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〈原著・症例〉

MRI studies in two cases of hypertensive encephalopathy

Kazuya SAKO, Naoki ITO, Chiharu TANAKA, Tomoko YOKOO,
Masahiro MIZOBUCHI, Nobuto MURAKAMI, Nobuo NAKAJIMA, Satoshi SHIBUYA,
Jyoji NAKAGAWARA*, Rihei TAKEDA*, Jun-ichi NAKAMURA* and Katsumi SUEMATSU**.

*Department of Neurology and * Neurosurgery, Nakamura Memorial Hospital, Sapporo, Japan and ** Hokkaido Brain Research Foundation, Sapporo, Japan.*

Summary : Magnetic resonance imaging (MRI) findings were analyzed in two patients with hypertensive encephalopathy. MRI demonstrated focal cortical and subcortical lesions of hyperintense T 2 signal and hypointense T1 signal lesions with diffuse brain swelling. Focal lesions were hardly explained by involvements of major arterial supplies. There were no neurological focal signs suggesting dysfunctions in the abnormal areas of MRI. These MRI studies further support the hypothesis that hypertensive encephalopathy is induced by vasogenic edema during breakthrough of cerebral autoregulation. Prompt diagnosis and reduction of blood pressure are key points for improving the clinical condition. MRI better defines the cerebral involvements in detail and would help proper diagnosis and therapeutic decisions.

Key words :
- hypertensive encephalopathy
- magnetic resonance imaging
- vasogenic edema

Introduction

Hypertensive encephalopathy is a neurologic syndrome characterized by rapid and severe rise in blood pressure associated with headache, nausea and vomiting, visual changes, convulsions, and disturbance of consciousness[1-4]. The disease may progress to a life-threatening condition if prompt diagnosis and proper treatment are not undertaken. Various etiologies have been suggested for the pathophysiology of the disease such as cerebral edema[5], vasospasm[2], breakdown of autoregulation of cerebral blood flow[6], and intravascular coagulation[7]. Pathologic reports have documented brain swelling with focal and diffuse edema, fibrinoid necrosis of the small vessels, and microinfarction with petechial hemorrhage[8]. Computed tomographic (CT) scans of the patients with hypertensive encephalopathy have demonstrated focal and diffuse cortical and white matter lesions[9-11]. MRI apparently has a great advantage in demonstrating slight changes by brain edema compared with CT scan[12]. We present two cases of hypertensive encephalopathy and discuss the clinico-radiological features of the disease.

Case Presentation

Case 1.

A35-year-old woman began complaining of headache four days before admission. The headache usually occurred on awakening and was often accompanied by nausea and vomiting. She saw a neurosurgeon and was administered an anti-hypertensive drug for her hypertension. No definite abnormalities were noted in a cranial CT scan and CSF at this time. However, her symptoms continued with multiple exacerbations. The patient was, therefore, admitted to our hospital for further evaluation of her symptoms. She had no history of hypertension before this episode. Physical examinations on admission revealed no

中村記念病院 神経内科 *同脳神経外科 **財団法人 北海道脳神経疾患研究所

abnormality except for hypertension (BP 220/120 mmHg). Fundi were normal without papilledema or hypertensive retinopathy. She had clear mental state and no focal deficits on neurological examination. Laboratory results were as follows : WBC 6900/mm^3, RBC 550 × 10^4/mm^3, Hb 15.3 g/dl, Ht 47.4 %, platelet 31.7 × 10^4/mm^3, total protein 8.9 g/dl, ALP 7.6 K-AU, GOT 41 K.U. , GPT 37 K.U., LDH 346 IU/dl, LAP 208 mIU/ml, γ - GTP 48 mIU/ml, total bilirubin 0.5 mg/dl, BUN 14.8 mg/dl, creatinine 0.8 mg/dl, Na 142 mEq/l, K 3.6 mEq/l, Cl 100 mEq/l, CRP (-), ANA (-), and RA (-). Mild dehydration was suggested by these blood data. CSF showed cell count 4 /mm^3, protein 88 mg/dl, glucose 65 mg/dl. Urinalysis disclosed no abnormality. EEG performed on the admission day was normal. CT performed on admission demonstrated low density areas in the bilateral occipital lobes and left cerebellum without contrast enhancement (**Fig. 1**). She was treated initially with sublingual administration of nifedipine (Ca-antagonist) followed by oral administration of the drug (30mg/day). All her symptoms disappeared by the next day. MRI on the second day of admission, however, revealed hyperintense T2 signal and hypointense T1 signal areas in bilateral occipital areas and left cerebellum with moderate brain swelling (**Fig. 2**). There were no contrast enhancement in MRI using gadolinium on the third day of admission (**Fig. 3**). Angiogram on the second day of admission revealed irregularities of arterial walls suggesting vasospasms, but no definite stenosis or obstruction (**Fig. 4**). Her blood pressure was effectively controlled around 120/80mmHg with nifedipine (30mg/day) alone and no headache or other symptoms was noted. MRI obtained on the 15th day of admis-

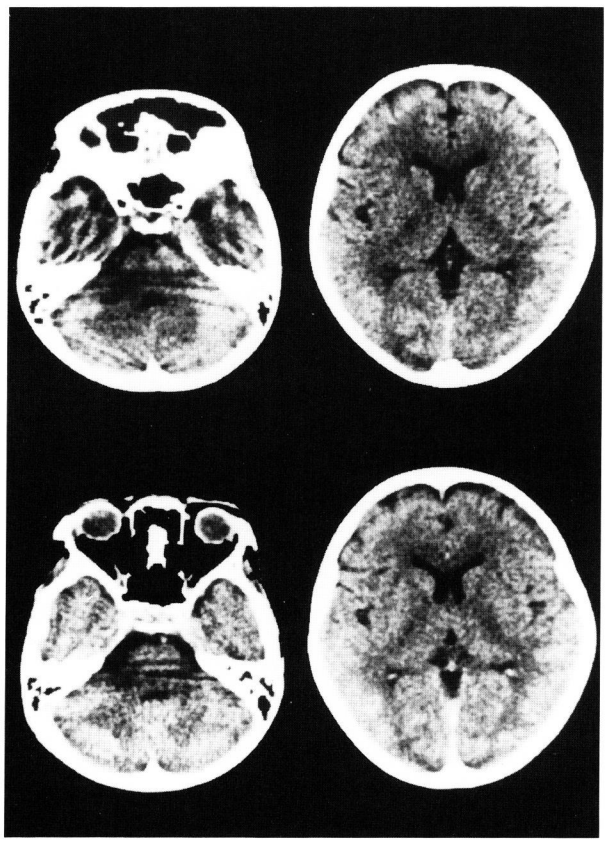

Fig. 1 A CT on the day of admission demonstrated decreased density areas in bilateral occipital areas and left cerebellum. No contrast enhancement was observed (lower panel).

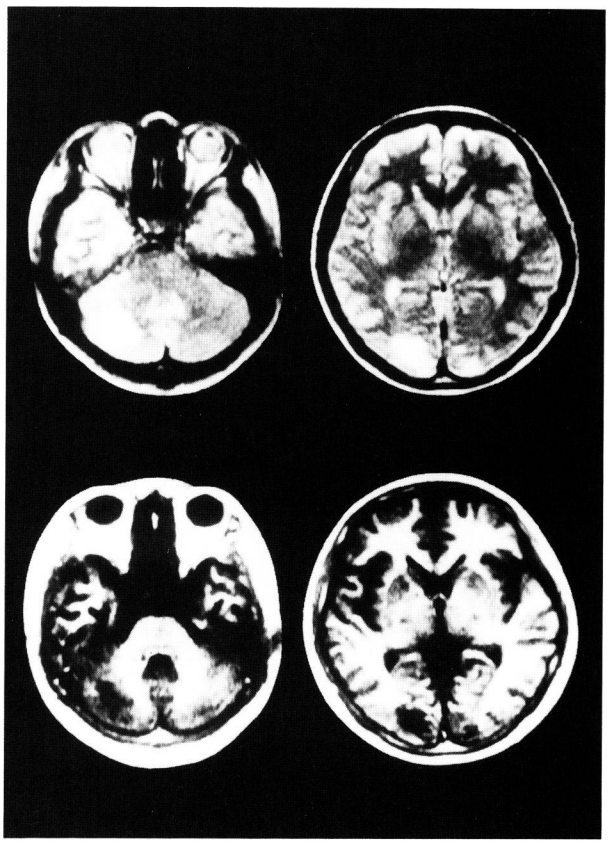

Fig. 2 T2-weighted MRI on the second day of admission reveals high intensity areas in bilateral occipital areas and left cerebellum. T1-weighted images (IR) demonstrated abnormal areas as low intensity areas. MRI scans were performed with T2-weighted spin echo (SE2000 /80) sequence and T1-weighted images (IR ; inversion recovery, 1500/44/300) in the axial plane used by a Sanyo SNR15P (permanent magnetic, 0.15T) system (Osaka, Japan).

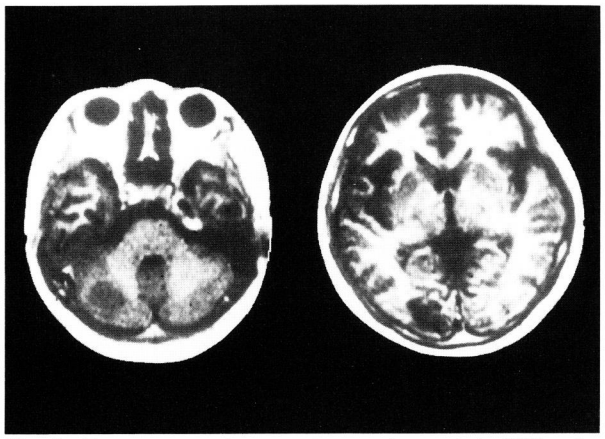

Fig. 3 No contrast enhancement was observed in MRI using gadolinium on the third day of admisson.

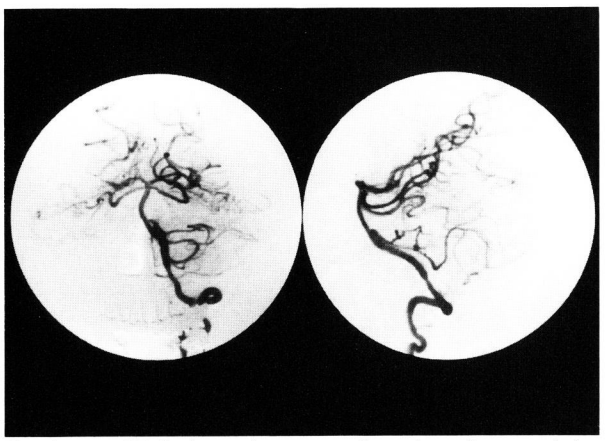

Fig. 4 Left VAG. An angiogram, performed on the second day of admission, revealed irregularities of arterial walls suggesting vasospasms, but no definite obstruction was noted.

sion demonstrated complete disappearance of abnormal intensity areas, better visualizations of sulci, and mild enlargement of lateral ventricles compared with the size on the admission day (**Fig. 5**).

Case 2.

A39-year-old woman started to complain of general weakness and headache. On the following day, She was found lying on the bed with drowsiness and urinary incontinence. She was brought to our hospital with an ambulance car. Her initial blood pressure was 270/150 mmHg, but the patient had no history of hypertension before. Physical examination revealed no abnormalities except for hypertension. She was in a stuporous state but no other definite focal neurological signs were noted. Papilledema or retinopathy was not detected on fundoscopy. Laboratory data were as follows : WBC 12400/mm^3, RBC 423 $\times 10^4/mm^3$, Hb 13.1 g/dl, Ht 40.0%, total protein 8.2 g/dl, GOT 62 K.U. GPT 15K.U., LDH 2880 IU/dl, ALP 11.9 K-AU, LAP 160mIU/ml, GTP 191.8mIU/ml, BUN 86.5mg/dl, creatinine 4.6mg/dl, Glucose 190 mg/dl, Na 137 mEq/l, K 3.9mEq/l, Cl 93 mEq/l, CRP (+), PT 12s, APTT 24.1s, TT 104%, fibrinogen 432 mg/dl, FDP$<$10mg/dl, ANA (-), and RA (-). These blood studies disclosed moderate renal and liver dysfunctions. CSF was clear and colorless with an opening pressure of 140 and a terminal 55 mmH_2O, cell count 4 /mm^3, protein 94mg/dl, and glucose 130mg/dl. Urinalysis disclosed +++ occult blood and +++ protein. EEG on the second day showed mild generalized slowing without laterality or

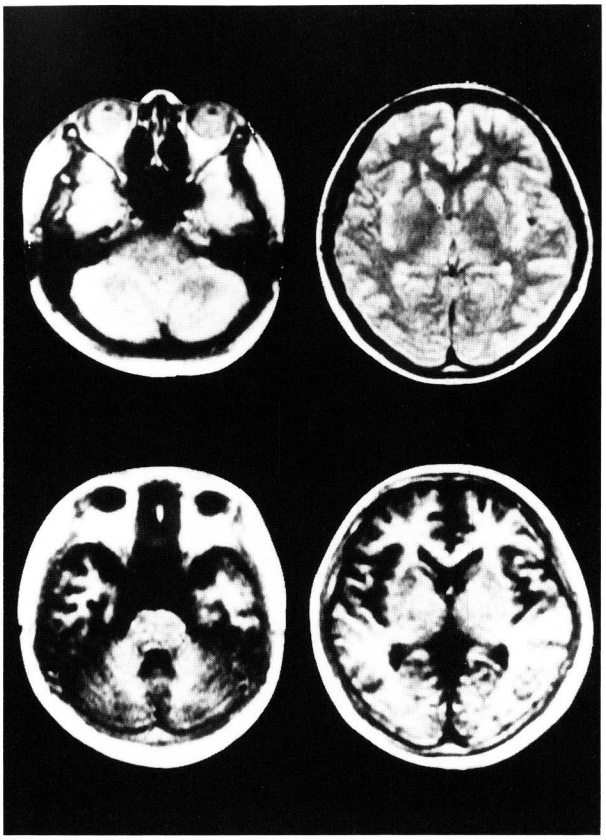

Fig. 5 MRI scans obtained on the 15th day of admission demonstrated complete disappearance of abnormal intensity areas, better visualizations of sulci, and mild enlargement of lateral ventricles compared to the size on the admission day.

triphasic pattern. Cranial CT on admission disclosed marked swelling and low density areas in the brain stem, left cerebellum, bilateral thalamus and basal ganglia without contrast enhancement in the low density areas (**Fig. 6**). MRI performed on the day of admission showed hyperintense T2 and hypointense T1 areas in the right temporal lobe, brain stem, left cerebellum, bilateral thalamus and basal ganglia (**Fig. 7**). Angiogram on admission revealed no definite obstruction or stenosis suggesting cerebral infarction (**Fig. 8**). Every effort with oral and intravenous anti-hypertensive agents failed to control blood pressure. Renal dysfunction and disturbance of consciousness gradually progressed despite intensive treatments. Abnormal lesions in the MRI were interpreted as those of hypertensive encephalopathy considering the features of her clinical symptoms and signs, and neuroimages. However, we also considered that metabolic dysfunctions might intensify the disturbance of consciousness. Therefore, she was transferred to other hos-

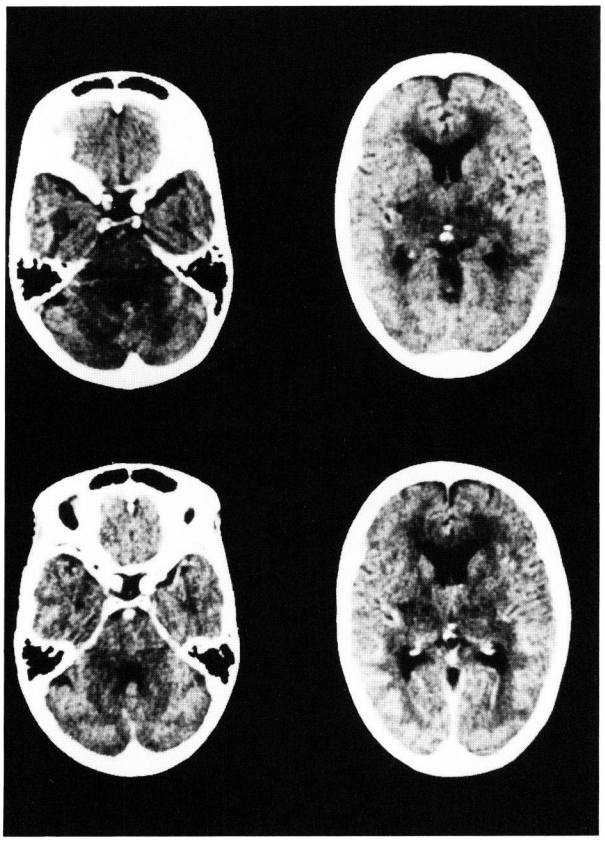

Fig. 6 A CT disclosed marked swelling and low density areas in brain stem, left cerebellum, bilateral thalamus and basal ganglia (upper panel). No contrast enhancement in the low density areas was demonstrated (lower panel).

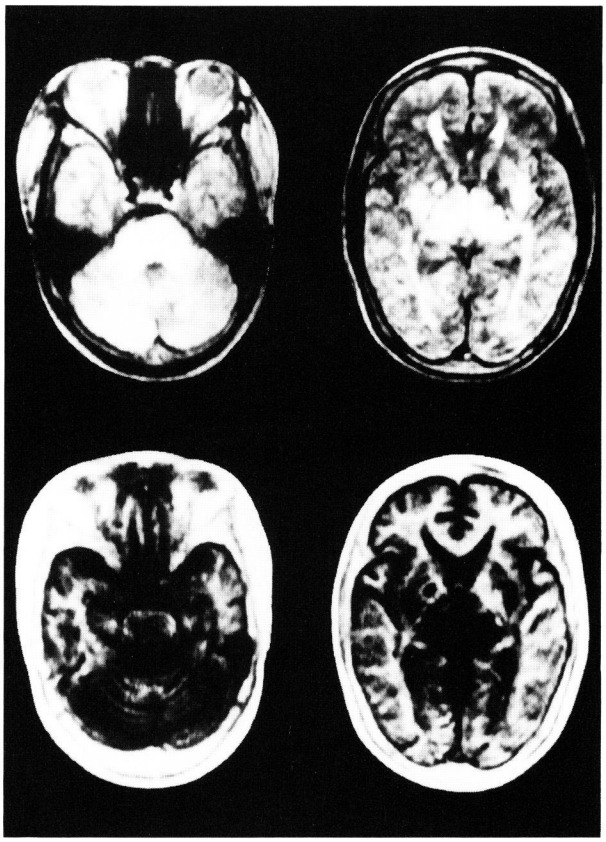

Fig. 7 T2-weighed MRI performed on the day of admission showed high intensity areas in right temporal lobe, brain stem, left cerebellum, bilateral thalamus and basal ganglia. T1-weighted images (IR) demonstrated abnormal areas as low intensity areas. Scanning was performed with the same condition as case 1.

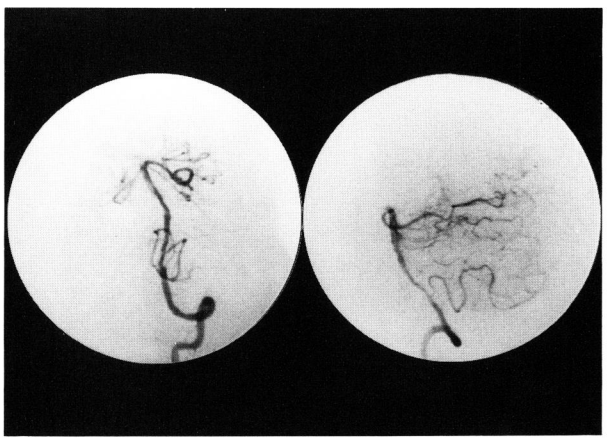

Fig. 8 Left VAG. An angiogram revealed no definite obstruction or stenosis suggesting cerebral infarction.

pital for dialysis on the 4th hospital day. MRI obtained on the 4th admission day demonstrated mild improvement of abnormal intensities and brain swelling (**Fig. 9**).

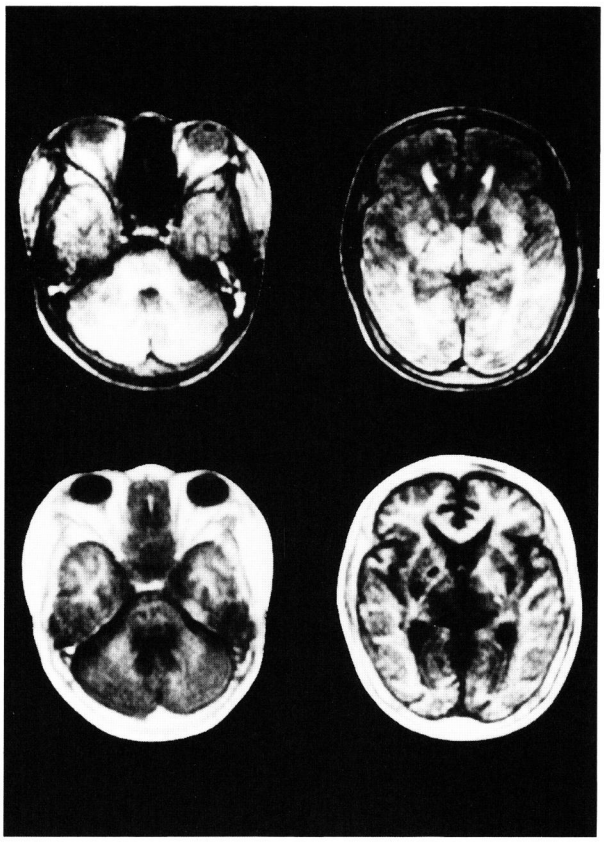

Fig. 9 MRI obtained on the 4 th day of admission demonstrated mild improvement of abnormal intensities and brain swelling.

Discussion

Clinical manifestations of the hypertensive encephalopathy are characterized by headache, nausea and vomiting, papilledema, and retinal hemorrhages and exudates. These may progress to seizures, visual disturbances, disturbance of consciousness, and other focal neurological deficits[1-4]. This syndrome is associated with acute elevatoin of arterial blood pressure but apparently unrelated to cerebral hemorrhage or infarction. During acute phase of the disease, diastolic pressure is usually 130mmHg or greater. The CSF pressure and protein values are generally elevated as in our cases. Complete or partial neurological recovery is expected by reduction of blood pressure, but death may ensue if anti-hypertensive treatments are not started promptly. Therefore, prompt diagnosis and effective antihypertensive therapy are necessary to reverse the condition. CT demonstrates focal low density areas concurrently with diffuse cerebral swelling[9-11]. MRI reveals focal high intensity areas in both subcortical and cortical areas[13-14]. The localization and degree of involvement are often asymmetrical like our cases, although there is a tendency for bilateral involvement. As shown in this report, MRI appears to better define the lesions than CT. Prominent features of MRI findings in our cases are summarized in three points : 1) Major arterial supplies can not explain the anatomical localizations as in the ordinary cerebral infarctions. 2) These lesions are resolved in a relatively short period as two weeks in our first case. 3) There are no neurological focal signs suggesting dysfunctions in the abnormal areas in MRI. These MRI studies support the hypothesis that hypertensive encephalopathy is induced by vasogenic edema during breakthrough of cerebral autoregulation. As in our first case, vasospasm or ischemic events are likely to be secondary phenomenon. Rapidity and degree of increase in blood pressure have been reported to correlate with autoregulation failure leading to vasogenic edema with or without blood brain barrier disturbance. Since the tension in the vessel wall increases with the internal radius and decreases with the wall thickness, dilated vessels would be expected to autoregulate less effectively when exposed to an abrupt increase in blood pressure. Several studies have shown that this is in fact the case[15-19]. Several studies exposing animals to an acute hypertension have confirmed that there is an upper limit of cerebral autoregulation[18-19]. The critical pressure is not reached at the same blood pressure level in all parts of the brain and focal areas of increased flow are observed, usually but not always associated with increased permeability[19]. The reason for this focal pattern is not fully understood.

Hypertensive encephalopathy has been reported in various clinical conditions such as essential hypertension, acute and chronic renal disease, disseminated vasculitis, eclampsia, pheochromocytoma and others[4]. Therfore,

hypertensive encephalopathy may also be accelerated by metabolic impairments in such clinical conditions. Renal dysfunction was, therefore, considered to predispose clinical deterioration in our second case. Prompt recognition and reduction of blood pressure are essential for improving the clinical conditions. MRI appears to detect abnormal lesions of hypertensive encephalopathy effectively and should help proper diagnosis and therapeutic decisions.

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