

Emergency Carotid Artery Thrombectomy for Postpartum Hemiplegia

JAMES T. STURM, MD, CAMILO TORO, MD,
BRUCE D. SNYDER, MD

The case of a 22-year-old patient who suffered from postpartum hemiplegia due to thrombosis of the internal carotid artery is reported. She was treated aggressively with pentobarbital coma, angiography, and thrombectomy and recovered with minimal deficit. (*Am J Emerg Med* 1987;5:291-293)

We recently evaluated and treated a young psychiatric patient whose initial presentation suggested either hysteria or reaction to phenothiazine-like drugs. The case is reported to review the clinical presentation and management of peripartum cerebral infarction.

REPORT OF A CASE

A 22-year-old right-handed white woman, gravida 1, para 0, was admitted to St. Paul-Ramsey Medical Center on July 4, 1985, with a blood pressure of 160/110 mm Hg, vomiting, diarrhea, and complaints of left-sided headache and visual blurring. The patient had a long history of major schizo-affective disorder treated with antipsychotic and antidepressant medications and was a documented emergency department abuser. She was admitted to the obstetrical service where 2 to 3+ proteinuria was noted and magnesium sulfate therapy was begun. Despite sedation, blood pressure remained high and a diagnosis of pre-eclampsia was made. After an initially stable period, her blood pressures rose as high as 174/136 mm Hg on July 7, and intravenous hydralazine was used to control it. Because of the inability to control her severe pre-eclampsia, an emergency cesarean section was performed and a low-birth-weight female infant was delivered. The patient developed bacteremia secondary to endometritis, which was treated with ampicillin and gentamicin. She defervesced, and her blood pressure gradually responded to oral alpha methyl dopa.

From the Departments of Emergency Medicine and Neurology, St. Paul-Ramsey Medical Center, St. Paul, Minnesota.

Manuscript received June 20, 1986; revision accepted November 25, 1986.

This study was supported in part by the St. Paul-Ramsey Foundation and the Ramsey Clinic.

Address reprint requests to Dr. Sturm: Department of Emergency Medicine, St. Paul-Ramsey Medical Center, 640 Jackson Street, St. Paul, MN 55101.

Key Words: Carotid artery, hemiplegia, hypercoagulability, stroke, thrombectomy.

0735-6757/87 \$0.00 + .25

The patient remained uncooperative and irritable throughout her course, and on July 14 again reported headache over her left orbit. She had been mentioning multiple somatic symptoms. On July 16, she reported clumsiness of her right hand, depression, agitation, numbness, and weakness. It was believed these symptoms had a functional basis. Perphenazine was begun, outpatient psychiatric follow-up was arranged, and the patient was discharged.

She awoke that night at 2:00 AM with difficulty speaking and flaccid paralysis of her right upper extremity. Evaluation in the emergency department revealed a labile, frustrated but conscious woman. Hysteria was suspected. The blood pressure was 136/70 mm Hg, the pulse 76 bpm, and respirations 20 breaths/min. The general physical examination was normal. Neurologic examination showed an alert, oriented, frightened, and uncooperative patient with a nonfluent aphasia, severe anomia but reasonably good comprehension. A right central facial paresis and right hemiparesis, maximal in the upper extremity, was present with a right Babinski response and right hyporeflexia. A dystonic reaction to phenothiazine-like drugs was ruled out by administration of 2 mg benztrapine intravenously. There was no evidence of peripheral embolization. There were no vascular bruits and the cardiac examination was normal. An emergency computed tomography scan showed subtle effacement of parietal cortical sulci and indistinctness of the gray-white interface, suggesting early infarction. The patient was given heparin and admitted to the neurology intensive care unit.

Admission laboratory data revealed a hemoglobin of 10.5 grams, and a total white count of 15,600 mm³ with a shift to the left. Coagulation parameters included a prothrombin time of 13 seconds, an activated partial thromboplastin time of 25.4 seconds, and a thrombin time of 16.7 seconds. Serum was negative for fibrin split products, but the fibrinogen level was 420 mg/dl (normal, 200 to 400 mg/dl). Factor V level was 216% (normal, 70 to 150%). The admission platelet count was 435,000 mm³.

A pentobarbital coma was instituted with a loading dose of 20 mg/kg intravenously and a maintenance infusion of 1 mg/kg/hr. The patient was placed on a ventilator and angiography performed, which revealed near-total occlusion of the left internal carotid artery with a tail of thrombus extending distally in the vessel (Fig. 1). A branch of the left middle cerebral artery was missing. Approximately 17 hours following onset of symptoms, the patient underwent a left carotid thrombectomy. A jelly-like thrombus, loosely adherent to an otherwise normal vessel wall, was removed.

Postoperatively, pentobarbital was discontinued, and the patient gradually improved over the next 36 hours. Heparin was continued. Postoperative carotid ultrasound studies revealed no thrombosis. Cardiologic evaluation and echocardiography were entirely normal. Serologic screening for collagen vascular disease was negative.

During the next few days, the patient showed progressive improvement in the use of her right arm, regaining almost full proximal strength. Her speech similarly improved such that it was virtually normal by discharge, nine days after admission. At one month follow-up, her aphasia had cleared completely but she had residual clumsiness of her right hand. She has been maintained on low-dose aspirin and was doing well at six months' follow-up.

DISCUSSION

Multiple studies attest to the increased risk of cerebral infarction during and immediately after pregnancy.¹ Unlike stroke in the older individual, underlying atherosclerosis is unusual. The mechanism appears to be spontaneous thrombus formation within the heart, extracranial carotid or vertebral arteries, the trifurcation of the middle cerebral artery, or intracranial venous structures. Arterial occlusions tend to occur during the second and third trimesters of pregnancy and during the first week after delivery. Venous thrombi usually are seen one to four weeks after childbirth. Other rarer causes of focal central nervous system syndromes must be considered and recently have been reviewed.¹

The experience we have reported is anecdotal. There are no published series of young patients, without atherosclerosis, who have undergone surgical treatment of acute thrombotic occlusion of the carotid artery. Previous reports of acute carotid thrombectomy have described older patients with atherosclerosis as the precipitating cause of acute thrombosis. The successful outcome of this patient may have been serendipitous.

The initial diagnostic evaluation of this patient began with a noninvasive CT scan that showed left parietal ischemia. The next test chosen was arteriography of the extracranial and intracranial circulation. Arteriography is invasive, but it is the only method of completely evaluating the entire circulation to the cerebrum. The noninvasive Doppler examination of the carotid circulation does not provide enough resolution to evaluate the patient for operation on the carotid artery, and has no ability to image the intracranial circulation. The noninvasive carotid ultrasound has been used in follow-up to ascertain that the thrombosis has not recurred.

The relatively higher risk of stroke in the pregnant woman warrants a higher index of suspicion on the part of clinicians evaluating a pregnant patient with neurologic symptoms. Our higher index of suspicion in this patient was diluted and offset by two prejudices. First, this psychiatric patient was an abuser (two to three times weekly) of the emergency department who was known by the treating physicians to have exaggerated symptoms many times in the past. Secondly, the patient had been treated previously for dystonic reactions to phenothiazine-like drugs. The initial differential diagnosis included hysterical paralysis and dystonic reaction to phenothiazine-like drugs. A detailed neurologic examination appeared to rule out hysteria, and a trial dose of 2



FIGURE 1. The angiogram shows a radiolucency representing thrombus that begins at the bifurcation of the external and internal carotid arteries (arrow) and tails distally into the internal carotid.

mg bntropine was given intravenously. Only when the bntropine failed was a genuine neurologic deficit considered.

This case is most instructive because it is a reminder that even hypochondriacs and emergency department abusers can present with catastrophic illnesses. The lesson learned here is that when a hysteric, hypochondriac, or emergency department abuser presents for medical attention, a common course of action is to assume that it is merely another instance of abuse. The appropriate protocol with such patients

should be to systematically disprove the possibility of serious illness and to document the disproof in the record.

The value of barbiturate coma is not proven by this case. We will discuss how barbiturate coma might have helped this patient. Animal studies have documented improved outcome in models of focal cerebral ischemia using barbiturate coma therapy.²⁻⁵ Reduced size of infarction and reduced clinical deficits in animal models have been documented. Progress in this area has been slow because of the extreme difficulty in producing a standard set of clinical conditions under which to test this approach in human beings. The clinical trials by Slogoff and Nussmeier^{6,7} seem to have identified a group in which this could be tested (patients undergoing open heart surgery or cardiopulmonary bypass) and have found convincing evidence, in a well-designed trial, that barbiturate anesthetics seem to prevent effectively the development of fixed focal deficits.

Prior to the Nussmeier study, small series and anecdotal cases supporting the use of barbiturate therapy for anticipated or actual focal cerebral ischemia had appeared in the literature.^{8,11} Some anecdotal negative experiences also have been reported.^{12,13} Based on current information, the effective use of barbiturate coma in circumstances of anticipated or actual focal cerebral ischemia should attempt to follow these guidelines: 1) Barbiturate coma should be induced either prior to the episode of ischemia or preferably within one hour of its onset. Note that this actually may be within an hour of progression of a preexisting minor deficit and, therefore, the clinical monitoring of stroke patients for progression must be quite close. 2) Barbiturate anesthesia is contraindicated in patients with massive, fixed deficits or indications of extensive hemispheric infarction on CT scan. 3) Barbiturate coma may be contraindicated in patients who have a fixed obstruction of a major intracranial vessel (e.g., middle cerebral artery) unless plans for revascularization (bypass or embolectomy) are made.

CONCLUSION

Cerebral ischemia is a rare but potentially fatal complication of pregnancy. Symptoms suggesting cerebral ischemia during pregnancy and the peripartum period should be investigated thoroughly.

REFERENCES

1. Donaldson JO: Neurology of Pregnancy. Philadelphia, WB Saunders, 1978, pp 115-156
2. Smith AL, Hoff FT, Nielson SL, et al: Barbiturate protection in acute focal cerebral ischemia. *Stroke* 1974;5:127
3. Michenfelder JD, Milde JH: Influence of anesthetics on metabolic, functional, and pathological responses to regional cerebral ischemia. *Stroke* 1975;6:405-410
4. Mosely JI, Laurent JP, Molinari GF: Barbiturate attenuation of the clinical course and pathologic lesions in a primate stroke model. *Neurology (Minneapolis)* 1982;25:870-874
5. Michenfelder JD: Barbiturates for brain resuscitation: Yes and no. *Anesthesiology* 1982;57:74-75
6. Slogoff S, Girgis KZ, Keats AS: Etiologic factors in neuropsychiatric complications associated with cardiopulmonary bypass. *Anesth Analg* 1982;61:903-911
7. Nussmeier NA, Ralund C, Slogoff S: Neuropsychiatric complications after cardiopulmonary bypass: Cerebral protection by a barbiturate. *Anesthesiology* 1986;64:165-170
8. McMeniman WJ, Fletcher JP, Little JM: Experience with barbiturate therapy for cerebral protection during carotid endarterectomy. *Ann R Coll Surg Engl* 1984;66:361-364
9. Gross CI, Adams HP, Sokoll MD, et al: Use of anticoagulants, electroencephalographic monitoring, and barbiturate cerebral protection in carotid endarterectomy. *Neurosurgery* 1981;9:1-5
10. Astrup J: Barbiturate protection in focal cerebral ischemia. *Scand J Clin Lab Invest* 1980;40:201-203
11. Steen PA: Barbiturate protection against ischemic brain damage. *Scand J Clin Lab Invest* 1980;40:205-207
12. Lawner PM, Sineone FA: Treatment of intraoperative middle cerebral artery occlusion with pentobarbital and extracranial/intracranial bypass. *J Neurosurg* 1979;51:710-712
13. Rockoff MA, Marshall LF, Shapiro HM: High dose barbiturate therapy in humans; clinical review of 60 patients. *Ann Neurol* 1979;6:494-499