SUMMARY  Cerebral hemorrhage, an unusual complication of endarterectomy, is often attributed to postoperative hypertension. A patient with a middle cerebral artery stroke had a hemorrhage without hypertension, 2 days postoperatively. Pronounced contrast enhancement of the infarction during the 7 weeks prior to surgery reflected severe disruption of the blood-brain barrier. Contrast CT scans may detect patients at risk for post-endarterectomy hemorrhage even 2 months after a stroke or TIA.

CEREBRAL HEMORRHAGE is a rare occurrence after carotid endarterectomy, usually ascribed to postoperative hypertension, and generally occurring in the region of recent infarction. We report a patient whose CT scan showed infarction with pronounced and prolonged contrast enhancement; a postoperative hemorrhage, seven weeks after the stroke, was unassociated with hypertension. Marked contrast enhancement may indicate extensive damage to vessels within or surrounding the infarction, perhaps warranting routine contrast infusion scans prior to an endarterectomy and delay of the procedure until enhancement subsides.

Case History

A 72 year old normotensive man had acute left sided weakness and slurred speech, preceded by several brief episodes of weakness of the left arm and leg. Carotid non-invasive studies showed severe right internal carotid stenosis and complete occlusion on the left. He was treated with twice daily aspirin and dipyridamole. Examination showed a BP of 150/70, mild left hemiparesis with facial weakness, and extinction of left sided visual and tactile stimuli. Laboratory values included Hct 43.3%, platelet count of 269,000, BUN 15 mg%, PT 10.4/10.5 seconds, PTT 29.4 seconds, and ESR 69. Transcranial doppler studies showed peak and mean doppler shift frequencies in the middle cerebral arteries of 2240 and 1240 Hz on the right, and 1720 and 1120 Hz respectively on the left, all normal for our unit. Flow in the proximal right anterior cerebral artery was toward the right. The patient was alert, napped frequently, and was at times disoriented. He usually denied his left sided weakness. Cerebral angiography 26 days after the stroke showed an occluded proximal left carotid artery with retrograde flow through the ophthalmic artery, filling the left hemisphere. Both anterior cerebral arteries filled from the right carotid injection. The left vertebral and posterior communicating arteries were patent. The right internal carotid artery had a greater than 90% stenosis in one projection and greater than 70% in another. The right middle and anterior cerebral arteries were normal. Specifically, there was no increased vascularity in the region surrounding the infarction.

Four weeks after the stroke, a CT scan (10 minute intravenous infusion of 300 ml of Urovist) showed gyriform and some white matter enhancement in the superior division of the right middle cerebral artery and anterior and lenticulostriate territories (fig. 1). A noncontrast scan 3 days later showed no definite hemorrhage and outlined the infarction. Because of prominent contrast enhancement, the right carotid endarterectomy was deferred. Aspirin was discontinued and intravenous heparin was begun. A CT scan with contrast infusion (same dose), 5 weeks and 4 days after the stroke, showed approximately the same degree and distribution of enhancement. Six weeks and 4 days after the stroke, a CT scan with contrast infusion (same dose) showed prominent but reduced gyral enhancement (fig. 2). Heparin was continued; PTT was between 42.5 and 95.4 seconds.

A right carotid endarterectomy was performed seven weeks after the stroke with an intraoperative shunt and EEG monitoring, without complications. During the 3 weeks prior to surgery, blood pressure remained between 120–160 mmHg systolic and 60–90 mmHg diastolic. Mild postinduction hypotension, 120/60 mmHg, was treated with neosynephrine. Heparin 3000 U was given intravenously after dissection of the vessels. The shunt was opened 4 minutes after clamping. Hypertension (165/85 mmHg) during the last 15 minutes of surgery was treated with intravenous nitroglycerin. The patient awakened quickly. Blood pressure measured every 15 minutes in the recovery room remained below 130 mmHg systolic except for 2 hours following surgery, when blood pressure of 190–220/80–100 mmHg responded to topical and intravenous nitroglycerin. Blood pressure remained normal without anti hypertensive medication beyond the immediate postoperative period. Heparin was not restarted. Forty-four hours after the endarterectomy, he had a tonic clonic convolution of the left arm with head deviation to the left, responding to intravenous anticonvulsants. A brief period of hypotension followed phenytoin administration. A CT scan showed hemorrhagic regions within and adjacent to the previous infarction, including a small round frontal hemorrhage partly within the anterior cerebral artery territory (fig. 3). Aspirin and dipyridamole, administered for one day, were discontinued. Residual dysarthria and left arm and leg weak-

From the Neurological/Neurosurgical ICU, Massachusetts General Hospital, Boston, Massachusetts 02114 U.S.A.

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Address correspondence to: Allan H. Ropper, M.D., Neurological/Neurosurgical ICU, Massachusetts General Hospital, Boston, Massachusetts 02114 U.S.A.

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CT Scan with contrast infusion (300 ml Urovisi) four weeks after right middle cerebral artery infarction, showing gyriform and periventricular enhancement of portions of the infarction.

FIGURE 1. CT scan with contrast infusion (300 ml Urovisi) four weeks after right middle cerebral artery infarction, showing gyriform and periventricular enhancement of portions of the infarction.

FIGURE 2. CT scan with contrast infusion (300 ml Urovisi), six weeks, 4 days after the stroke showing less gyriform enhancement than 2½ weeks previously (fig. 1); some frontal enhancement remains. The infarction is now seen.

FIGURE 3. CT scan without contrast, 2 days after endarterectomy showing blood in the right frontal and parietal lobes and probably subarachnoid blood in sulci posteriorly. There is mass effect that was not present previously.

Discussion

Cerebral hemorrhage occurs infrequently after carotid endarterectomy; 0.6% in a large series two decades ago, and 0.4% recently. Patients usually have had recent strokes or TIA's on the operated side, but other factors such as hypertension, anticoagulants including aspirin, or excessive regional perfusion following the stroke may be involved. Most surgeons...
state that hemorrhage is a risk only in the several weeks following a stroke.5,6 Breutman has suggested that postoperative hemorrhage has several possible origins, emphasizing "luxury perfusion" seen on the CT scan as the most important cause.1,7 Our patient had hemorrhages in the region of a 7 week old infarction, 2 days and again 2 months after endarterectomy, without immediately preceding hypertension. Prominent contrast enhancement surrounding the infarction may have indicated breakdown of the blood-brain barrier or excessive regional perfusion.

The mechanism of prominent contrast enhancement is uncertain. It has generally been attributed to disruption of the vascular barrier rather than luxury perfusion or loss of autoregulation, both phenomena that occur earlier and should not have persisted for so long in our patient. A relation of contrast enhancement to elevated cerebral blood flow and convulsions or hemorrhages after endarterectomy has been suggested but is uncertain.2 Ipsilateral headache following endarterectomy may also be related to hyperperfusion of a previously hypoperfused hemisphere. Endarterectomy in a normotensive man resulted in a fatal cerebral hemorrhage emphasizing "luxury perfusion" seen on the CT scan as the most important cause.1,7 Our patient had hemorrhages in the region of a 7 week old infarction, 2 days and again 2 months after endarterectomy, without immediately preceding hypertension. Prominent contrast enhancement surrounding the infarction may have indicated breakdown of the blood-brain barrier or excessive regional perfusion.

The frequency of contrast enhancement in cerebral infarction, up to two-thirds by the fourth week,8-11 depends on the method of contrast infusion. With bolus infusions almost all infarctions enhance within a month; with infusion of dye over approximately 10 minutes, 25%9 to 40% enhance. The proportion of cases with enhancement is generally highest at the end of the second week.

The nature of the hemorrhage is similar in most cases following endarterectomy. A heterogeneous, sometimes widespread lesion occurs, and convulsions are common at the outset. The degree and duration of contrast enhancement of the infarction in our patient led to repeated delay of the planned endarterectomy. In some reports, similar patterns of enhancement have simulated arterio-venous malformations.10,12,13 This probably represents extreme breakdown of the blood-brain barrier, a common but less prominent feature after most strokes. Exposing the disrupted vasculature to normal or increased perfusion pressure may risk bleeding into the infarction. Perhaps postoperative hypertension causes bleeding in patients with less severely disrupted cerebral vessels; normotensive hemorrhages might occur only when there is a severe disturbance of vessels. Contralateral carotid occlusion in our patient may have caused a greater than usual blood flow on the operated side. It has been suggested, though pathological confirmation is lacking, that contrast enhancement indicates minimal hemorrhagic infarction not visible on an unenhanced CT.14

This case of postoperative hemorrhage without hypertension does not diminish the need to prevent hypertension after endarterectomy. It may, however, be advisable to perform CT scans with contrast infusions if a stroke or TIA has occurred within several weeks of an endarterectomy.7 Patients with contralateral carotid stenosis or occlusion may be at additional risk for postoperative hemorrhage.

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Prevention of Early Restenosis and Thrombosis-Occlusion After Carotid Endarterectomy by Saphenous Vein Patch Angioplasty

JOSEPH P. ARCHIE, JR., PH.D., M.D.

SUMMARY The hypothesis that saphenous vein patch angioplasty protects against early postoperative restenosis and thrombosis-occlusion was tested by comparing the clinical outcome and carotid artery status of 100 carotid endarterectomies with and 100 without saphenous vein patch angioplasty performed by a single surgeon over a 30-month period. The patient population, selection, perioperative management, and the technical aspects of the operation, except for the vein patch, were essentially identical in both groups. Carotid artery status was assessed by direct continuous wave Doppler and Gee OPG at three to six months and again at one year postoperatively. There were two hospital deaths, both in the nonpatched group, one cardiac and the other neurologic due to internal carotid thrombosis. Two reversible neurological deficits due to thrombosis and one due to restenosis occurred in the non-patched group. Asymptomatic > 50% diameter restenosis occurred in four and asymptomatic occlusion in one non-patched carotid. There were no restenosis, no occlusions and no neurologic symptoms in the patched group. Morbidity, mortality, restenosis or thrombosis-occlusion occurred in 9/100 (9%) of non-patched and 0/100 (0%) patched arteries (p < 0.01 by Chi Square). Restenosis or thrombosis-occlusion occurred in 9/100 (9%) of non-patched and 0/100 (0%) patched arteries (p < 0.01). These results support the use of saphenous vein patch angioplasty reconstruction of carotid endarterectomy to protect against early restenosis and thrombosis-occlusion.

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Contrast enhancement CT scan and post-endarterectomy hemorrhage.
A H Ropper and S M Kehne

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