

Reversal of a Dense, Persistent, Holohemispheric Neurological Deficit after an Endarterectomy of the Carotid Artery: Case Report

Rickey L. McKenzie, M.D., Issam A. Awad, M.D., M.S., and Cathy A. Sila, M.D.

Departments of Neurological Surgery (RLM, IAA) and Neurology (CAS), The Cleveland Clinic Foundation, Cleveland, Ohio

The role and timing of a carotid endarterectomy in the setting of an acute ischemic stroke-in-evolution remain controversial. Although computed tomographic (CT) scans typically show no abnormalities in the acute stage, it is generally agreed that a dense neurological deficit (hemiplegia) and/or multiple modality neurological disturbance (involving motor, sensory, gaze, and visual field impairment) represent contraindications to surgical intervention. We present a case of an acute right holohemispheric neurological deficit including dense hemiplegia, hemisensory loss, gaze disturbance, hemineglect, and impaired level of consciousness. This persisted for 4 days while serial CT scans showed no evidence of infarction. Angiography revealed pre-occlusive stenosis of the right internal carotid artery with sluggish antegrade flow. The anterior collaterals of the circle of Willis were impaired, and the right middle cerebral artery territory filled via the posterior communicating artery. Despite the dense neurological deficit persisting for 4 days, a carotid endarterectomy was performed. Gradual neurological improvement was noted within hours of the operation, and all neurological deficits resolved within the subsequent 3 days. This case is consistent with prolonged holohemispheric hemodynamic compromise below the threshold of neurological dysfunction, but above the threshold of tissue infarction ("idling neurons"). Features assisting in the recognition of this unusual scenario and the indications and risks of revascularization in this setting are discussed. (*Neurosurgery* 29:261-264, 1991)

Key words: Carotid endarterectomy, Hemodynamic dysfunction, "Idling neurons," Stroke-in-evolution

INTRODUCTION

The role of the carotid endarterectomy in the setting of acute stroke-in-evolution remains controversial (3, 5-7, 10, 11). The early literature suggests that surgery increases the risk of a reperfusion injury without significant benefit in the prognosis or long-term outcome (16, 17). More recently, it has become apparent that surgery is well tolerated in the absence of profound neurological deficit or extensive infarction on computed tomographic (CT) scans (3, 9-11). It is thought generally that a dense neurological deficit (hemiplegia) and/or multiple modality neurological impairment (including motor, sensory, gaze, and visual field disturbance) represent contraindications to surgery (4, 6, 9, 11). The first few hours of a stroke-in-evolution pose a special therapeutic dilemma in that the CT scan does not reveal the extent of ischemic damage this soon after the insult.

We present a case of a dense right holohemispheric neurological deficit that persisted for 4 days but was unaccompanied by any evidence of infarction on serial CT scans. The reversal of the neurological deficit occurred promptly after a carotid endarterectomy.

CASE REPORT

A 70-year-old, right-handed, former professional boxer presented to the Cleveland Clinic with acute weakness of the left side. One day before the onset of the weakness, he had noticed left hemisensory changes described as "numbness." The past medical history was significant for a transient ischemic attack of the right hemisphere involving the left arm 8 years previously. In addition, the family had noted a subtle gradual decline in mental function in the past several years, attributed by them to the history of professional boxing. The patient had smoked one pack of cigarettes per day for 30 years and had

mild essential hypertension treated with diuretics. There was no history of symptomatic heart disease, peripheral vascular disease, or diabetes.

The initial neurological examination revealed a drowsy patient who was oriented to person, place, and time. Speech was slow and dysarthric, but the patient was able to answer simple questions, spell short words, and name objects and familiar persons. Pupils were symmetric and reactive, but there was a conjugate right gaze deviation, with

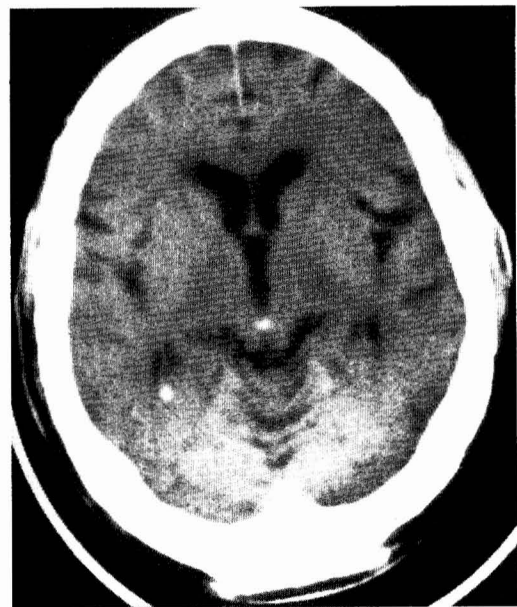


FIG. 1. CT scan of the brain without contrast enhancement performed 3 days after the onset of a dense holohemispheric neurological deficit. There is no evidence of a parenchymal infarction.

inability to look to the left. Strength and tone were normal in the right upper and lower limbs. There was no voluntary movement of the left upper or lower limbs. The left arm extended to painful stimulation, whereas the left leg flexed. Tonus was increased in the left upper and lower limbs. There was decreased light touch, pinprick, and joint position sense in a left hemisensory distribution. The patient appeared to ignore the left side. Deep tendon reflexes were increased, and the Babinski sign was present on the left.

Diagnostic evaluation and initial management

On the day of admission, a carotid Doppler ultrasound examination was performed and revealed severe stenosis of the right internal carotid artery (ICA) at its origin. The CT scan was negative for a hemorrhage or infarction. Because of the dense multimodality neurological deficits implying holohemispheric cortical and subcortical neurological dys-

function, the initial working diagnosis was that of a large hemispheric infarction. The patient was taken to the Neurosurgical Intensive Care Unit where Swan-Ganz catheterization was performed, and intensive hemodynamic support was initiated, aiming for systolic blood pressure above 180 mm Hg and pulmonary capillary wedge pressure in the range of 14 to 18 mm Hg. Pressors administered intravenously and intermittent colloid boluses were used to achieve these objectives. The peripheral hematocrit concentration was maintained in the range of 33 to 38%. Because of the pre-occlusive carotid stenosis, it was elected to heparinize the patient, aiming for partial thromboplastin times in the range of 50 to 60 seconds, with the tentative plan to discontinue heparinization at the first clinical or imaging evidence of a hemorrhagic infarction.

Medical therapy was continued under the presumption that a large infarction of the middle cerebral artery (MCA) would soon appear on

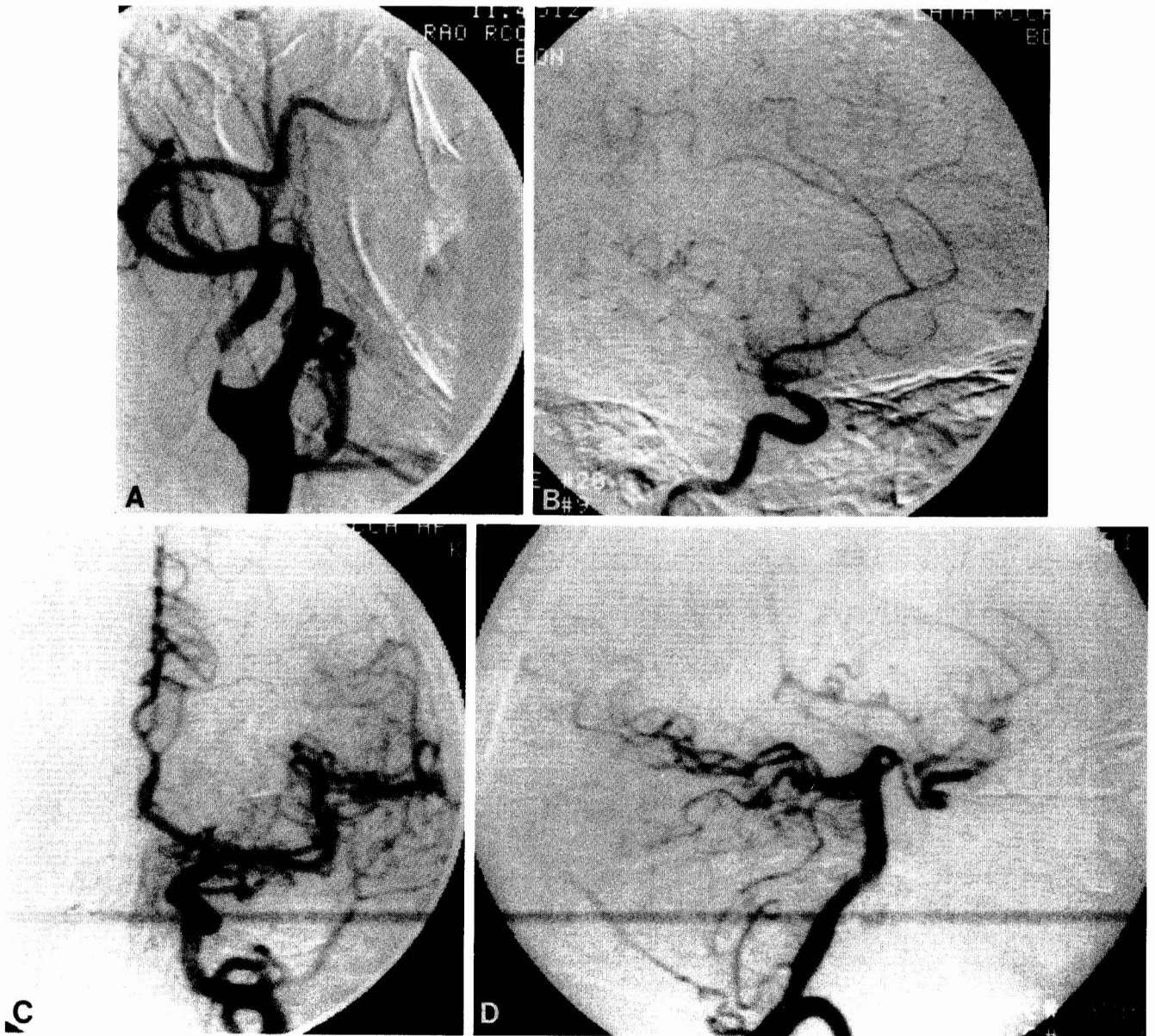


FIG. 2. Intra-arterial digital subtraction angiograms performed 3 days after the onset of the neurological deficit. *A*, cervical view of the right ICA reveals pre-occlusive stenosis near the origin of this vessel, with sluggish antegrade flow. *B*, lateral intracranial view of the same injection revealing impaired filling of the right MCA territory. *C*, anterior view of the left (contralateral) internal carotid injection shows incompetent anterior collaterals of the circle of Willis. *D*, lateral view of the vertebrobasilar injection reveals antegrade filling of the right MCA via the posterior communicating artery.

the CT scans; however, daily serial CT scans performed over the subsequent 3 days showed no infarction (Fig. 1). Magnetic resonance (MR) imaging was attempted, but the image quality was suboptimal because of poor patient cooperation and an unstable hemodynamic situation. The MR image revealed no obvious extensive infarction, however, and no evidence of extensive periventricular white matter changes.

Cerebral digital subtraction arteriography was performed on the third day after the onset of symptoms via retrograde femoral artery catheterization. This revealed pre-occlusive stenosis of the right ICA just distal to its origin (Fig. 2A). There was very sluggish antegrade flow and delayed faint filling of the right MCA territory on this injection (Fig. 2B). The anterior collaterals of the circle of Willis were incompetent (Fig. 2C). The vertebrobasilar injection filled the right MCA territory via the posterior communicating artery (Fig. 2D).

Meanwhile, the patient's neurological status remained unchanged, but the level of consciousness appeared to deteriorate significantly whenever systolic arterial blood pressure drifted below 160 mm Hg. Because of the lack of improvement despite aggressive medical therapy, the tendency toward neurological worsening with normalization of blood pressure, the absence of parenchymal infarction on CT scans after 4 days, and the patency of the ICA and MCA, it was decided to offer the patient the option of surgical intervention. Because of the dismal neurological condition, the family was warned that the prognosis was guarded.

Operation

Under general anesthesia administered endotracheally and continuous electroencephalographic monitoring, a microsurgical carotid endarterectomy was performed. Systolic arterial blood pressure was maintained above 160 mm Hg during the neck dissection and carotid cross-clamping. Systolic arterial blood pressure was controlled closely below 120 mm Hg after the restoration of carotid flow. Additional measures of cerebral protection included phenytoin (1000-mg bolus) and mannitol (1 g/kg-bolus) administered intravenously before the carotid cross-clamping. The patient remained fully heparinized during the neck dissection (partial thromboplastin time, 60 s), and an additional 2000 IU of heparin were administered just before the carotid clamping. Heparin was discontinued but not reversed on the restoration of carotid flow.

The baseline electroencephalogram showed moderate slowing diffusely over the right cerebral hemisphere. This did not change during the 24 minutes of clamping. No luminal shunt was used.

Postoperative course

Postoperative intensive care management was aimed at prevention of reperfusion complications. Systolic arterial blood pressure was controlled closely in the range of 100 to 120 mm Hg. Phenytoin (300 mg/d) and mannitol (1 g/kg/d) were administered intravenously during the first 3 postoperative days, with a gradual tapering of both agents over the subsequent 4 days. Aspirin therapy (325 mg/d) was instituted on the discontinuation of heparin.

Within 3 hours of awakening from anesthesia, the patient regained voluntary antigravity strength in the left lower limb. There was total resolution of all focal neurological deficits over the subsequent 48 hours. Postoperative CT scans of the brain showed no abnormalities. Postoperative MR images were unremarkable except for subtle signal alterations in the subcortical parenchyma in the right suprasylvian region (Fig. 3). Postoperative digital subtraction angiograms using intravenously administered contrast medium confirmed the patency of the operated vessel.

The patient was discharged from the intensive care unit 3 days after the operation and from the hospital 6 days later. At the time of the last follow-up 3 months after the operation, the patient had returned to his baseline functional status, exhibited no focal neurological abnormalities, and had experienced no recurrent ischemic spells.

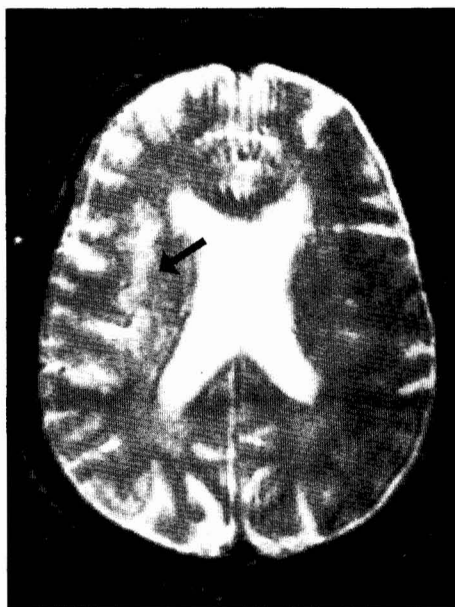


FIG. 3. T2-weighted MR image without gadolinium enhancement performed 3 days after the operation reveals increased subcortical signal intensity in the right suprasylvian region (arrow). This may represent subtle, residual, parenchymal ischemic damage or an incidental finding commonly observed in the elderly. The CT scan showed nothing abnormal.

DISCUSSION

This case raises several issues regarding surgical intervention in the setting of an acute ischemic stroke-in-evolution. There is general agreement that patients with an extensive thromboembolic infarction do not benefit, and may possibly be harmed, by the acute revascularization of the infarcted territory. The presence of a profound neurological deficit and multimodality neurological impairment are thought to represent evidence of such an extensive infarction and, therefore, a contraindication to surgical intervention (4). The present case illustrates a rare exception to this general rule.

Despite profound hemiplegia, hemisensory loss, gaze disturbance, and a fluctuating level of consciousness for 4 days, the patient did not exhibit a detectable infarction on neuroimaging studies. Angiography demonstrated patency of the ICA (pre-occlusive stenosis) with sluggish antegrade flow. Although the MCA territory did not fill promptly on carotid injection, there did not appear to be an occlusion of the MCA. The posterior circulation injection was helpful by confirming the patency of the MCA and prompt filling of its territory via the posterior communicating artery. There was no evidence of artery-to-artery embolism or thromboembolic occlusion of the MCA or its branches.

Although MR imaging can delineate the extent of parenchymal ischemic damage earlier than CT scans, it is frequently difficult to perform in an emergency situation especially in the setting of neurological and hemodynamic instability. In this case, MR imaging was performed 48 hours after the onset of symptoms; despite suboptimal image quality, it did rule out an extensive hemispheric infarction. The consistent absence of any evidence of parenchymal infarction over the period of 4 days prompted us to consider surgical options despite the dismal neurological status.

We thought this patient was at risk for reperfusion-breakthrough complications, including cerebral edema, hemorrhage, and/or seizures in the postoperative period. Judicious

intensive care management aimed at the prevention of these complications consisted of the prophylactic treatment of arterial hypertension to levels below the patient's preoperative normal range and mannitol and phenytoin administered intravenously.

The presentation, diagnostic findings, clinical course, and outcome of this case are consistent with hemodynamic compromise below the cerebral blood flow threshold of neurological dysfunction but above that of tissue infarction (14, 15). This is in support of the pathophysiological concept of "idling neurons," which has been demonstrated rarely in humans (1, 8, 12, 13). Further proof of this pathophysiological scenario would have been provided by positron emission tomography (2). Although this modality is available at our institution, logistic reasons and the patient's neurological and systemic status prevented its application in this particular case.

This report is not meant to endorse the concept of an emergency carotid endarterectomy in all cases of acute stroke and a devastating neurological deficit. It demonstrates, however, that dense multimodality neurological deficits do not always imply a massive cerebral infarction and do not consistently preclude a favorable outcome from a carotid endarterectomy.

Received for publication, September 19, 1990; accepted, final form, February 13, 1991.

Reprint requests: Issam A. Awad, M.D., M.S., Department of Neurological Surgery, The Cleveland Clinic Foundation—S80, 9500 Euclid Avenue, Cleveland, OH 44195.

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COMMENTS

The authors present a most interesting case that seems to be a good example of a holohemispheric ischemic penumbra. Undoubtedly, some would question the acute institution of hypertensive, hypervolemic, and anticoagulant therapy in a patient who has such a deficit, but the authors' rational and "cautiously aggressive" approach proved correct with an excellent outcome. Reliable cerebral blood flow measurements such as might be obtained with single photon emission tomography or positron emission tomography would have been of great interest in this case. Also, it is interesting to speculate as to what the approach might have been if angiography had demonstrated an occluded internal carotid artery, inasmuch as it might be asserted that, in this event, a bypass procedure would have been indicated.

David C. Piepgras
Rochester, Minnesota

McKenzie and co-workers describe a condition that must occur once or twice every decade or so among the population of the world. The transition between ischemia and death in the large cells of the cerebral cortex must be fairly abrupt. Perhaps, this is why we do not see these cases more often.

For those of us who would operate on patients with acute stroke secondary to carotid artery disease, a word of caution, however, may be appropriate. A carotid endarterectomy in patients with the acute onset of dense neurological findings often ends in disaster. A computed tomographic scan or magnetic resonance image showing no abnormalities in this early phase is no assurance that cellular death has not already occurred. Unfortunately, in this case, we are not given the benefit of blood flow studies nor of transcranial Doppler velocity studies, which might have given us a clue that we were dealing with a low-flow state. Perhaps, the major clinical feature of this case that would have suggested a favorable outcome was the continued angiographic flow in the large vessels despite high-grade stenosis.

Robert R. Smith
Jackson, Mississippi