Intracerebral Hemorrhage Following Carotid Endarterectomy: A Hypertensive Complication?

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SUMMARY Two patients with transient ischemic attacks and subsequent minor cerebral infarction had repair of very tight carotid stenosis, 4 and 5 weeks respectively after their stroke. Each developed intracerebral hemorrhage when hypertension was uncontrolled during the post-operative period. Hypertension is a significant complication of carotid endarterectomy, and may be a prominent factor in the development of intracerebral hemorrhage post-carotid endarterectomy.

CAROTID endarterectomy has become a common operative procedure for ischemic cerebrovascular disease. Morbidity and mortality has gradually declined with further surgical experience and careful selection of appropriate surgical candidates.1, 2 Cerebral ischemia during operation, and perioperative myocardial infarction are the most frequent serious complications. Previous reports have cautioned against the performance of carotid endarterectomy during the first 3 weeks after an established cerebral infarction because of the increased risk of subsequent hemorrhage into the region of infarction.3, 4 Wylie et al.5 commented in 1963: “There appears to be a critical period following cerebral infarction during which restoration of blood flow may cause serious or fatal intracranial hemorrhage adjacent to the infarcted zone and that reconstructive vascular operations may be performed safely during this period only when infarction has not occurred. Additional clinical and experimental observations are now needed in order to define more accurately the limits of this critical period.”

The present report describes 2 hypertensive individuals with severe unilateral carotid stenosis who underwent carotid endarterectomy 5 and 4 weeks respectively after cerebral infarction. Each suffered fatal intracerebral hemorrhage when hypertension was not controlled in the post-operative period. We wish to emphasize the possible occurrence of this complication, even when operation is delayed for 5 weeks after a cerebral infarct, and to emphasize the importance of hypertension in its pathogenesis.

Case Reports

Patient 1: A 57-year-old white female had approximately 12 transient spells of numbness of her left fingers or face. Each lasted less than a minute. On November 26, approximately 2 months after the initial transient episode, she awoke from a nap with slight numbness and weakness of the left arm. By the next morning, a moderate left hemiparesis had evolved, which improved during the next week.

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response and required controlled ventilation. An electroencephalogram was flat. She remained unresponsive without brainstem reflexes or electrocerebral activity until Jan. 3, when ventilatory support was discontinued. Postmortem examination was refused.

**Patient 2:** A 45-year-old black female was admitted to the hospital following an episode of weakness of the right arm and leg and inability to express herself. Within a few minutes she had regained enough strength in the lower extremity to walk to the car with minimal assistance. On arrival at the hospital 40 minutes later, almost full limb strength had returned, but she still had clumsiness in her fingers and difficulty in finding the proper words to make her needs known. The latter problem persisted for another 3 days, but after that it was apparent only when she was fatigued.

The patient had experienced 2 similar episodes of shorter duration during the previous 3 months. She had been on antihypertensive medication for 2 years with good control of her blood pressure; however, on one or two occasions it had been as high as 190/110 mm Hg.

Her admission blood pressure was 170/100 mm Hg, decreasing to 160/95 after 24 hours of bedrest and administration of methyldopa (Aldomet), 250 mg t.i.d. No bruits were heard over the cervical arteries. A computerized axial tomogram revealed a small infarct in the left posterior frontal region. Because of this lesion, it was decided to defer arteriography for several weeks during which the patient would receive physical therapy. Approximately 1 month after her last acute episode, selective 4-vessel aortocranial arteriography was performed by femoral artery catheterization, and the procedure was well tolerated. A pinhole lumen was demonstrated at the origin of the left internal carotid artery, and stenosis of about 50% was present on the right at the same level. There was no evidence of cross filling to either side. The day following the arteriographic study, with the patient under general anesthesia, left carotid endarterectomy was performed employing an internal shunt. Intraoperative or postoperative arteriography was not done. The patient was alert when she awakened from the anesthetic, and seemed to have less difficulty com-
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municating than she did before the operation. Her blood pressure recorded in the recovery room was 200/115 mm Hg, and it remained elevated in spite of vigorous efforts to reduce it. The following morning, while the patient was being helped to the bathroom, she had a tremendous headache and slumped to the floor. By the time she was returned to bed she had developed a right hemiplegia and aphasia. Soon thereafter, she became comatose, her pupils were dilated and fixed, and she was dead within 90 minutes. Postmortem examination showed a massive hemorrhage in the left frontotemporal region with rupture into the ventricular system. The previously infarcted area could not be identified, but some small petechial hemorrhages were present adjacent to the lateral and superior margins of the zone of hemorrhage.

**Discussion**

These 2 patients had the following features in common:

1. Significant hypertension, a fixed minor neurological deficit of at least 4 weeks duration, and very severe unilateral carotid artery stenosis preoperatively.
2. A symptom-free interval postoperatively, followed by the abrupt onset of systemic hypertension and sudden progressive cerebral hemorrhage on the side of the carotid endarterectomy and prior cerebral infarction.

Prior reports detail 15 cases of intracerebral hemorrhage in the period following carotid endarterectomy. In 11 of these cases, transient ischemic attacks had preceded a stroke. Thirteen patients had clinically recognizable neurological deficits at the time of surgery (the other 2 patients had focal slowing of the EEG on the side of the carotid stenosis). Eleven patients had systemic blood pressures in the hypertensive range recorded preoperatively (in 4 patients, the blood pressure was not mentioned). Six carotid endarterectomies were performed for carotid occlusion and 9 for stenosis. In 9 of the 15 cases, significant postoperative recovery of neurological function was said to have occurred. Intracerebral hemorrhage developed 2 hours to 6 days (average: 68 hours) postoperatively, and was heralded in 4 patients by vomiting and seizures. Fourteen patients had an intracerebral hemorrhage on the same side as the carotid surgery. The exception (reference 4, case 4) was a patient whose clinical deficit was limited to aphasia, and in another patient, bloody cerebrospinal fluid under high pressure and progressive central nervous system deficit indicated the presence of a cerebral hemorrhage. Blood pressure data during the postoperative period were not mentioned in 14 of the case analyses.

Hypertension was recognized as a complication of carotid endarterectomy by Lehv, Salzman, and Silen. These authors noted an elevation of mean systolic blood pressure of greater than 15 mm Hg in 15 of 27 patients undergoing unilateral carotid endarterectomy. In 5 of these 15 patients, the systolic and diastolic blood pressure was very high, and ranged from 195 to 250 mm Hg over 105 to 130 mm Hg. Seven of the 15 hypertensive patients developed neurological complications, and 1 developed severe neurological deterioration and died.

Hypertension following carotid endarterectomy may be related to disturbance of the carotid sinus reflex and is more common after bilateral carotid surgery. In the experience of one of the authors (LRC) 2 other patients have developed severe hypertension following carotid endarterectomy. One patient had a clinical picture consistent with hypertensive encephalopathy, and in the other patient, severe headache, vomiting and lethargy coincided with the blood pressure rise. In some patients with severe bilateral carotid stenosis, systolic hypertension with a wide pulse pressure may aid clinical compensation by increasing cerebral perfusion pressure. This type of blood pressure alteration may normalize after carotid endarterectomy.

In the present 2 patients, as in the previously reported cases, preoperative studies demonstrated severe stenosis of the ipsilateral carotid artery and a preoperative cerebral infarction. Diastolic hypertension has almost invariably been present preoperatively, and has been recognized in our patients postoperatively. The usual explanation given for the occurrence of cerebral hemorrhage following endarterectomy in patients with previous infarction relates bleeding to the sudden increase of blood flow into an ischemic area of abnormal brain tissue in which capillary and arterial permeability is abnormal. This leaves unexplained the frequent delay in the onset of hemorrhage (days to weeks) after endarterectomy, the occasional location of hemorrhage outside the infarcted zone, and the frequently noted improvement of cerebral function immediately after endarterectomy. The role of postoperative hypertension in the pathogenesis of this problem has not been emphasized. Postoperative blood pressure levels are frequently not cited in previous reports.

Some patients with hypertensive intracerebral hemorrhage are first noted to be hypertensive at the time of their stroke. While it is possible that asymptomatic hypertension may have long antedated the hemorrhage, the absence of retinal, renal or cardiac signs of hypertension in some patients does not support this possibility. New onset of hypertension may present cerebral capillaries with blood under high pressure and lead to capillary rupture and intracerebral hemorrhage. During the course of systemic
hypertension, arterial and arteriolar hypertrophy may subsequently develop, which protects the capillary bed from the high head of systemic pressure. Increased afterload leads to cardiac hypertrophy. A comparable situation occurs in the pulmonary vascular bed of patients with mitral stenosis. During the early course of progressive stenosis of the mitral valve, the pressure in the left atrium and pulmonary veins rises, presenting the pulmonary capillary bed with blood under high pressure. Hemoptysis may occur. Later in the course of this disease, pulmonary artery and arteriolar hypertrophy occurs, which protects the pulmonary capillary bed from a high pressure, but leads to increased pulmonary artery resistance and right ventricular strain.

The patient with an occluded or very stenotic carotid artery, particularly if longstanding, may have a protected low pressure cerebral vascular bed in the territory usually supplied by the ipsilateral carotid artery. This was observed in patient #1, whose carotid stump pressure, a reflection of collateral flow, was only 29 mm Hg. In fact, the retinal vessels on the side of a severe carotid stenosis may not show retinopathy, despite systemic hypertension. If the carotid stenosis is removed and systemic hypertension acutely develops, the previously protected cerebral capillary bed will be exposed to a high pressure. This is especially likely to happen in the ischemic zone near previously infarcted tissue, but may occur elsewhere.

Careful surveillance for elevated blood pressure in the postoperative period and prompt treatment if it develops, may be critical to the prevention of intracerebral hemorrhage. Furthermore, extreme caution should be exercised in performing endarterectomy in patients with pre-existing hypertension, especially if they have had a cerebral infarction. CT scanning now provides an additional method of identifying the patient with significant cerebral infarction in the absence of prominent clinical neurological abnormalities. The optimal interval to delay carotid endarterectomy in patients with cerebral infarction has not been delineated, but 4 to 5 weeks appears to be an insufficient period to give adequate protection against the development of this complication in the hypertensive patient.

References
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