

CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

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Case 5-2004: A 57-Year-Old Man with Slurred Speech and Left Hemiparesis

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PRESENTATION OF CASE

A 57-year-old right-handed man was admitted to the hospital because of the sudden onset of slurred speech and left hemiparesis.

The patient had been well until two days earlier, when he fell and fractured his left humerus. His daily aspirin was discontinued because of the fracture. He was last seen well by his mother-in-law at 2 p.m.; at 2:15 p.m., she noted that he was drooling, had slurred speech, and could not swallow pills. She called his wife, who came home from work immediately and found him unable to move his left arm or leg. He stated that he did not have a headache, nausea, or neck pain. He was brought to the hospital by ambulance at 4:30 p.m.

The patient had a history of coronary and bilateral carotid-artery atherosclerosis. Combined coronary-artery bypass grafting and left internal carotid endarterectomy had been performed 16 months before admission. He had asymptomatic, mild-to-moderate stenosis of the right internal carotid artery, well-controlled hypertension, hyperlipidemia, degenerative arthritis, and borderline diabetes and was obese. His medications were fluvastatin, irbesartan, ibuprofen, aspirin, and acetaminophen with oxycodone as needed for the recent fracture. He was allergic to penicillin. He lived with his wife and was employed as a civil servant. He had smoked and consumed alcohol in moderation in the past but had stopped doing both at the time of coronary-artery bypass grafting. There was no history of atrial fibrillation, clotting disorders, cardiac valvular disease, or use of illicit drugs.

The temperature was 36°C, the pulse 94 beats per minute, and the respirations 20 breaths per minute. The blood pressure was 178/84 mm Hg. The oxygen saturation was greater than 98 percent while the patient was breathing ambient air. On physical examination, he appeared acutely ill. The neck was supple and nontender. The carotid pulses were equal, and there were no bruits. The lungs were clear, and he could lie flat without aspiration or respiratory difficulty. The heart sounds were normal. The limbs were well perfused, and the abdomen was normal.

On neurologic examination, he was alert and oriented, and although he was inattentive he was able to recall recent events. He exhibited dense neglect (lack of aware-

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ness) with respect to objects or stimuli on his left side. He was unaware of his own deficits (a state termed anosognosia) and thought he was in the hospital for his heart condition. His left arm was in a sling, and when it was held in front of him he identified it as belonging to the examiner. He had mild dysarthria, but his speech was fluent and his comprehension, repetition, and naming abilities were intact.

He had complete left-sided homonymous hemianopia. The optic disks and retinal vessels were normal, the pupils were equal and reactive, and there was no ptosis. There was a conjugate rightward gaze deviation; he could not direct his gaze voluntarily past midline or to the left. On the left side of the face, he had severe weakness and no sensation of a pin prick. The head was turned to the right. The tongue was midline and moved well.

There was normal tone and full strength in the right arm and leg. He could not make voluntary movements with his left arm and leg, although occasionally the left leg spontaneously stiffened in extension. The deep tendon reflexes were normal on the right side and absent on the left. Plantar stimulation produced plantar flexion on the right and triple flexion (flexion of the hip and knee and dorsiflexion of the ankle) on the left. Gait testing was deferred. His score on the National Institutes of Health Stroke Scale (NIHSS) (which ranges from 0 to 34, with higher scores indicating greater deficits) was 20.

The results of urinalysis included 3+ glucose but were otherwise normal. Other laboratory values are shown in Table 1.

An electrocardiogram showed a sinus rhythm at a rate of 94 beats per minute with minor, nonspecific ST-segment and T-wave abnormalities. Chest radiographs showed evidence of a previous median sternotomy, clear lungs, and a normal cardiomedastinal silhouette. A cardiac ultrasonographic examination showed no vegetations, intracardiac thrombus, segmental wall-motion abnormalities, intracardiac shunts, or valvular disease.

Dr. James D. Rabinov: A computed tomographic (CT) scan of the head, obtained without the use of contrast material, shows a dense right middle cerebral artery, calcification of the basal ganglia on the left side, and no evidence of acute intracranial hemorrhage (Fig. 1A). Subtle hypoattenuation is seen in the right insular region, lentiform nuclei, and frontal lobe, with effacement of the sulci in the right frontal lobe.

DIFFERENTIAL DIAGNOSIS AND MEDICAL MANAGEMENT

Dr. Lee H. Schwamm: This 57-year-old man had a sudden onset of lateralizing neurologic deficits in the context of multiple vascular risk factors (Table 2) and presented to the hospital within three hours

Table 1. Laboratory Values on Admission.*

Variable	Value
Sodium (mmol/liter)	137
Blood urea nitrogen (mg/dl)	11
Creatinine (mg/dl)	0.8
Glucose (mg/dl)	270
White cells (per mm ³)	12,300
Hematocrit (%)	39.6
Platelets (per mm ³)	232,000
Prothrombin time (sec)	12.8†
Activated partial-thromboplastin time (sec)	21.4
Calcium (mg/dl)	7.1
Magnesium (mmol/liter)	0.65
Creatine kinase (U/liter)	201
Creatine kinase isoenzyme (ng/ml)	1.3
Troponin T (ng/ml)	<0.01
Albumin (g/dl)	2.1
Cholesterol (mg/dl)	
Total	147
High-density lipoprotein	37
Low-density lipoprotein	88
Triglyceride (mg/dl)	112
Functional protein S (% of normal value)	71
Functional protein C (% of normal value)	79
Anticardiolipin antibody‡	
IgG (GPL units)	2.2
IgM (MPL units)	1.8
Lupus anticoagulant	Negative
Activated protein C resistance	2.6

* To convert the value for blood urea nitrogen to millimoles per liter, multiply by 0.357. To convert the value for creatinine to micromoles per liter, multiply by 88.4. To convert the value for glucose to millimoles per liter, multiply by 0.05551. To convert the value for calcium to millimoles per liter, multiply by 0.250. To convert the value for magnesium to milliequivalents per liter, divide by 0.5. To convert the values for cholesterol to millimoles per liter, multiply by 0.02586. To convert the value for triglyceride to millimoles per liter, multiply by 0.01129.

† The control value was 12.3 seconds.

‡ GPL denotes IgG phospholipid, and MPL IgM phospholipid.

after the onset of symptoms. A patient, such as this one, with suspected acute ischemic stroke must be evaluated immediately in order to assess his or her eligibility for thrombolytic therapy. The evaluation includes rapid general and neurologic evaluations, diagnostic laboratory studies to rule out coagulopathy and electrolyte and hematologic disturbances, brain imaging, and review of the hospital's protocol for thrombolysis in acute stroke to identify factors that increase the risk of adverse outcomes.¹ Conditions that may contraindicate thrombolysis (e.g., brain hemorrhage and coagulopathy) and those that may simulate acute stroke must also be identified (Table 3). If the presence of ischemic stroke is confirmed, an evaluation of its possible causes should be performed during hospitalization so as to identify opportunities for the prevention of secondary strokes.

Thrombolytic therapy with intravenous tissue plasminogen activator (t-PA) for acute ischemic stroke increases by 30 percent the likelihood that the patient will have minimal or no disability at three months, although there is an increased rate of symptomatic hemorrhage during the first 36 hours after the start of treatment.² Treatment within the first 90 minutes after the onset of symptoms is associated with a better outcome than later treatment,³ and the response to treatment is not affected by evidence of ischemic changes on CT.⁴ Current guidelines recommend the intravenous administration of t-PA for all patients with acute ischemic stroke and no contraindications who can be treated within three hours after the onset of stroke symptoms (grade A). In patients for whom the three-hour window has passed, intraarterial fibrinolysis through a catheter within three to six hours after the onset of symptoms may be beneficial if there is occlusion of the middle cerebral artery (grade B).^{5,6}

In this patient, the CT finding of a bright linear signal in the region of the right middle cerebral ar-

tery suggests the presence of a blood clot, and the subtle early ischemic changes in the territory served by this artery reinforce the diagnosis of evolving infarction. The CT study rules out the presence of in-

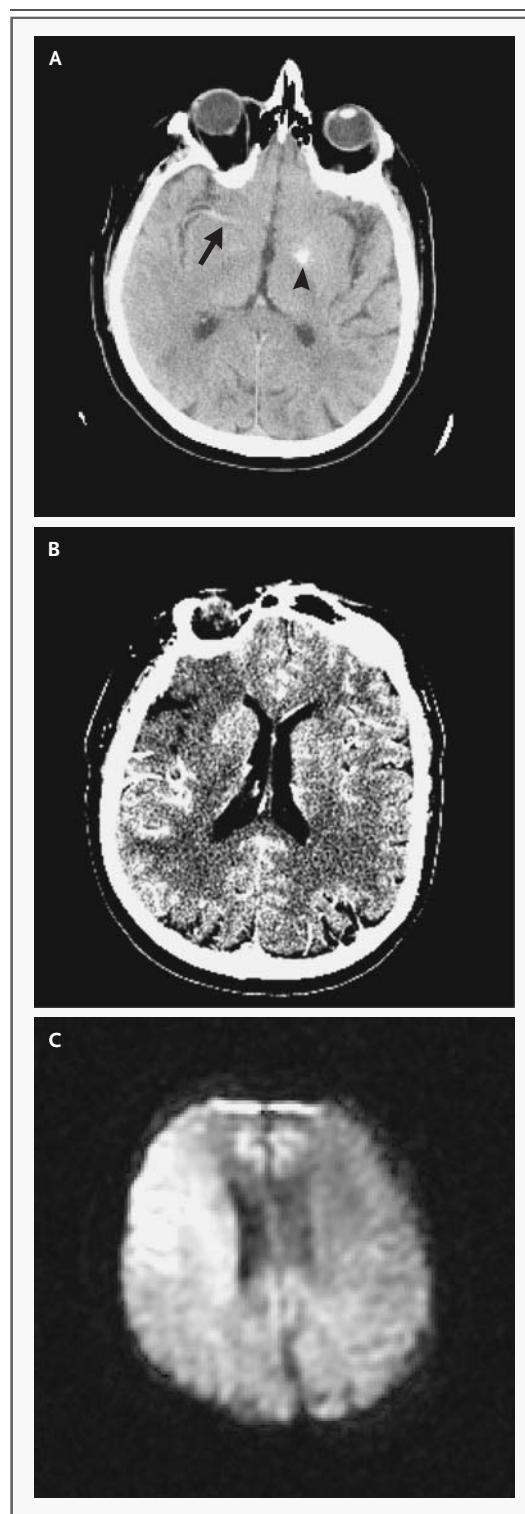


Figure 1. Images of the Brain on Admission.

A cranial CT scan, obtained without the use of contrast material (Panel A), shows a dense right middle cerebral artery (arrow), indicating the presence of a clot in the vessel. Benign calcifications are present in the basal ganglia on the left side (arrowhead). A perfusion window of the axial image of the CT angiogram (Panel B) shows hypoperfusion in the right frontal operculum, manifested as a dark area within the cortex. A diffusion-weighted MRI scan (Panel C) shows that diffusion is restricted (bright area) in the right frontal lobe, indicating the presence of acute infarction.

Table 2. Neurologic Findings on Presentation.

System	Finding
Mental status	Alert, dense left-sided neglect, and dysarthria, but no aphasia
Cranial nerves	Left-sided hemianopia, facial weakness, and sensory loss with rightward gaze deviation
Motor system	Complete left hemiplegia with triple flexion response
National Institutes of Health Stroke Scale*	20

* The scale ranges from 0 to 34, with higher scores indicating more severe deficits.

Table 3. Conditions That Mimic Ischemic Stroke.

Cerebrovascular disease	
Intracerebral hemorrhage	
Subdural or epidural hematoma	
Hypertensive emergency	
Toxic or metabolic state	
Hypoglycemia	
Electrolyte disturbance (especially involving sodium)	
Uremia	
Intoxication	
Severe infection or sepsis	
Other	
Complex partial seizure with Todd's paralysis	
Classic migraine	
Psychiatric disorder	

intracranial bleeding, and the laboratory studies do not identify other nonischemic causes of the patient's symptoms or contraindications to the use of t-PA. He is thus eligible for intravenous t-PA administered within three hours after the onset of symptoms, and this option should be discussed with the patient and family. The recent arm fracture may represent a contraindication, and there is an increased risk of hemorrhage in patients older than 75 years of age, those with elevated blood glucose levels, and those with severe deficits (NIHSS score, >22). In the National Institute of Neurological Disorders and Stroke Recombinant t-PA Stroke Study, higher glucose levels on admission were associated with a significantly lower likelihood of a good clinical outcome and a significantly higher likelihood of symptomatic intracranial hemorrhage, regardless of the use of t-PA treatment.⁷

Do we need to worry about this patient's elevated blood pressure? Many patients have hypertension after an ischemic stroke, but few require urgent treatment. Elevated blood pressure after a stroke is not a hypertensive emergency unless there are other medical problems (e.g., myocardial infarction or aortic dissection). Antihypertensive treatment is reserved for patients who have markedly elevated blood pressures, those with specific medical indications, and those with ischemic stroke who are candidates for fibrinolytic therapy and have pressures in excess of 180/105 mm Hg.^{2,7} Thus, emergency management of hypertension is not indicated in this patient.

Effective recanalization of the occluded vessel after treatment with t-PA is less likely with proximal vascular occlusions than it is with distal ones; reperfusion rates in cases of carotid occlusion are only about 8 percent.⁸ In several studies of occlusion of the middle cerebral artery that was treated within six hours after the onset of symptoms, catheter-based thrombolytic therapy was successful in producing recanalization in up to 66 percent of the cases, improved the clinical outcome,^{9,10} and confirmed the adverse effect of hyperglycemia on the risk of hemorrhage.¹¹ Major complications of catheter-based thrombolytic therapy also include systemic hemorrhage and increased intracranial pressure due to progressing stroke, reperfusion edema, or intracranial hematoma. Patients with both occlusion of the internal carotid artery and occlusion of the middle cerebral artery are often excluded from major catheter-based trials, and other reports have suggested that this pattern of vascular injury is associated with a poor clinical response after intravenous t-PA therapy.^{12,13} Successful thrombolysis of occlusion of the middle cerebral artery after angioplasty of the totally occluded internal carotid arteries has been reported.^{14,15}

Additional cerebral and neurovascular imaging may be helpful in confirming the diagnosis of ischemic stroke and in assisting in the management of patients such as this one, especially if there is proximal vascular occlusion that may be preferably treated with catheter-based reperfusion techniques.¹⁶ CT angiography with perfusion (whole-brain imaging of the perfused blood volume) is a rapid, well-tolerated, minimally invasive method of viewing the cerebral vasculature and may identify sites of occlusion as well as regions of microvascular collapse destined for infarction.^{17,18} New magnetic resonance imaging (MRI) techniques, such as

magnetic resonance angiography and diffusion- and perfusion-weighted MRI, may help delineate the site of occlusion or the region of the brain at risk for infarction.¹⁹ They may also help to predict the final infarct volume and clinical outcome.²⁰

After we reviewed the imaging findings and discussed the risks and benefits of various reperfusion strategies with the patient's wife, she elected to proceed to catheter-based angiography with possible intraarterial thrombolysis. The endovascular team was called, and while we awaited their arrival, the patient was taken to the angiography suite and general endotracheal anesthesia was induced.

Dr. Rabinov: After CT of the brain, CT angiography was performed from the aortic arch to the vertex with rapid injection of intravenous contrast material. Multiplanar reformatted and three-dimensional images were obtained for evaluation. The contrast-enhanced axial images of the brain showed reduced perfused blood volume in the right insular region, lentiform nuclei, and frontal lobe, with sparing of the right caudate head (Fig. 1B). CT angiographic images showed occlusion of the right internal carotid artery at the bifurcation; there was no contrast opacification up to the right middle cerebral artery stem, and few distal right branches of the middle cerebral artery were seen. There was flow in the right anterior cerebral artery, most likely as a result of cross-filling.

A limited MRI sequence was obtained approximately three hours after the onset of the stroke. On the diffusion-weighted images (Fig. 1C), there was a large area of abnormally restricted diffusion, mostly in the superior division of the right middle cerebral artery but also involving part of the inferior division and extending just below the right motor strip.

Diagnostic transfemoral angiography was begun 3 hours and 30 minutes after the onset of the stroke. Injection of the right common carotid artery confirmed the abrupt occlusion of the right internal carotid artery approximately 1 cm beyond its origin (Fig. 2A); the stump had an irregular contour. The occluded segment (Fig. 2B) was crossed with a wire, and repeated angiography showed patency of the distal cervical internal carotid artery, with high-grade stenosis and very slow flow.

After angioplasty and stenting and injection into the internal carotid artery of 5 mg of t-PA, the right internal carotid artery became patent (Fig. 2C). Injection of the right internal carotid artery showed partial occlusion of the right anterior cerebral ar-

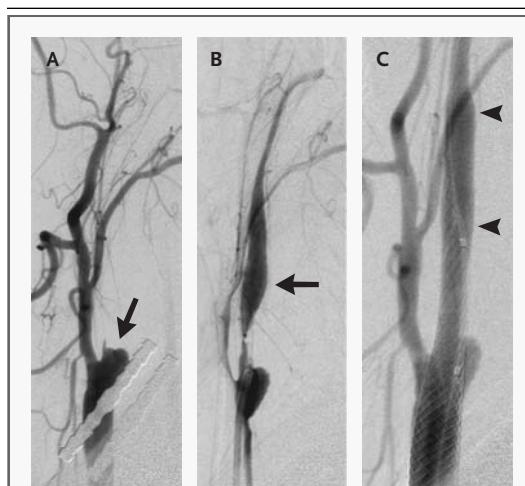


Figure 2. Angiograms of the Right Common Carotid Artery in the Lateral View.

The right internal carotid artery is cut off at its origin (Panel A, arrow). After exploration with a microcatheter and wire, severe narrowing of that artery and poor distal flow (Panel B, arrow) are visible. After thrombolysis and stent placement, the right internal carotid artery is patent (Panel C, arrowheads).

tery and the superior division of the right middle cerebral artery (Fig. 3A). Thrombolysis of the middle and anterior cerebral arteries was performed with 8 mg of t-PA. On completion of the procedure, at 7 hours and 30 minutes after the onset of the stroke, blood flow was normal in all segments of the right internal carotid artery, the right anterior cerebral artery, and the middle cerebral artery except its inferior division, in which recanalization was never achieved (Fig. 3B). CT scanning of the brain immediately after the procedure showed a small hemorrhage in the right putamen and contrast staining in the right frontal lobe — findings indicating improved perfusion.

Dr. Schwamm: In summary, the CT angiogram confirmed the presence of embolic occlusion of the right middle cerebral artery and identified the likely source as an embolism from the acutely occluded right internal carotid artery. In some patients, a clot may extend from the origin of the internal carotid artery all the way up to its distal bifurcation, but here (as in many cases), there is merely collapse of the intervening lumen, and the opportunity for reperfusion remains. The diffusion-weighted MRI scan confirms early acute infarction in part of the territory of the middle cerebral artery but identifies a

mismatch between the clinical picture (dysfunction of the entire territory of the middle cerebral artery) and the radiographic picture (partial ischemia of this territory).

After thrombolysis, the patient was transferred to the neurology intensive care unit, where he began to show some movement of his left side. However, over the next 12-hour period he became increasingly somnolent, despite normal flow in the middle cerebral artery, according to transcranial Doppler ultrasonography.

Dr. Rabinov: CT scanning of the brain at 20 hours revealed progressive edema with hemorrhagic transformation of the infarction in the area of the right middle cerebral artery and a leftward shift of the midline (Fig. 4A).

Dr. Schwamm: At this point, aggressive therapy to lower the intracranial pressure and maintain cerebral perfusion is mandatory to prevent compromise of the salvaged cortical brain tissue and potential uncal herniation. Choices include hyperventilation, hyperosmotic therapy, barbiturates, external ventricular drainage, hypothermia, or hemicraniectomy with duraplasty (Table 4). Mannitol (100 g) was administered intravenously, and a vascular neurosurgeon was consulted.

Dr. Rolf Pfannl: The consequences of cerebral edema due to stroke are often documented at au-

topsy. The brain swelling associated with acute cerebral ischemia is the result of a combination of factors. When vascular occlusion occurs, edema initially results in part from shifts in ions between the intracellular and extracellular compartments; this change leads to an accumulation of excess water within astrocytes and other cells, a change that in turn leads to swelling. With subsequent damage to

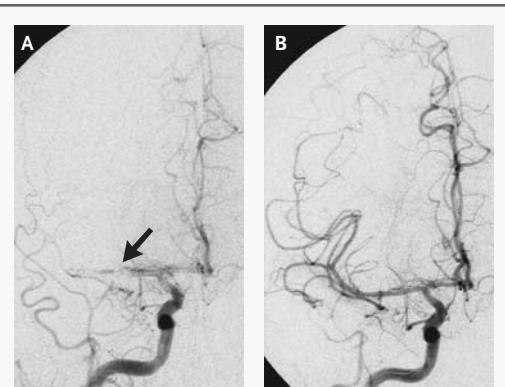
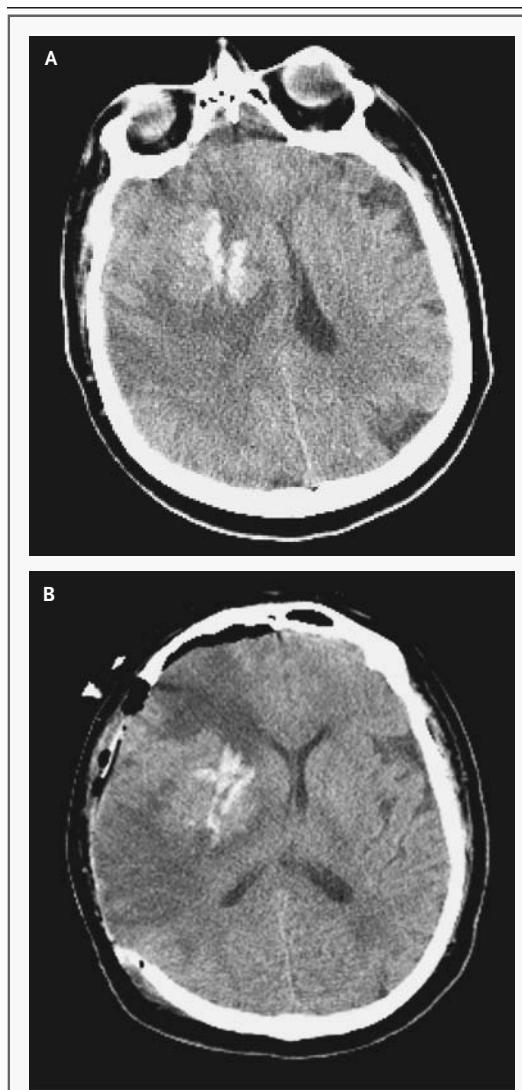


Figure 3. Angiograms of the Right Internal Carotid Artery and Its Branches after Stenting.

An initial angiogram (Panel A) shows partial occlusion of the right anterior cerebral artery and of the superior division of the right middle cerebral artery (arrow). An angiogram obtained after thrombolysis (Panel B) shows that flow has been reestablished in the right anterior cerebral artery and in the superior division of the right middle cerebral artery.

Figure 4. CT Scans of the Brain.

Twenty hours after admission, there is progressive swelling in the right hemisphere, with a shift of the midline toward the left (Panel A). After hemicraniectomy (Panel B), the brain has herniated out through the bony defect, and the infarction has evolved; however, the midline shift has resolved.

endothelial cells, there is a disruption of the blood-brain barrier, leading to the passage of water from the intravascular to the extravascular space, and reperfusion at this time may contribute to the vaso-genic edema. This acute swelling of the brain leads to swelling of the gyri, narrowing of the sulci, and flattening of the gyri at the site of compression against the overlying skull, as well as a shift of the brain across the midline (Fig. 5A). Nonatherosclerotic vessels are compressed and may be occluded. With increasing edema, the swelling brain herniates across the tentorium (Fig. 5B), leading to pressure necrosis of the herniated brain and to increased pressure on the brain stem. Large autopsy series have shown that most deaths that occur during the first week after a stroke are due to such transtentorial herniation.²¹

SURGICAL MANAGEMENT

Dr. Bob S. Carter: This patient's initial infarction volume was about 170 cm³, and the final radiographic studies show a midline shift of about 1 cm. For massive infarctions caused by occlusion of the trunk of the middle cerebral artery or the internal carotid artery, mortality can be 80 to 90 percent. Most of these deaths are due to transtentorial herniation, the so-called malignant middle-cerebral-artery edema syndrome.^{21,22}

Who is at risk for this syndrome? In 2000, Oppenheim et al.²³ reported that the best predictor of progression to a full-blown clinical syndrome of uncal herniation was an initial infarction volume on diffusion-weighted MRI of more than 145 cm³. In this patient, the CT scan that was obtained just before our consultation shows hemorrhagic transformation in the ipsilateral hemisphere and a 1-cm midline shift. The overall volume of infarction had

progressed to more than 200 cm³. Conservative measures, including osmotherapy with mannitol, had already been initiated, but the patient's condition continued to deteriorate.

Surgical decompression strategies for cerebral infarction fall into two broad categories: measures that actually open the intracranial contents and

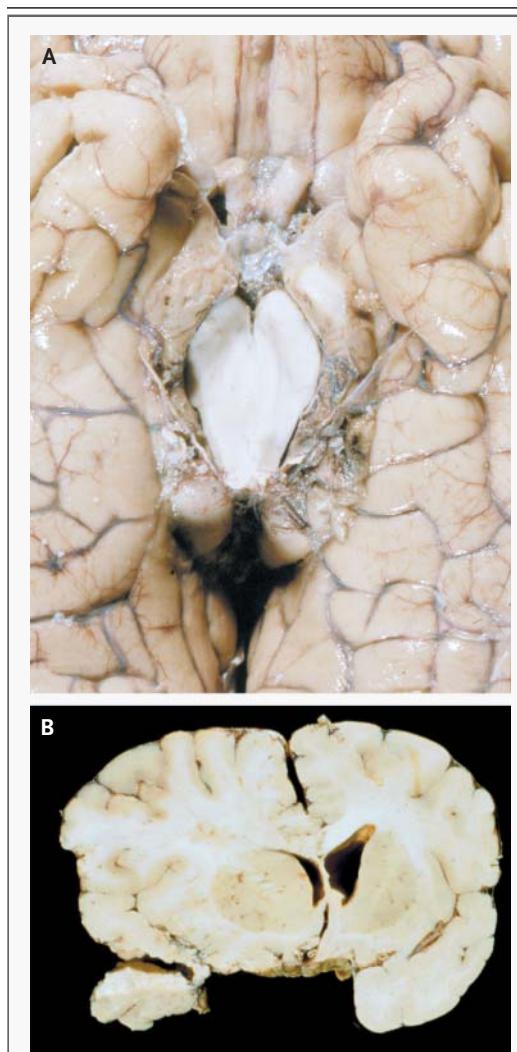


Figure 5. Photographs Obtained on Gross Examination of the Brain (from Other Patients), Showing the Effects of Stroke on the Brain.

A coronal section of the brain with an infarct of the right middle cerebral artery shows swelling of the right cerebral hemisphere and a shift of the midline structures to the left (Panel A). A basilar view shows transtentorial herniation of the temporal lobe with compression of the brain stem (Panel B).

Table 4. Management Options for Increased Intracranial Pressure.

- Avoidance of hypoxemia and hypoventilation
- Control of agitation and pain
- Hyperventilation
- Osmotic therapy
- Barbiturates
- External ventricular drainage
- Hypothermia
- Hemicraniectomy with duraplasty

measures that open the confines of the intracranial vault. The removal of infarcted tissue, or "strokectomy," is a well-established strategy for treating large, life-threatening cerebellar infarctions with impending brain-stem compression. Hemicraniectomy and duraplasty (opening of the dura mater) to expand the intracranial space are used primarily to reduce the risk of death associated with the malignant middle-cerebral-artery edema syndrome. A secondary goal is to improve the overall neurologic outcome by intervening before the development of uncal herniation and the fixed neurologic deficit that results from brain-stem compression. In addition, early intervention may prevent recruitment of additional vascular territories into the infarction — a process that would result from compression of the posterior cerebral artery or compression of the anterior cerebral artery at the edges of the tentorium and falx, respectively. Finally, early intervention may improve cerebral blood flow in regions at the periphery of the infarction and offer the hope of improvement in functional outcomes.

The initial studies of hemicraniectomy focused on patients in whom frank uncal herniation had developed as a result of edema after cerebral infarction.^{24,25} In our initial experience with 13 patients who had uncal herniation after a stroke and who were treated with decompressive craniectomy with duraplasty, the mortality rate was 20 percent, as compared with an expected mortality of 80 to 90 percent among untreated patients. In addition, 8 of the 11 survivors were ambulatory, and 3 of those who could walk could do so without assistive devices. Similar results were reported in another early study.

Hemicraniectomy performed before the onset of uncal herniation may improve the outcome.²⁶ Age and hemispheric language dominance are important considerations in selecting patients. The best outcomes have been observed in younger patients.^{27,28} In our current series of 38 patients, those who could walk without assistance were an average of 36 years old, those who could walk with an assistive device were an average of 48, and those who were unable to walk were an average of 60. Most studies have limited the use of this procedure to nondominant hemispheric infarctions, because patients with an infarction in the dominant hemisphere frequently have profound aphasia. However, we do offer this surgery to young patients with incomplete language deficits, having observed functionally independent outcomes in these patients as well.

Considerations in the case of the patient under discussion are as follows. The size of his stroke and his worsening clinical syndrome raise the concern of imminent herniation, but a full-blown herniation syndrome has not yet developed, so the opportunity to prevent irreversible brain-stem injury may be available. At the age of 57, he is not in the most favorable group; however, patients in this age range can achieve outcomes associated with physical independence. His infarction is in the nondominant hemisphere. After consultation with the patient's family, we elected to proceed with decompressive hemicraniectomy.

In the operating room, the skin of a large area of the right side of the scalp was reflected anteriorly and a large portion of the frontal, parietal, and temporal bones was removed. Our goal was to achieve a craniectomy 125 cm² in area. As the dura was opened, the infarcted brain herniated out through the dural defect. This dural opening is thus very important for allowing normalization of the intracranial pressure.^{29,30} In addition, to avoid brain-stem compression by the uncus of the temporal lobe, which occurs in many patients, we performed an anterior temporal lobectomy to decompress the brain stem.

In these procedures, we do not attempt to reapproximate the dura in order to close the deficit; rather, we place a graft of pericranium or artificial collagen to allow further expansion of the infarcted swollen brain over the next several days. The bone that was removed was implanted subcutaneously in the abdominal wall, so that it could be replaced when the cerebral swelling had resolved.

Dr. Rabinov: A CT scan of the brain obtained after the operation shows correction of the midline shift and herniation of the brain out through the bony defect (Fig. 4B).

Dr. Carter: The volumetric expansion of the intracranial vault by the removal of bone allowed us to normalize the intracranial pressure, which was measured postoperatively to be 1 to 5 cm of water. The patient awakened, his trachea was extubated, and he began to follow commands briskly on the right side and could wiggle toes on the left. His left arm remained paralyzed. Eight days after the stroke, anti-coagulant therapy was reinitiated with aspirin and clopidogrel. Seventeen days after the stroke he had an episode of aspiration, which required reintubation. A percutaneous endoscopic gastrostomy tube and a tracheostomy were placed to protect his airway during rehabilitation. He was discharged to a rehabilitation facility one month after the stroke.

Three months after his stroke, we performed cranial reconstruction with his autologous bone flap.

Dr. Randie M. Black-Schaffer (Physical Medicine and Rehabilitation): The patient spent five months in inpatient rehabilitation, with his stay interrupted by the cranial reconstruction surgery. On presentation for rehabilitation, he still had no voluntary movements on the left side. By the time of discharge, he had achieved moderate recovery of the motor function of his left leg, with a Medical Research Council strength of 2/5 in the proximal left leg and no distal movement. There was no recovery in the left arm. The left-sided hemianopia persisted; the left-sided neglect and anosognosia showed modest improvement with occupational therapy. He achieved functional ambulation, requiring a contact guard (the therapist would guide him with a hand on his trunk or shoulder) and minimal assistance (the therapist performed up to 25 percent of the work of the task) to walk 250 feet with rests, with the use of a large-base quad cane and a custom orthosis for the ankle and foot. He could transfer from a bed to a chair with a contact guard and minimal assistance. He became independent in upper-body bathing and dressing but required moderate-to-maximal assistance (25 to 75 percent of the work done by the

therapist) for the lower body, particularly with placement of the brace. This pattern of motor and functional recovery is typical of patients with a stroke in the territory of the right middle cerebral artery. During inpatient rehabilitation, the patient's tracheostomy and gastrostomy tubes were removed, and he was able to resume a normal diet. He was discharged six months after his stroke, and at eight months he was able to return to work on an approximately half-time basis.

Dr. Paul Chapman (Neurosurgery): Were noninvasive carotid ultrasound studies done before the procedure was performed?

Dr. Schwamm: CT angiography provides vascular contrast imaging from the aortic arch to the vertex, giving a complete view of the course of the internal carotid artery. As a result, it is unnecessary to obtain carotid duplex Doppler ultrasound images, unless severe calcification limits the interpretation of the study.

FINAL DIAGNOSIS

Stroke involving the right middle cerebral artery, with the malignant middle-cerebral-artery edema syndrome.

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