JOURNAL OF NEUROSURGERY:
Case Lessons

J Neurosurg Case Lessons 6(17): CASE23476, 2023
DOI: 10.3171/CASE23476

Asymptomatic subarachnoid hemorrhage following carotid endarterectomy: illustrative case

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BACKGROUND Carotid endarterectomy (CEA) and carotid artery stenting are common surgical interventions for internal carotid artery stenosis. Cerebral hyperperfusion syndrome (CHS) is a well-known complication of both procedures that can lead to intracranial hemorrhage and worsen clinical outcomes. Here, the authors report a rare case of non-aneurysmal subarachnoid hemorrhage (SAH) following CEA and review the relevant literature.

OBSERVATIONS A 70-year-old woman with hypertension and diabetes presented with progressive visual loss in the right eye and was diagnosed with ocular ischemic syndrome. Imaging revealed severe right cervical carotid artery stenosis. CEA was performed with no complications. Postoperatively, the patient's blood pressure was tightly controlled, with no evidence of CHS. However, an asymptomatic SAH was detected on postoperative day 7. Careful observation and blood pressure control were maintained. Since follow-up magnetic resonance imaging (MRI) showed no enlarging of the SAH and the patient was asymptomatic, she was discharged on postoperative day 15 with a modified Rankin scale score of 0.

LESSONS This case highlights the potential occurrence of non-aneurysmal SAH as a rare complication of CEA, even in asymptomatic patients. Repeated postoperative MRI is necessary to detect such complications. It is crucial to carefully control blood pressure after CEA regardless of symptoms.

https://thejns.org/doi/abs/10.3171/CASE23476

KEYWORDS carotid endarterectomy; subarachnoid hemorrhage; cerebral hyperperfusion syndrome

Carotid endarterectomy (CEA) and carotid artery stenting (CAS) are common surgical treatments for internal carotid artery stenosis (ICS). Cerebral hyperperfusion syndrome (CHS) is a well-known postoperative complication common to both procedures, with reported incidences of 1.9% and 1.1% in patients undergoing CEA and CAS, respectively.[1] Intracerebral hemorrhage (ICH), which occurs in 44% of CHS cases, requires special attention, as it can significantly worsen clinical outcomes.[2] The incidence of ICH after CEA is reported to be between 0.2% and 0.6%, whereas reports of subarachnoid hemorrhage (SAH) are extremely rare.[1,3–6] Herein, we report a case of non-aneurysmal SAH that occurred after CEA without any symptoms and review the literature.

Illustrative Case

History and Examination

A 70-year-old woman who had hypertension, diabetes, and no history of coronary artery disease or smoking had presented to a local ophthalmologist with progressive visual loss in the right eye over several months. She had been diagnosed with right ocular ischemic syndrome and was referred to our hospital for further

ABBREVIATIONS ASL = arterial spin labeling; CAS = carotid artery stenting; CBF = cerebral blood flow; CEA = carotid endarterectomy; CHS = cerebral hyperperfusion syndrome; CSHI = cortical-sulcal hyperintensity sign; CT = computed tomography; CTA = computed tomography angiography; DSA = digital subtraction angiography; FLAIR = fluid-attenuated inversion recovery; ICH = intracerebral hemorrhage; ICS = internal carotid artery stenosis; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; SAH = subarachnoid hemorrhage; SNR = signal-to-noise ratio; SSEP = somatosensory evoked potential; TCD = transcranial Doppler ultrasonography.

INCLUDE WHEN CITING Published October 23, 2023; DOI: 10.3171/CASE23476.
SUBMITTED August 27, 2023. **ACCEPTED** September 27, 2023.

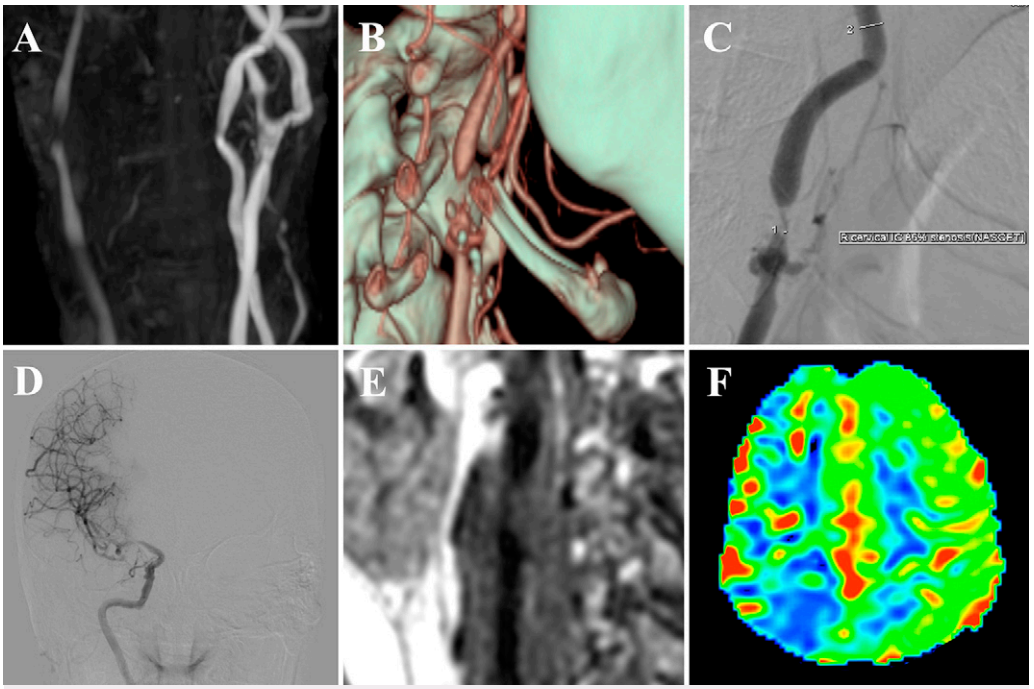

FIG. 1. Preoperative imaging. Magnetic resonance angiography (MRA) on admission showing a decreased signal intensity in the right carotid artery, suggesting a stenotic lesion (**A**). Computed tomography angiography (**B**) and digital subtraction angiography (**C**) demonstrate severe stenosis of the right cervical carotid artery. Intracranial aneurysms were not observed on cerebral angiography (**D**). The plaque is depicted as a partial high-signal intensity on T1-weighted black-blood imaging, suggesting vulnerability (**E**). Arterial spin labeling (ASL) showing decreased cerebral blood flow in the right middle cerebral artery territory (**F**).

evaluation. On admission, her right best corrected visual acuity was counting fingers; however, no other neurological deficits were observed.

Magnetic resonance imaging (MRI) showed no ischemic changes in the brain; however, magnetic resonance angiography (MRA) revealed a stenotic lesion in the right cervical carotid artery (Fig. 1A). Digital subtraction angiography (DSA) was performed for further evaluation. Right carotid angiography revealed severe stenosis (North American Symptomatic Carotid Endarterectomy Trial method, 86%) of the right cervical carotid artery (Fig. 1B and C). Cerebral angiography revealed no intracranial aneurysms (Fig. 1D). The plaque was depicted as an area of partially high-signal intensity on T1-weighted black-blood images, suggesting vulnerability (Fig. 1E). Decreased cerebral blood flow (CBF) in the right middle cerebral artery territory was observed on arterial spin labeling (ASL), compared with the contralateral side (Fig. 1F).

Surgical Procedure

Antiplatelet therapy with clopidogrel 75 mg was initiated, and CEA was performed with the patient under general anesthesia and somatosensory evoked potential (SSEP) monitoring to improve ocular ischemic syndrome and prevent ischemic stroke. After exposing the carotid artery, an internal shunt was placed with temporary occlusion for 14 minutes, and the atheromatous plaque was peeled off. The plaque was yellowish-white in appearance. No major changes in the SSEPs were reported. Arterial suturing was performed using a Hemashield Finesse (Getinge AB). No adverse events or unanticipated events were observed.

Postoperative Course

Because the patient had severe stenotic lesions and hypertension, she was considered to be at a high risk for postoperative CHS. We continued intubation and sedation management in the intensive care unit and maintained systolic blood pressure at $<$130 mm Hg. MRI on postoperative day 1 revealed no acute cerebral infarction (Fig. 2A), and the stenotic lesion had improved on MRA (Fig. 2B). No global hyperperfusion was observed on ASL (Fig. 2C), sedation was discontinued, and the patient was extubated. The patient's postoperative course was uneventful, and her right visual acuity improved. No evidence of CHS, including seizures or hemispheric symptoms, was observed during the postoperative course. Follow-up ASL was performed on postoperative day 4; however, no hyperperfusion was observed. Postoperative systolic blood pressure was generally $<$130 mm Hg.

Fluid-attenuated inversion recovery (FLAIR) MRI and computed tomography (CT) performed on postoperative day 7 revealed SAH on the side ipsilateral to the CEA (Fig. 2D and E). We performed high-resolution CT angiography (CTA) immediately after SAH detection for vascular assessment, which showed no evidence of intracranial aneurysms. ASL performed on the same day also showed no global hyperperfusion (Fig. 2F). Careful observation and antihypertensive treatment were continued. As the patient was asymptomatic and the follow-up MRI showed no enlargement of the SAH, she was discharged on postoperative day 15 with a modified Rankin scale score of 0.

Patient Informed Consent

The necessary patient informed consent was obtained in this study.

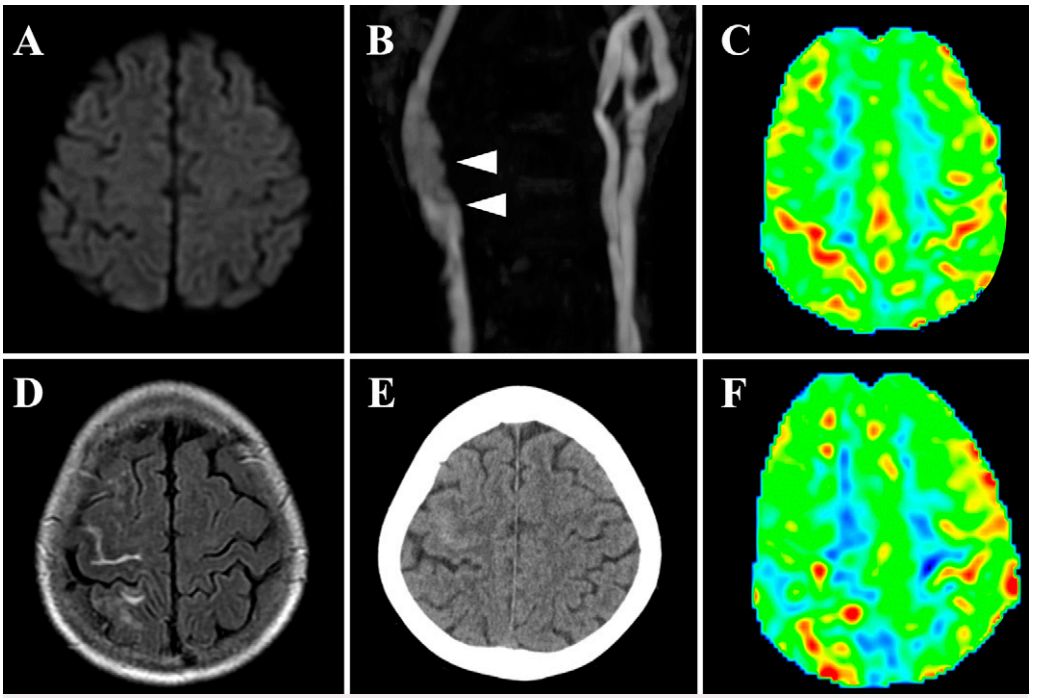

FIG. 2. Postoperative imaging. Diffusion-weighted imaging on postoperative day 1 revealed no acute cerebral infarction (**A**), and the stenotic lesion (*white arrowheads*, **B**) had improved on MRA. ASL on postoperative day 1 showed no hyperperfusion in the right hemisphere (**C**). Fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging (**D**) and computed tomography (**E**) on postoperative day 7 showed a thin subarachnoid hemorrhage in the right hemisphere. No global hyperperfusion was observed on ASL on the same day (**F**).

Discussion

Observations

The present case was incidentally diagnosed as an asymptomatic SAH on MRI without any symptoms of CHS after CEA. In general, intracranial hemorrhage, a complication of hyperperfusion syndrome after CEA/CAS, often leads to serious outcomes. The patient was asymptomatic, and her clinical outcomes were favorable. Reports of CEA-related non-aneurysmal SAH are very rare. In a previous report by Ogasawara et al.,[1] no CEA-related SAHs were observed among 1596 cases. We found only four previous reports of similar cases (Table 1).[3–6] All cases were symptomatic, and two cases had poor outcomes with impaired consciousness or seizures. In addition, all four cases were accompanied by CHS. In three cases, the time of detection was approximately 1 week after CEA, which coincided with the peak of CHS.[1] Treatment mainly comprised antihypertensive therapy, and anticonvulsants or sedation were administered depending on the case. The present case was characterized by an asymptomatic presentation without preceding hyperperfusion syndrome.

The mechanisms of ICH after CEA/CAS are suggested to be impaired CBF autoregulation due to the sudden improvement in CBF postoperatively or postoperative hypertension.[1] With regard to postoperative hemorrhagic complications, ICH is the most common after CEA, whereas SAH occurs in 10%–20% of cases after CAS.[1,2,7] Intra- and postoperative microembolism specific to CAS has been suggested as the cause of SAH.[1] It has been hypothesized that endothelial damage after reperfusion following embolization may cause cortical small artery rupture and hemorrhage. Furthermore, disruption of the blood–brain barrier by the osmotic pressure exerted by contrast agents and decreased CBF autoregulation are also considered mechanisms.[8,9] Although our case was a CEA-related SAH, vulnerable plaques may have been dispersed during vascular exposure or shunt insertion during surgery. Additionally, contrast agents used

TABLE 1. Previous cases of non-aneurysmal subarachnoid hemorrhage after carotid endarterectomy

Authors & Year	Age (yrs)/Sex	Symptoms	CHS	Time of Detection	Treatment	mRS Score
Dalton, 1992[4]	62/F	Headache	+	POD 5	Antihypertensive therapy	1
Bodenant et al., 2010[5]	74/M	Seizure, paralysis	+	POD 9	AED, antihypertensive therapy	4
Thanabalasundaram et al., 2013[6]	66/M	Coma, seizure	+	POD 6	AED, antihypertensive therapy	3
Ishida et al., 2019[3]	50/M	Disquiet	+	POD 1	Sedation, antihypertensive therapy	1
Present case	70/M	Asymptomatic	–	POD 7	Sedation, antihypertensive therapy	0

AED = antiepileptic drug; CHS = cerebral hyperperfusion syndrome; mRS = modified Rankin Scale; POD = postoperative day.

before surgery may have exacerbated the vascular endothelial damage. We speculate that the background is similar to that of CAS-related SAH. Although we did not perform transcranial Doppler ultrasonography (TCD) in this case, the detection of microembolic signals on postoperative TCD may be useful not only for the risk assessment of postoperative cerebral infarction[10] but also for the detection of hyperperfusion syndrome or ICH/SAH.

With regard to patch use, Ishida et al.[3] reported a case of CHS similar to ours. They suggested that the use of a patch contributed to local increases in CBF and SAH. The use of patches should be carefully considered in patients with severe stenosis and decreased CBF.

High postoperative FLAIR signals in the sulcus have also been reported after bypass surgery and have been described as the de novo ivy sign[11] or the cortical-sulcal hyperintensity sign (CSHI).[12] These findings are considered signs of local hyperperfusion/hyperemia after bypass surgery, which clearly differ from those of SAH. In our case, CT revealed a high-density area corresponding to a high FLAIR signal, suggesting SAH. However, the de novo ivy sign and CSHI should be considered even in CEA cases with high FLAIR signals in the sulcus.

In our case, ASL was used for preoperative and postoperative CBF evaluation. ASL has the advantage that it can be easily performed in a short time without the use of contrast media or radioisotopes. However, the quality of ASL images can be limited by their low signal-to-noise ratio (SNR) and by several types of artifacts that are important to know.[13] Although no global hyperperfusion was observed on ASL, it is possible that asymptomatic focal hyperperfusion/hyperemia was present in this case. In patients at high risk for CHS, considering imaging studies that are quantitative and have higher SNR, such as single-photon emission CT or positron emission tomography, is necessary. In the present case, asymptomatic SAH was diagnosed on the basis of multiple follow-up MRI scans. FLAIR MRI has a sensitivity of almost 100% for the detection of acute SAH, and the detection of postoperative SAH is not difficult.[14] We also performed high-resolution CTA to exclude aneurysmal SAH. DSA should be considered a first-line diagnostic technique instead of the less sensitive CTA, especially for small aneurysms.[15] However, no intracranial aneurysms were confirmed in the preoperative DSA. In addition, no head trauma or signs of infection were observed during hospitalization; therefore, we did not suspect a traumatic or mycotic aneurysm. For these reasons, we determined that high-resolution CTA was sufficient to rule out aneurysmal SAH.

Lessons

Subarachnoid hemorrhage should be considered as a complication of CEA, even in asymptomatic patients. Repeated follow-up MRI may be needed for the early diagnosis of SAH, and attention should be paid not only to ischemic lesions but also to hemorrhagic lesions, including SAH, after CEA. It is crucial to carefully control blood pressure after CEA regardless of symptoms. Our patient had a favorable outcome, probably because of prolonged postoperative sedation and long-term blood pressure management.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Nemoto, Yamashita, Kurita. Acquisition of data: Nemoto, Yanagawa, Torii, Kiyomoto, Tanaka, Kono. Analysis and interpretation of data: Nemoto. Drafting of the article: Maeda, Nemoto, Kurita. Critically revising the article: Maeda, Yamashita, Kono, Kurita. Reviewed submitted version of the manuscript: Maeda, Nemoto, Yanagawa, Kono, Kurita. Approved the final version of the manuscript on behalf of all authors: Maeda. Administrative/technical/material support: Nemoto, Harada. Study supervision: Sato, Hatayama, Kurita.

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