

INTRACEREBRAL HEMORRHAGE AFTER CAROTID ENDARTERECTOMY ASSOCIATED WITH ASYMPTOMATIC PERIOPERATIVE CEREBRAL ISCHEMIA DETECTED BY CEREBRAL PERFUSION IMAGING: CASE REPORT

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Ogasawara K, Inoue T, Kobayashi M, Endo H, Fukuda T, Ogawa A. Intracerebral hemorrhage after carotid endarterectomy associated with asymptomatic perioperative cerebral ischemia detected by cerebral perfusion imaging: case report. *Surg Neurol* 2004;62:319–23.

BACKGROUND

Risk factors for intracerebral hemorrhage following carotid endarterectomy (CEA) include perioperative cerebral ischemia, postoperative cerebral hyperperfusion, and postoperative anticoagulation therapy, and at least 2 of these risk factors are typically present in the context of intracerebral hemorrhage (ICH).

CASE DESCRIPTION

A 75-year-old man with severe bilateral cervical internal carotid artery stenosis and a minor stroke resulting in left motor weakness underwent a right CEA. The operation was uneventful, and the patient did not experience new neurologic deficits upon recovery from anesthesia. Brain single photon emission computed tomography (SPECT) obtained immediately after CEA showed a perfusion defect in the right parietal lobe and absence of cerebral hyperperfusion. A computed tomography (CT) scan showed no new abnormal findings. Aspirin therapy was instituted postoperatively. On the second postoperative day, the patient experienced abrupt worsening of left hemiparesis, and subsequent CT imaging demonstrated a hematoma in the right parietal lobe. Cerebral hyperperfusion was absent on repeat SPECT.

CONCLUSION

Perioperative cerebral ischemia can result in intracerebral hemorrhage after CEA even in the absence of cerebral hyperperfusion and/or anticoagulation therapy. Further, cerebral perfusion imaging performed immediately after CEA is a useful modality for the identification of occult cerebral ischemia or hyperperfusion that may lead

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KEY WORDS

Carotid endarterectomy, intracerebral hemorrhage, cerebral ischemia, cerebral perfusion imaging.

Intracerebral hemorrhage (ICH) following carotid endarterectomy (CEA) occurs in fewer than 1% of cases but may account for up to one quarter of all postoperative complications [1,4,8–10,13] and has a high morbidity and mortality rate in community-based series [4,8–10,13]. Some characteristics of patients who have had an ICH after CEA have been identified and compared with those in controls. Cerebral hyperperfusion following CEA has been proposed as the main mechanism [1,3,4,9]. Perioperative cerebral ischemic events and postoperative anticoagulation therapy may be other important risk factors [3,4,9,12].

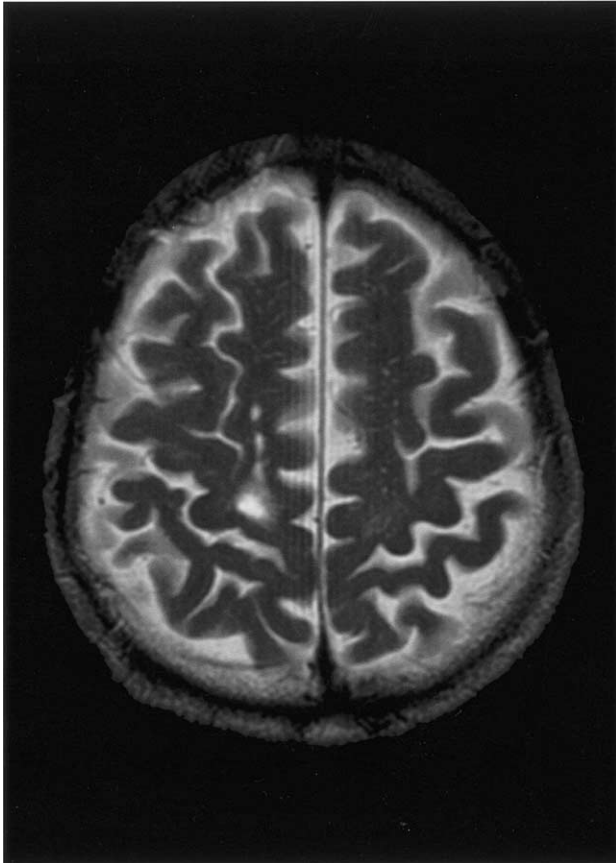
The following case describes a patient with asymptomatic perioperative cerebral ischemia as detected by cerebral perfusion imaging that developed an ICH after CEA. Further, this occurred in the absence of post-CEA hyperperfusion. Regional cerebral blood flow (CBF) was quantitatively measured using single photon emission computed tomography (SPECT) and [123I]N-isopropyl-p-iodoamphetamine [5].

CASE REPORT

A 75-year-old man with severe bilateral cervical internal carotid artery (ICA) stenosis was admitted

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1 Preoperative magnetic resonance scan showing small infarcts in the cerebral white matter in the posterior portion of the right frontal lobe.

for CEA after experiencing a minor stroke that resulted in left motor weakness. The patient's medical history was significant for hypertension and diabetes mellitus, and his medication profile included aspirin, perindopril, and glibenclamide. Upon admission, a neurologic examination revealed slight left hemiparesis. A magnetic resonance imaging scan demonstrated small infarcts in the cerebral white matter in the posterior portion of the right frontal lobe (Figure 1). Cerebral angiography revealed 85% stenosis of the right cervical ICA and 83% stenosis of the left cervical ICA. Brain SPECT demonstrated reduced CBF in the bilateral cerebral hemispheres (Figure 2 left). Each value was less than the mean—2SD of the control value (Table 1) [5].

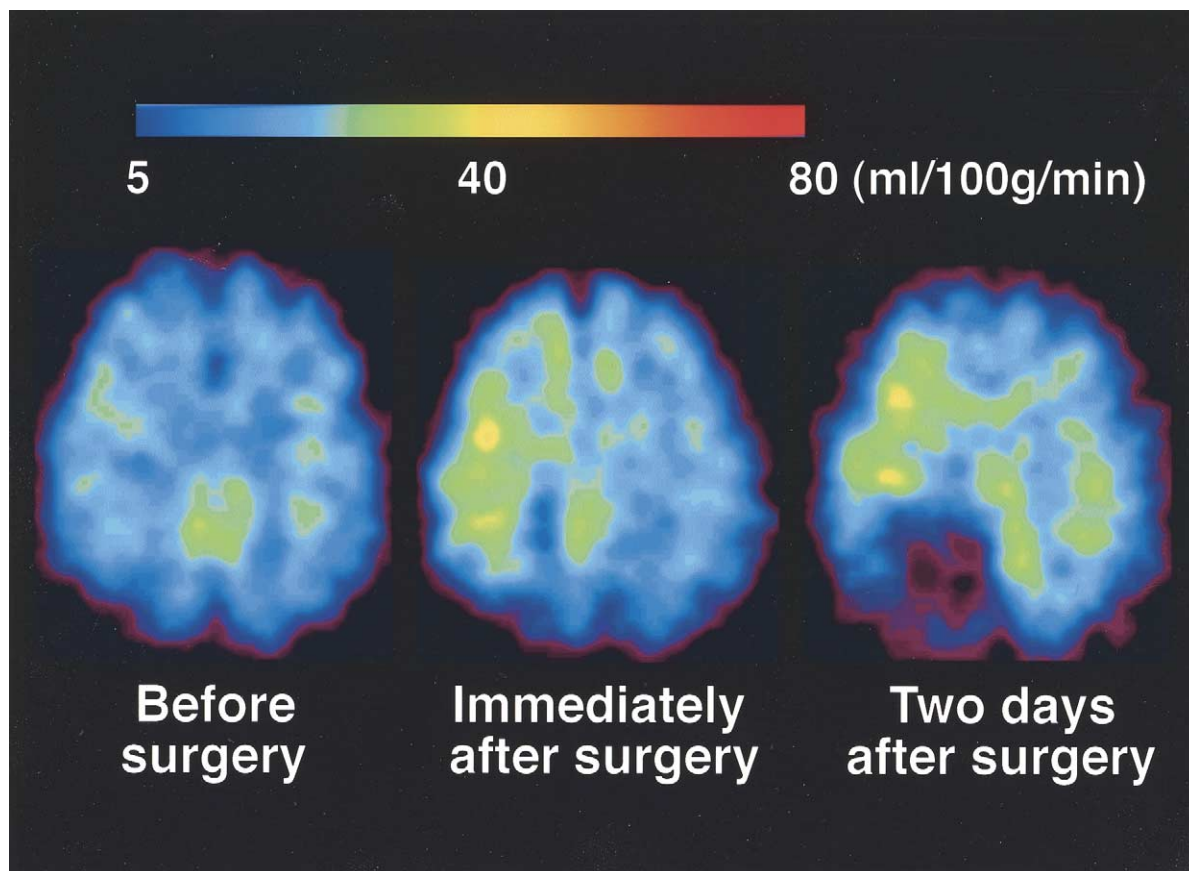
Sixty days after onset of the symptoms, the patient underwent a right CEA under general anesthesia. An intraluminal shunt was not used. A single bolus of heparin (5000 units) was given before the right ICA was cross-clamped, and the heparin was reversed using 30 mg of protamine sulfate after declamping of the ICA. The cross-clamping time was

28 min. Intraoperative electroencephalographic monitoring revealed no abnormal findings. The operation was uneventful, and the patient did not experience new neurologic deficits upon recovery from anesthesia. SPECT obtained immediately after CEA showed an increase in right cerebral hemisphere CBF (1.3-fold control values, Table 1) except in the right parietal lobe, where a wedge-shaped perfusion defect was noted (Figure 2 middle). Subsequent computed tomography (CT) scan showed no new abnormal findings. Magnetic resonance angiography revealed a widely patent right cervical ICA and no abnormal findings in the main trunks of the intracranial arteries. Although intensive control of blood pressure was not instituted after surgery, postoperative systolic blood pressure ranged from 120 to 140 mm Hg. Aspirin was reinitiated on the first postoperative day, but anticoagulation therapy was not instituted.

The patient had an uneventful course until late on the second postoperative day, when he experienced abrupt worsening of his left-side hemiparesis. A CT scan demonstrated a hematoma in the right parietal lobe, corresponding to the wedge-shaped perfusion defect noted on the postoperative SPECT study (Figure 3). Subsequent SPECT showed a large perfusion defect corresponding to the hematoma (Figure 2 right). The value of CBF in the right cerebral hemisphere (excluding the perfusion defect) was essentially unchanged from postoperative values (Table 1). A partial thromboplastin time obtained immediately after the occurrence of ICH was within normal limits. The subsequent clinical course was uneventful, and the patient experienced gradual improvement in his left-side motor function.

DISCUSSION

Most authors recommend institution of strict control of postoperative blood pressure to prevent hyperperfusion syndrome and ICH [2,9,11,13–15]. In our institution, we measured cerebral perfusion pre- and postoperatively using SPECT in all patients undergoing CEA and utilized antihypertensive medication postoperatively in patients diagnosed with cerebral hyperperfusion [6,7]. In our series of patients, post-CEA hyperperfusion was observed in 14%. While 21% of these patients developed cerebral hyperperfusion syndrome, none experienced intracerebral hemorrhage. Thus, cerebral perfusion imaging was performed immediately after CEA in the present case despite the absence of new neurologic deficits upon recovery from anesthesia.



2 Serial quantitative cerebral blood flow images using [¹²³I]N-isopropyl-p-iodoamphetamine SPECT. Preoperative SPECT showed reduced cerebral blood flow in the bilateral cerebral hemispheres (left). SPECT performed immediately after surgery showed recovery of perfusion in the right cerebral hemisphere except in the right parietal lobe, where a wedge-shaped perfusion defect was noted (middle). SPECT performed after worsening of left-sided hemiparesis on the second postoperative day demonstrated a large perfusion defect in the right parietal lobe and normal perfusion in the surrounding tissue (right).

In the present case, the lack of ischemic changes with cross-clamping on electroencephalography studies and a wedge-shaped perfusion defect on brain SPECT obtained immediately after surgery indicated the presence of perioperative cerebral ischemia likely secondary to cerebral embolism. The SPECT also demonstrated a 1.3-fold increase in ipsilateral cerebral hemisphere CBF when excluding the area of the perfusion defect. Cerebral hyperperfusion after CEA is defined as a CBF increase of

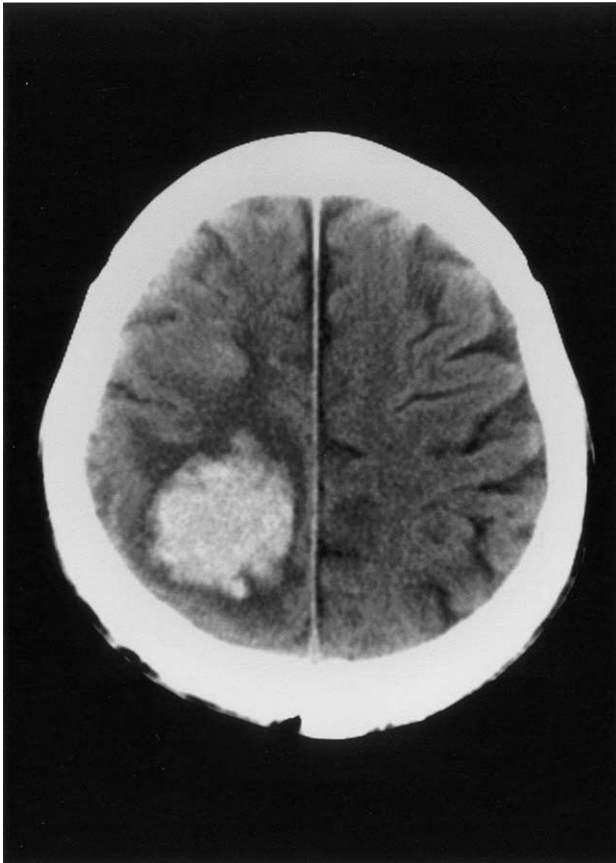
≥100% (i.e., a doubling) compared with preoperative values [14]; thus, this patient did not exhibit cerebral hyperperfusion.

Henderson et al reviewed the clinical and radiologic characteristics of patients with ICH following CEA and described several risk factors, including cerebral hyperperfusion, perioperative cerebral ischemia and anticoagulation therapy [4]. Further, in that series of patients, at least two of these risk factors were typically present in the context of ICH.

1 Pre- and Postoperative Cerebral Blood Flow* Measured Using Single Photon Emission Computed Tomography

	BEFORE SURGERY	IMMEDIATELY AFTER SURGERY	TWO DAYS AFTER SURGERY	CONTROL (MEAN ± SD)
Right cerebral hemisphere†	27.3	36.0	36.2	35.9 ± 4.4
Left cerebral hemisphere	28.5	29.6	31.4	

*: mL/100g/min; †: except for a perfusion defect.



3 A CT scan 2 days after surgery showing hemorrhage in the right parietal lobe.

In particular, ICH in the setting of a perioperative cerebral ischemic event was always compounded by the cerebral hyperperfusion and/or anticoagulation therapy. Based on these findings, Henderson et al conclude that an intraoperative cerebral ischemic event, in which damage to the microvasculature also occurs, could compound the cascade of events caused by cerebral hyperperfusion following reopening of a tight stenosis. Further, they speculated that the mechanism of ICH could not be solely because of hemorrhagic transformation of an infarct [4]. However, our patient experienced ICH after CEA in the absence of cerebral hyperperfusion or anticoagulation therapy. These findings suggest that perioperative cerebral ischemia caused by cerebral embolism can be a sole cause of ICH after CEA.

In the present case, perioperative cerebral ischemia occurring in the right parietal lobe was asymptomatic, and the diagnosis was only established by brain SPECT. Thus, this case demonstrates the utility of cerebral perfusion imaging in the early identification of occult cerebral ischemia or hyperperfusion that may lead to ICH after CEA.

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