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T Ito, T Sakai, S Inagawa, M Utsu and T Bun

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MR Angiography of Cerebral Vasospasm in Preeclampsia

Tatsuhiko Ito, Tsuneo Sakai, Shoichi Inagawa, Masaji Utsu, and Takao Bun

Summary: Cerebral vasospasm was demonstrated with MR angiography in a patient with preeclampsia. MR angiography 5 days after the onset of symptoms clearly demonstrated diffuse intracranial vasospasm. The follow-up study confirmed the resolution of vasospasm successfully.

Index terms: Magnetic resonance angiography; Brain, magnetic resonance; Vasospasm

Intracranial vasospasm in preeclampsia/ eclampsia patients has been reported by several authors using conventional cerebral angiography. Although it is crucial to depict cerebral vasospasm in preeclampsia/eclampsia patients, conventional angiography is not easy to carry out in clinical practice. We report a case of preeclampsia in which intracranial vasospasm was clearly demonstrated, and successfully followed up using magnetic resonance (MR) angiography.

Case Report

A 28-year-old woman in the 34th week of gestation who had headache for 2 days became confused and was admitted to a local hospital. Termination of pregnancy was indicated immediately after admission because of her preeclampsia. After delivery of a 1600-g female infant by cesarean section, she was transferred to our hospital because she became increasingly confused. There was no history of hypertension or renal disease. On admission to our hospital, her blood pressure measured 180/100 mm Hg. She had facial edema with minimal extremity edema, and weight gain since prepregnancy was 8 kg. Neurologic examination showed that the patient was disoriented and had mild right hemiparesis and constructional apraxia. Laboratory studies showed hemoconcentration and liver and kidney dysfunction with proteinuria. Computed tomography (CT) on admission showed multiple low densities. No hemorrhagic lesion was seen. Arterial/venous thrombosis and vasospasm were considered possible causes. MR including MR angiography was carried out on

a 1.5-T scanner 3 days after admission, 5 days after the onset of headache. T1- and T2-weighted spin-echo MR images revealed multiple cerebral lesions with prolonged T1 and T2 values involving bilateral occipital lobes, bilateral temporal lobes, left internal and external capsule, left putamen, right thalamus, periventricular white matter including central corona radiata in the left cerebral hemisphere, splenium of the corpus callosum, periaqueductal region, right red nucleus, and pons. (Fig 1). These lesions were considered to represent a mixture of infarction and edema.

MR angiographic evaluation was performed using three-dimensional time-of-flight technique. The MR angiographic study clearly demonstrated diffuse cerebral vasospasm (Fig 2) involving bilateral internal carotid arteries, anterior cerebral arteries, middle cerebral arteries, posterior cerebral arteries, and superior cerebellar arteries. Treatment included hemodilution, crystalloid and colloid infusion, and intravenous nicardipine hydrochloride. Ozagrel sodium was also given to relieve vasospasm. The symptoms improved significantly in 3 days. The patient left the hospital 18 days after admission without any neurologic deficits.

Follow-up MR images obtained 26 days after the initial examination showed several punctate foci suggesting infarction in the brain (Fig 3). Vasospasm had resolved on the MR angiographic images (Fig 4).

Discussion

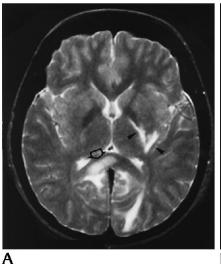
Hypertensive diseases are one of the most frequent complications of pregnancy and are always extremely hazardous for both mother and child (1–3). Hypertensive disorders during pregnancy are classified into pregnancy-induced hypertension, coincidental hypertension, and pregnancy-aggravated hypertension. Pregnancy-induced hypertension is divided into three categories: hypertension alone, preeclampsia, and eclampsia. Preeclampsia is a syndrome manifested by hypertension, edema, proteinuria, and activation of the coagulation

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From the Departments of Neuroimaging Service (T.I.), Neurosurgery (T.S., S.I.), and Obstetrics and Gynecology (M.U.), Seirei Hamamatsu General Hospital, Hamamatsu, Shizuoka, Japan; and the Department of Neurosurgery, Numazu City Hospital, Japan.

Address reprint requests to Dr Tatsuhiko Ito, Neuroimaging Service, Seirei Hamamatsu General Hospital, 2-12-12 Sumiyoshi, Hamamatsu, Shizuoka 430, Japan.

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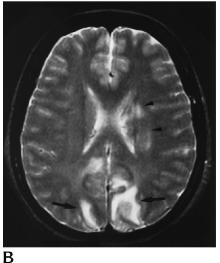


Fig 1. Spin-echo MR images obtained 3 days after admission (5 days after the onset of headache).

A, An oval-shaped hyperintense lesion is shown in the splenium of corpus callosum (blank arrow). Similar lesions are seen in the left internal and external capsule (arrowheads).

B, High-intensity regions in the left corona radiata (*arrowheads*) and bilateral occipital lobes (*arrows*) are demonstrated.

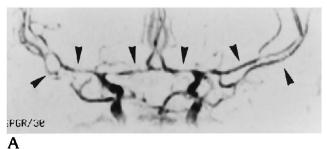


Fig 2. MR angiography performed 3 days after admission. *A*, Anteroposterior maximum intensity projection image. Cerebral vessels including the middle cerebral arteries and the anterior cerebral arteries reveal vasospasm (*arrowheads*).

B, Regional maximum intensity projection image including the superior cerebellar arteries (*small arrowheads*) and posterior cerebral arteries (*large arrowheads*). This image is created like a 30° half-axial view. Diffuse vasospasm of the posterior circulation system is demonstrated.

cascade. Untreated or in its severe forms, preeclampsia can progress to eclampsia, as manifested by convulsion.

Although the precise pathogenesis of pregnancy-induced hypertension is still uncertain, the maternal pathophysiologic reactions in response to immunologic events are considered crucial, and generalized peripheral vasospasm is a common event (3,4). An increased production of vasoconstrictors (eg, thromboxane A_2 [TXA $_2$], angiotensin) and reduced syntheses of vasorelaxing agents (eg, epoprostenol [PGI $_2$], dinoprostone [PGE $_2$]) result from endothelial

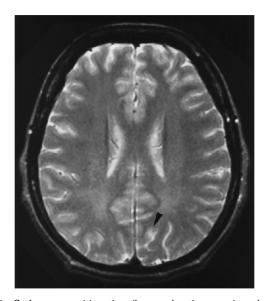


Fig 3. Long-repetition-time/long-echo-time spin-echo MR image dated 26 days after the initial examination. Tiny lesion seeming to be infarction and/or gliotic change is seen in the left occipital lobe (*arrowhead*). Almost all lesions have completely disappeared and are considered edema.

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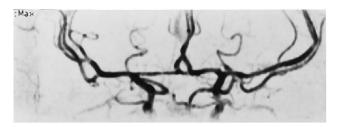


Fig 4. MR angiography obtained 26 days after the initial examination. Same scanning parameters used in the two studies. Vasospasm demonstrated on the prior study have already resolved, and all vessels imaged show normal diameter.

cell disorders in preeclampsia/eclampsia. This increased ratio between vasoconstricting and vasodilating eicosanoids is causative for diffuse vasospasm (5). Recently, endothelin, endothelium-derived peptide, has been considered to participate in the vasospasm of preeclampsia.

Several investigators reported cerebral vasospasm in preeclampsia/eclampsia patients using conventional angiography (6, 7). Vasospasm was seen diffusely including large to small caliber vessels. It was recognized that vasospasm had an important role in ischemic cerebral injury in such patients. In some cases vasospasm was associated with subarachnoid hemorrhage or parenchymal hemorrhage, but in the other cases vasospasm was demonstrated without the evidence of hemorrhage and was attributable to another mechanism. Although conventional angiography can offer the most correct diagnosis, its use for pregnancyinduced hypertension patients in clinical practice is limited.

There are abnormal CT findings such as infarction, hemorrhage, and edema in 29% to 100% of preeclampsia/eclampsia patients (7, 8). Exact ability of MR in delineating the abnormalities of preeclampsia/eclampsia is not completely defined. But it is obvious that up-to-date MR should overcome CT in many aspects, and MR findings may lead the physicians to more intense and urgent care (7, 9). However, abnormal findings including edema, infarction, and hemorrhage seen on standard spin-echo MR merely imply a final appearance of the disease. Also, it is sometimes difficult to clarify the causative pathophysiology (eg, arterial/venous thrombosis, vasospasm, and vasodilation). Some authors pointed out the possibility of mismanagement from misinterpretation of CT findings.

MR angiography is a reliable method evaluating a variety of intracranial vascular diseases. In the present case, a routine 3-D time-of-flight technique clearly demonstrated diffuse vasospasm associated with preeclampsia. Follow-up examination was performed, and it revealed disappearance of vasospasm. Although MR angiography is not sensitive in the evaluation of third- or higher-order arterial branches, it is not crucial to depict such small-caliber vessels in diagnosing cerebral vasospasm. MR angiography also made it clear that dural sinus thrombosis, another possible cause, did not exist in this case.

MR angiography can provide useful information in evaluating intracranial vessels in pregnancy-induced hypertension patients. Relationships between vasospasm and other pathophysiologic changes associated with preeclampsia/eclampsia has not been fully defined. Detection of vasospasm with MR angiography may offer useful information in managing pregnancy-induced hypertension. In conclusion, addition of MR angiography to a routine MR study is useful in evaluating and treating preeclampsia/eclampsia patients.

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References

- Doan-Wiggins L. Hypertensive disorders of pregnancy. Emerg Med Clin North Am 1987;5:495–508
- Barton JR, Sibai BM. Cerebral pathology in eclampsia. Clin Perinatol 1991:18:891–910
- Clark SL, Divon MY, Phelan JP. Preeclampsia/eclampsia: hemodynamic and neurologic correlations. Obstet Gynecol 1985;66:337– 340
- 4. Gilstrap LC III, Gant NF. Pathophysiology of preeclampsia. Semin Perinatol 1990;14:147–151
- Zahradnik HP, Schäfer W, Wetzka B, Breckwoldt M. Hypertensive disorders in pregnancy: the role of eicosanoids. *Eicosanoids* 1991; 4:123–136
- Trommer BL, Homer D, Mikhael MA. Cerebral vasospasm and eclampsia. Stroke 1988;19:326–329
- Royburt M, Seidman DS, Serr DM, Mashiach S. Neurologic involvement in hypertensive disease of pregnancy. Obstet Gynecol Surv 1991;46:656–664
- Brown CEL, Purdy P, Cunningham FG. Head computed tomographic scans in women with eclampsia. Am J Obstet Gynecol 1985;159:915–920
- Digre KB, Varner MW, Osborn AG, Crawford S. Cranial magnetic resonance imaging in severe preeclampsia vs eclampsia. Arch Neurol 1993;50:399–406