a patient with hypertensive encephalopathy and serial MRI-documented reversible lesions throughout cerebral hemisphere white matter, brainstem, thalamus, basal ganglia, and cerebellum.

CASE REPORT

A 80-year-old, previously demented woman complained of progressive weakness in four limbs for 10 days before admission. Her past medical history was remarkable except for poorly controlled hypertension. She had no history of alcoholism and did not take any

From the Department of Neurology, Buddhist Dalin Tzu Chi General Hospital, Chiayi, Taiwan.
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Reprint requests and correspondence to: Chin-Shih Fong, MD. Department of Neurology, Buddhist Dalin Tzu Chi General Hospital, No. 2, Min Sheng Road, Dalin, Chiayi, Taiwan.
E-mail: csfong@tcts.seed.net.tw

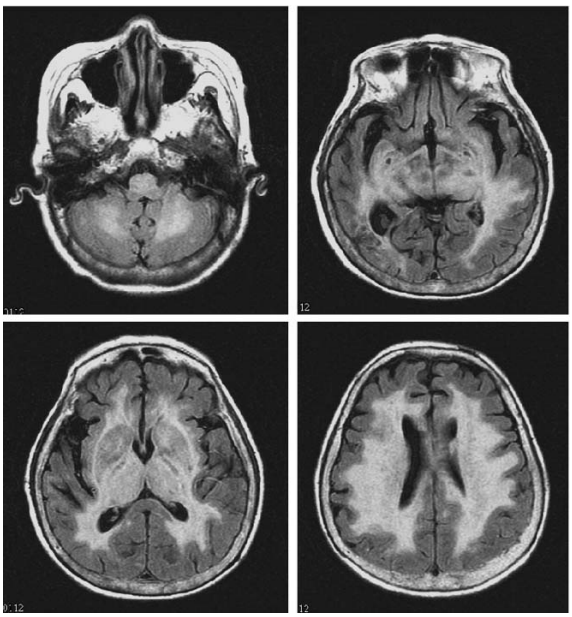

Figure 1. A FLAIR MRI on admission shows extensive edema in the hemisphere white matters, brainstem, thalamus, basal ganglia, and cerebellum.

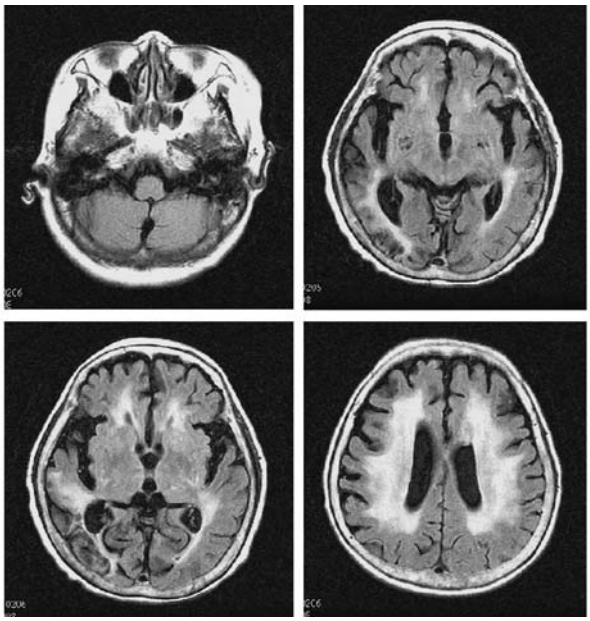

Figure 2. Follow-up FLAIR MRI 3 weeks later shows gradual disappearance of the lesions with high signal intensities on the previous examination.

immunosuprressive agents. On the day of admission, she had severe headache, nausea, vomiting and blurred vision. Later she became disoriented and was brought to our hospital. She did not develop seizure. Blood pressure at admission was 250/130 mm Hg. Pulse rate was 80 beats per min and regular. She did not have heart murmur or respiratory rales. The mental status was confused and Glasgow coma scale showed E3V4M6. Ophthalmological examination revealed bilateral retinal hemorrhage and papilledema. Neurological examination showed normal functions of cranial nerves and quadriparesis with increased deep tendon reflexes. Plantar responses were extensor bilaterally. An electrocardiogram showed left ventricular hypertrophy. Chest roentgenogram found cardiomegaly. Blood biochemistry test revealed fasting sugar 139 mg/dL, normal liver function, renal function and electrolyte levels. Blood count and urinalysis were within normal limit. Cranial T2-weighted and fluid-attenuated inversion recovery (FLAIR) MRI performed after arrival demonstrated widespread edema in the bilateral cerebral white matter, cerebellum, basal ganglia, thalamus, and brainstem (Fig. 1). T1-weighted images detected hypointensities in these areas. There was no obvious midline shift, no hemorrhage, and the ventricles are not dilated. Diffusion-weighted MRI did not show the increased intensity in these edematous lesions. MRA revealed poor opacification of the branches of bilateral middle cerebral arteries.

The hypertension was managed with beta blockers, angiotensin converting enzyme inhibitors and calcium channel blockers and her blood pressure returned to 140/70 mmHg within 2 days. A hyperosmolar glycerol agent intravenously to lessen brain edema was also administered. She resumed consciousness gradually over a period of 3 days and quadriparesis had resolved completely at the time of discharge 7 days later. On repeated MRI performed on day 21, the severity of brain edema reduced (Fig. 2).

DISCUSSION

The clinical presentation of this patient is suggestive of hypertensive encephalopathy, including headache, confusion, and blurred vision associated with severe

hypertension and subsequent improvement after normalization of blood pressure. Previous studies on hypertensive encephalopathy have reported brain edema predominately in the posterior portions of the white matter. The neuroimaging features of this patient were unusual in that the edema involved cerebellum, basal ganglia, thalamus, and brainstem in addition to cerebral white matters. The distributions of edema seemed to correlate with severity of blood pressure[4,7,8]. Mild elevation of hypertension produced edema in the supratentorial white matter with little involvement of the infratentorial compartments. Severe elevation of hypertension created extensive supratentorial edema extending into brainstem, basal ganglia, thalamus and cerebellum. In normotensive and spontaneously hypertensive rats, the upper limit of the autoregulatory plateau of cerebral blood flow in the thalamus is higher than that in the cerebral cortex[9]. Thus, severe elevation of hypertension may be necessary for the dysfunction of autoregulation and breakdown of the blood-brain barrier in the deep structures such as thalamus, basal ganglia and brainstem. This patient had poorly controlled hypertension before and maybe suffered from severe elevation of hypertension in 10 days before admission. That could underlie the mechanisms of edemain both supratentorial and infratentorial areas.

The abnormality on neuroimaging in this patient was widespread edema involving the cerebral white matter, predominantly in the posterior parietal-occipital regions of the brain. The calcarine and paramedian occipital-lobe structures are spared, a fact that distinguishes RPLS from bilateral infarction of the posterior cerebral artery territory. Simultaneous infarction in the territory of bilateral posterior cerebral arteries occurs in patients with embolism to the rostral basilar artery, known as “top of the basilar syndrome”[10]. The calcarine regions are invariably involved and often there are accompanying thalamic and midbrain infarcts. In this patient who had follow-up MRI scans, there was improvement of white-matter abnormalities, suggesting transient edema rather than infarction[11].

Parietal-occipital edema is a recognized feature of hypertensive encephalopathy. The reason for this regional pathological variation in hypertensive encephalopathy remains unclear. One hypothesis suggests that sympathetic innervation of the anterior cerebral vasculature may be protective, and conversely, the relative lack of sympathetic innervation in the vertebrobasilar vasculature may predispose the parietal-occipital region to the development of cerebral edema in hypertensive encephalopathy[12].

Central pontine myelinolysis (CPM) is a demyelinating disease of the pons and often associated with demyelination of other areas in the central nervous system. The term ‘osmotic demyelination syndrome’ (ODS) is used for pontine and extrapontine myelinolysis (EPM)[13]. Usually, EPM accompanies CPM but can occur in isolation. Rapid correction of severe chronic hyponatremia with hypertonic saline, chronic alcoholism and administration of immunosuppressive agent cyclosporine have been known to cause ODS. Magnetic resonance imaging of the ODS shows lesions in the pons, cerebellum, lateral geniculate body, thalamus, putamen, cerebral cortex and subcortex. Clinical features reflect damage to the involved areas and include spastic tetraparesis, pseudobulbar paralysis, locked-in syndrome, cerebellar ataxia, dystonia, rigidity, bradykinesia, and tremors. ODS often has a grave prognosis. This patient had normal serum sodium level, no history of alcohol abuse, no taking of cyclosporine, and improvement of outcome which did not support the diagnosis of ODS.

Thrombosis of cerebral veins and venous sinuses usually develops in relation to infections of the ears or paranasal sinuses, taking of birth control pills, postoperative or postpartum states, hypercoagulable states in cancer, cyanotic congenital heart disease, and meningitis[14]. The common MRI appearance is hyperintense signal within dural sinus and bilateral parasagittal fronto-parietal hemorrhage which is not within any arterial territory distribution. This patient did not illustrate any one of these condition, not suggesting venous thrombosis.

The exact cellular mechanisms leading to RPLS have two divergent theories. Initially, investigation suggested that RPLS patients would develop vasospasm secondary to sudden and marked rise of blood pressure, which leads to ischemia of the brain tissues. However,

the imaging studies showed no signs of cytotoxic edema or infarction and the reversibility of imaging abnormalities after an appropriate immediate treatment were not consistent with the hypothesis of vasospasm and cerebral ischemia[15]. Recent investigations have suggested that RPLS resulted from a rapid rise of blood pressure which might exceed the normal autoregulation of cerebral blood flow. This breakthrough in autoregulation produces dilation of the cerebral arterioles with opening up of endothelial tight junctions and leakage of plasma and red cells into the extracellular space, leading to vasogenic edema[16]. Diffusion-weighted MR imaging may be used to determine whether the edema in this syndrome is cytotoxic or vasogenic in origin[17]. Regions with cytotoxic edema demonstrate diffusion coefficients that are decreased compared to those of normal white matter. Conversely, regions with vasogenic edema demonstrate diffusion coefficients that are increased compared to those of normal white matter and these lesions of vasogenic edema appeared as iso- or hypointensity on the diffusion weighted images.

The diffusion-weighted MR imaging in this patient did not show increased intensity in the regions with brain edema. These findings indicate that the lesions were vasogenic edema.

In conclusion, the most common neuroimaging abnormalities of hypertensive encephalopathy is edema involving the white matter in the posterior regions of the cerebral hemisphere. Atypical presentation of hypertensive encephalopathy extending into brainstem and deep structures should be promptly recognized, since it is reversible and readily treated by controlling blood pressure.

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