

# Postpartum cerebral vasospasm treated with hypervolemic therapy

Paul T. Akins, MD, PhD, Kirk J. Levy, MD, Anne H. Cross, MD, Mark P. Goldberg, MD, and Marc H. Schieber, MD, PhD

St. Louis, Missouri

In a 23-year-old woman, gravida 1, para 1-0-0-1, headaches and seizures developed 1 week after an uncomplicated delivery. Cerebral angiography revealed severe, diffuse cerebral vasospasm. Her symptoms resolved with hyperosmolar, hypervolemic therapy and nimodipine. Magnetic resonance angiography on postpartum day 23 confirmed persistent, severe vasospasm, and repeat magnetic resonance angiography on postpartum day 33 demonstrated interval improvement. This report documents the time course of a case of postpartum vasospasm and its response to hypervolemic, hyperosmolar therapy and nimodipine. (*Am J Obstet Gynecol* 1996;175:1386-8.)

**Key words:** Cerebral vasospasm, angiography (magnetic resonance), eclampsia, puerperium

Late postpartum eclampsia is an uncommon neurologic complication of pregnancy. In a small subset of patients cerebral angiography has demonstrated severe narrowing of cerebral blood vessels consistent with vasospasm.<sup>1-3</sup> We describe the time course of a complicated case diagnosed and monitored by serial neurologic imaging and the response to hyperosmolar, hypervolemic therapy and nimodipine, a widely used treatment for vasospasm caused by subarachnoid hemorrhage.

## Case report

A 23-year-old woman, gravida 1, para 1-0-0-1, had throbbing headaches and two generalized seizures on postpartum day 7 and was admitted to an outside hospital. She had an uncomplicated term pregnancy and spontaneous vaginal delivery without epidural anesthesia, and she did not receive bromocriptine. Results of medical, pelvic, and neurologic examinations were normal. On admission, the blood pressure was 140/92 mm Hg but elevated to 190/120 on postpartum day 10. Findings of routine laboratory studies and cerebrospinal fluid examination were normal except for a uric acid level of 6.5 mg/dl. A magnetic resonance imaging scan of the brain was normal. Electroencephalography performed while the patient was awake demonstrated sharp activity in the left temporal region, and acyclovir was administered for possible encephalitis. On postpartum day 10 she was discharged on a regimen of phenytoin and acyclovir.

On postpartum day 11 the patient became unable to walk and asked the same questions repetitively. She was seen at Barnes Hospital on postpartum day 12. Blood pressure was 122/70 mm Hg. Neurologic examination revealed a normal mental status except for a delayed response to commands, a narrow, shuffling, apractic gait, and brisk deep tendon reflexes that were more pronounced in the legs than arms. Cerebral angiography on postpartum day 12 revealed diffuse, severe narrowing of arteries (Fig. 1, *A* and *B*) with patent cerebral veins and sinuses and no aneurysms. Repeat laboratory analysis was remarkable for a creatinine level of 1.6 mg/dl and clear cerebrospinal fluid with an opening pressure of 12 cm, 7 white blood cells/mm<sup>3</sup> (6 mononuclear leukocytes, 1 polymorphonuclear leukocyte), 161 red blood cells/mm<sup>3</sup>, a glucose value of 47 mg/dl (serum 86 mg/dl), and a protein level of 67 mg/dl. There were no elevations in urine protein, antinuclear antibody, or anticardiolipin antibody levels or in VDRL (Venereal Disease Research Laboratory) test results. Erythrocyte sedimentation rate was 19 mm/hr.

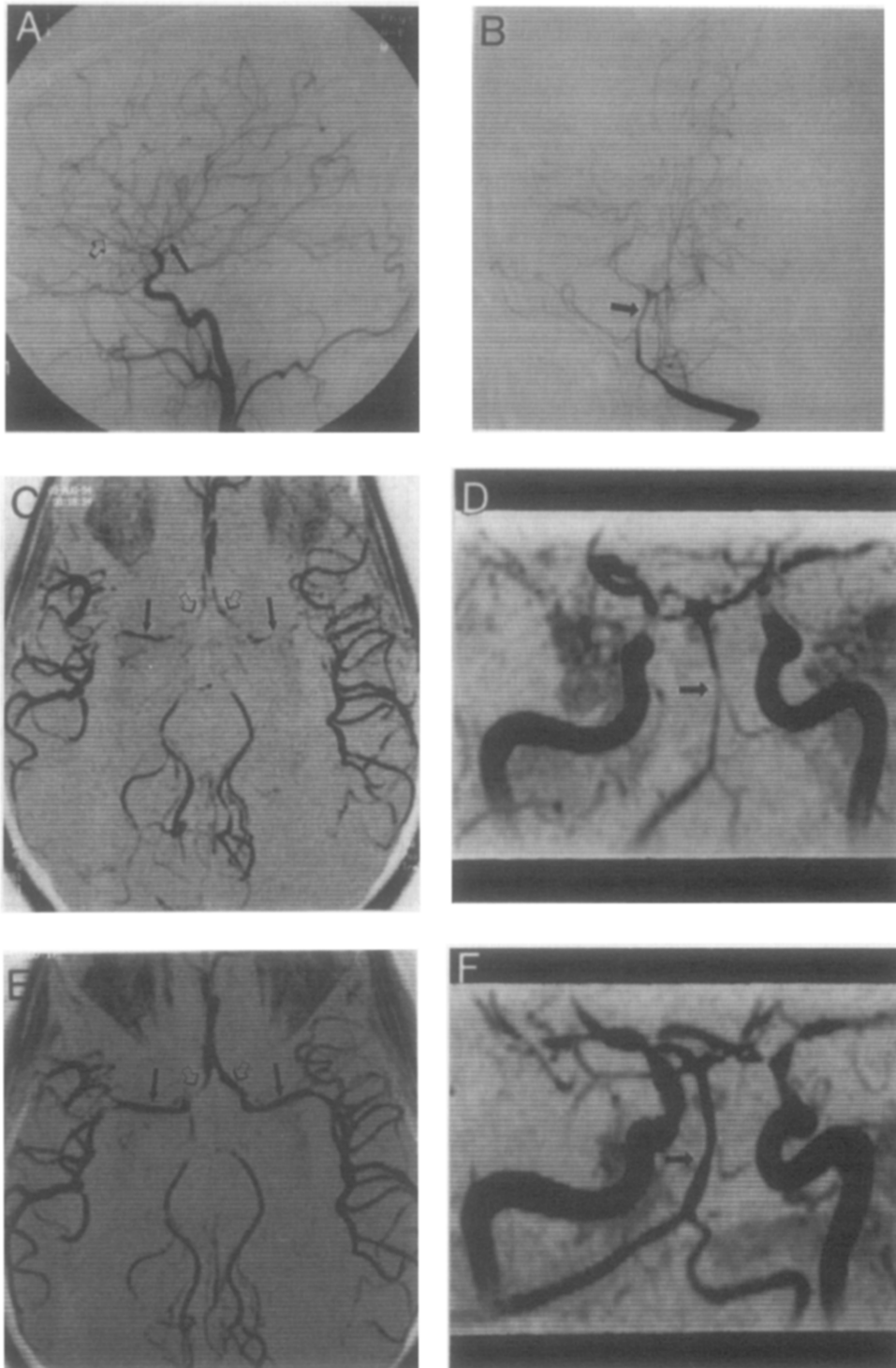
The patient was treated with volume expansion with 5% dextrose in normal saline solution at 400 ml/hr and 12.5 gm of albumin infused every 6 hours. The blood pressure rose to 160/100 mm Hg, and the symptoms resolved. She was weaned from therapy over postpartum days 14 to 17. The blood pressure declined to 126/92 mm Hg. A headache developed on postpartum day 15, and she fell as a result of the recurrence of the gait disorder on postpartum day 18. Hypervolemic therapy was reinstated, and nimodipine (60 mg orally every 4 hours) was added. Blood pressure increased to 180/100 mm Hg, the headache resolved, and gait returned to normal. Despite a normal appearance of the magnetic resonance imaging scan, three-dimensional time-of-flight magnetic resonance angiography (Siemens, Germany; 1.5 T) on postpartum day 23 demonstrated persistent, severe arterial narrowing (Fig. 1, *C* and *D*), and repeat magnetic resonance angiography on postpartum day 33 (Fig. 1, *E* and *F*) indicated partial reso-

*From the Department of Neurology, Washington University School of Medicine.*

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*Reprint requests: Paul T. Akins, MD, PhD, Department of Neurology, Washington University School of Medicine, Box 8111, 660 South Euclid Ave., St. Louis, MO 63110.*

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**Fig. 1.** Postpartum vasospasm was monitored with serial neuroimaging. **A** and **B**, Cerebral angiography on postpartum day 12 revealed severe arterial narrowing after right carotid (**A**, lateral view) and left vertebral artery (**B**, anteroposterior view) injections. **C** and **D**, Magnetic resonance angiography on postpartum day 23 showed persistent arterial narrowing. **E** and **F**, Repeat study on postpartum day 33 revealed interval improvement. Collapsed axial images of circle of Willis and proximal cerebral vessels were viewed in a dorsal-ventral orientation (**C** and **E**). Posterior circulation was imaged in an anteroposterior (**D** and **F**) projection. Narrowing was present in anterior (*open arrows*) and middle (*solid arrows*) cerebral arteries (**A**, **C**, and **E**) and in basilar artery (*solid arrows*, **B**, **D**, and **F**).

lution. She was weaned off hypervolemic therapy over postpartum days 33 to 40. Nimodipine was administered for a total of 21 days, and phenytoin was discontinued 1 month after discharge. In follow-up at 6 months she had returned to work, results of examination were normal except for a persistently brisk right patellar reflex, and blood pressure was 116/84 mm Hg.

### Comment

Cerebral vasospasm as a complication of pregnancy is rare. It was first recognized in the European literature.<sup>2</sup> Other terms have been applied to it, such as postpartum angiopathy (vasculopathy or arteriopathy) or isolated angiitis. The pathogenesis is poorly understood. Although some cases have been viewed as isolated central nervous system vasculitis and treated with immunosuppressant agents, pathologic documentation was not available. In the single reported autopsy study<sup>3</sup> the cerebral blood vessels had slight intimal thickening without perivascular inflammation. We favor vasospasm as the explanation for the angiographic findings. The pathologic characteristics of eclampsia suggest that vasospasm may affect other organs such as the liver.

We identified 20 patients described in the English literature with angiographically documented vasospasm in the peripartum period. Most patients had a severe prodromal headache. Some patients had fluctuating focal deficits. Visual field deficits, dysphasia, dyscalculia, and coma were also reported. The condition is not benign. Five patients had strokes, and one died.<sup>3</sup> Patients with pregnancy-associated vasospasm were first seen from the day of delivery to 3 weeks post partum. The duration of this condition is variable. Clinically, symptoms resolved in many patients within a week, whereas others had prolonged courses lasting several weeks and complicated by strokes.

We treated our patient with hypervolemic therapy and nimodipine because of the success of this treatment for cerebral vasospasm caused by subarachnoid hemorrhage. The patient had a well-defined clinical response to the initiation of treatment. A relapse occurred when hypervolemic therapy was tapered off, and improvement occurred when it was reinstated. Hypertensive agents such as phenylephrine were not used and, in theory, could

exacerbate the condition. Other reported treatments for postpartum cerebral vasospasm have included intravenous magnesium sulfate, antihypertensive agents, nimodipine, and steroids. There was no clear correlation between treatment modality and stroke, although the only patient who received verapamil (240 mg/day) died.<sup>3</sup>

In contrast to our patient's normal magnetic resonance imaging scan, it is more common in eclampsia to find increased T2-weighted signal intensity at the junction of cortex and white matter, particularly in the parietooccipital regions. The preferential involvement of the occipital lobes may be the source of the visual symptoms reported by patients with eclampsia. Only a minority of patients with these findings will have vasospasm demonstrated by angiography. If the edema represents disruption of the blood-brain barrier, hyperosmolar, hypervolemic therapy may be detrimental in eclamptic patients with increased T2-weighted signal intensity.

This case report provides some useful observations. Cerebral angiography was necessary to diagnose postpartum vasospasm and exclude aneurysms and sagittal sinus thrombosis. Magnetic resonance angiography provided a noninvasive method to follow its course. Hypertension associated with postpartum cerebral vasospasm may be beneficial to maintain perfusion to ischemic brain, and antihypertensive agents in such patients with known vasospasm should be avoided. In patients with symptoms of postpartum cerebral vasospasm, hypervolemic therapy and nimodipine may alleviate cerebral ischemia while spontaneous resolution of this condition is awaited.

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