Case Report

Delayed cerebral infarct following anterior cervical diskectomy and fusion

Christopher S. Graffeo, Ross C. Puffer, Eelco F. M. Wijdicks[1], William E. Krauss

Departments of Neurologic Surgery and [1]Neurology, Mayo Clinic, Rochester, Minnesota, USA

E-mail: Christopher S. Graffeo - graffeo@gmail.com; Ross C. Puffer - Puffer.Ross@Mayo.edu; Eelco F. M. Wijdicks - Wijdicks.Eelco@Mayo.edu;
*William E. Krauss - Krauss.William@Mayo.edu
*Corresponding author

Abstract

Background: Ischemic stroke following anterior cervical diskectomy and fusion (ACDF) is an exceedingly rare complication. There are only three previous cases focusing on this problem in the literature; here, we present the fourth case.

Case Description: A patient, cared for at an outside institution, developed a delayed ischemic stroke 3 days following an ACDF. This complication was attributed to carotid manipulation precipitating vascular injury in the setting of multiple comorbid vascular and coagulopathic risk factors, including previously undiagnosed carotid atherosclerosis, a prior history of pulmonary embolus requiring Warfarin anticoagulation (held perioperatively), acute dehydration, and atrial fibrillation.

Conclusions: This case demonstrates the importance of focused history and examination in appropriate patients prior to ACDF, with special consideration given to the significance of age, comorbidities including coagulopathy and arrhythmia, and potential underlying vascular disease as markers for increased risk of perioperative thrombotic stroke associated with carotid manipulation. Patients at higher risk warrant comprehensive preoperative assessment, including medical evaluation, carotid imaging, and consideration for alternative surgical approaches.

Key Words: Anterior cervical diskectomy and fusion, carotid disease, cerebral infarct, screening

BACKGROUND

Cerebral infarct due to carotid injury is a very rare complication of anterior cervical diskectomy and fusion (ACDF) surgery; there are only three prior cases reported in the literature. In each of these cases, the patient awakened from anesthesia with a new neurologic deficit, having sustained a cerebral infarct. Subsequent investigations revealed a wide range of underlying stroke etiologies, including prolonged retraction compressing the carotid artery, a congenitally abnormal Circle of Willis, and hypotension in the setting of atherosclerosis.[1,2,9,11] We report the case of a 61-year-old man with a history of pulmonary embolism (PE) and atrial fibrillation, who developed delayed stroke

3 days following an ACDF, most likely attributable to severe but asymptomatic and undiagnosed carotid atherosclerosis, vessel manipulation, coagulopathy, and acute dehydration, which collectively precipitated a large watershed infarct.

CASE DESCRIPTION

A 61-year-old man with a history of PE fibrillation requiring warfarin anticoagulation and hypertension presented to an outside hospital with 8 months of progressive neck pain and right upper extremity pain, which had worsened to the point of awakening him at night.. Multiple attempts at physical therapy, chiropractic manipulation, and other nonoperative modalities had failed to relieve this patient's pain. His neurologic examination demonstrated 3/5 strength in the right triceps and pectoralis major, with obvious muscle atrophy; there were no signs of myelopathy, hyperreflexia, pathologic reflexes, or gait disturbance. Records from this community practice—including but not limited to preoperative assessment, imaging studies, and operative report—were reviewed extensively following his ultimate transfer to our facility and are presented here in a retrospective review.

Magnetic resonance imaging (MRI) of the cervical spine demonstrated a moderate near-midline left herniation of the C5-C6 intervertebral disk compressing the cervical cord, as well as a large right paramedian herniation of the C6-C7 intervertebral disk compressing the right C7 nerve root [Figures 1a and b]. Review of records from the outside institution revealed the following preoperative thought process: Although the C6-C7 paramedian disk herniation could potentially have been decompressed from either an anterior or posterior approach, large central disk components were noted at both levels, and an anterior approach would be required to access the C5-C6 midline disk herniation. Correspondingly, the patient was scheduled for an elective two-level ACDF at the outside hospital; no further preoperative assessment was conducted at that time.

Warfarin anticoagulation was held for 10 days preoperatively. Surgery included a routine right-sided exposure to perform a two-level ACDF at C5-C6 and C6-C7, which was completed using the Smith–Robinson technique. Notably, a self-retaining radiolucent retractor system was placed without difficulty and was periodically relaxed throughout the case; a monitored endotracheal tube was also used as surveillance for recurrent laryngeal nerve injury, and no abnormalities were noted.

There were no intraoperative complications, and careful inspection of the carotid artery, jugular vein, and surrounding structures prior to closure revealed no abnormalities. The wound was closed in the normal anatomic layers, and the patient awoke from general anesthesia without neurologic deficit and recovered uneventfully. Postoperative radiographs demonstrated normal alignment of the cervical spine with appropriate positioning of the fusion construct [Figure 2]. At discharge, the patient's right upper extremity pain was completely resolved, and he reported no dysphagia, new weakness, numbness, or pain. The patient was instructed to continue holding warfarin until postoperative day 7.

Three days after the discharge, the patient experienced multiple episodes of acute-onset large-volume diarrhea and vomiting overnight. The following morning, he had difficulty using his left arm and leg, with associated slurring of his speech. He was urgently transported to a local emergency department, where computed tomography (CT) of the head demonstrated subtle early ischemic changes involving the right hemisphere, including the insula and deep white matter of the frontal lobe (not shown). CT angiogram (CTA) of the head and neck revealed near-complete occlusion of the right internal carotid artery and greater than 50% stenosis of the left internal carotid artery, with otherwise normal arterial anatomy without evidence of dissection [Figure 3]. The patient was not deemed a candidate for lytic therapy, and he was transported to our facility in stable condition for intensive care.

On arrival, National Institute of Health Stroke Scale score was 14 with right gaze preference and left homonymous hemianopsia, lower facial droop, left hemiplegia, sensory loss, and hemineglect. The surgical site was clean, dry, and intact, with no signs concerning for hematoma, infection, mass, or other compressive process. MRI/MRA of the head and neck showed greater than 50% occlusion of the left internal carotid artery without evidence of dissection, complete occlusion of the right internal carotid artery at its origin with slow reconstitution at the petrous level, slow intracranial flow distally indicating insufficient collateral flow, and extensive diffusion restriction and fluid-attenuated inversion recovery changes in a typical watershed distribution throughout the right hemisphere [Figures 4 and 5].

Electrocardiogram was noteworthy for new-onset atrial fibrillation, with ventricular rate to 130, which was
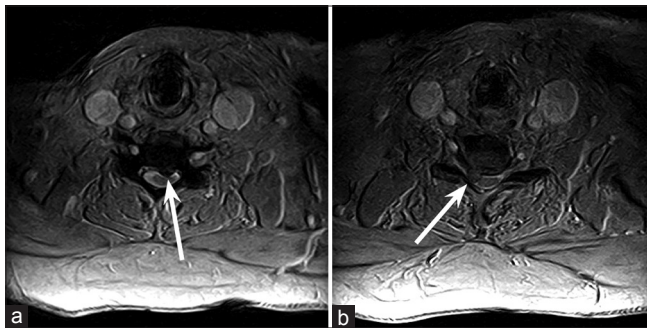

Figure 1: T2-weighted axial magnetic resonance imaging of the cervical spine through the C5-C6 and C6-C7 levels, demonstrating a moderate near-midline left herniation of the C5-C6 intervertebral disk compressing the cervical cord (a), as well as a large right paramedian herniation of the C6-C7 intervertebral disk compressing the right C7 nerve root (b)

Figure 2: Sagittal non-contrast computed tomography of the cervical spine, showing appropriate placement of the fusion construct at the C5-C7 vertebral levels
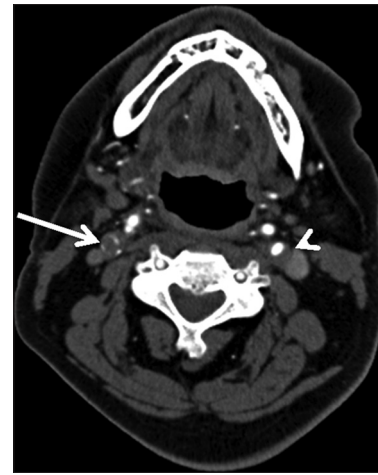

Figure 3: Axial computed tomography angiogram of the neck demonstrating near-complete occlusion of the right internal carotid artery (arrow), as well as greater than 50% stenosis of the left internal carotid artery (arrowhead)
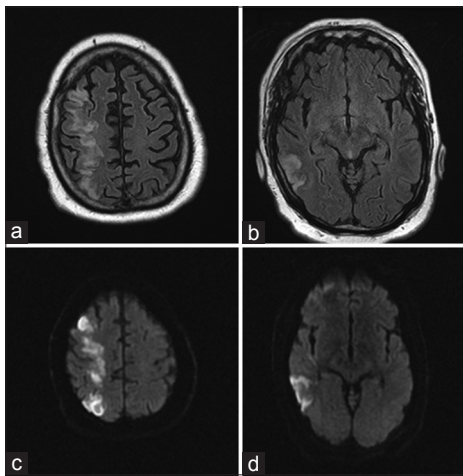

Figure 4: T2 fluid-attenuated inversion recovery (a, b) and diffusion weighted (c, d) images demonstrating multiple right hemispheric infarcts in a watershed pattern, involving frontal, parietal, and temporal territories of the anterior circulation
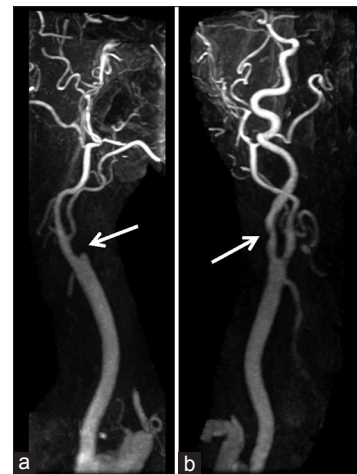

Figure 5: Time-of-flight magnetic resonance angiogram of the neck demonstrating absent flow through the right internal carotid artery at its origin (a), as well as greater than 50% stenosis of the left internal carotid artery with prominent irregularity (b)

medically managed using diltiazem and metoprolol. Echocardiogram showed no evidence of cardiac thrombus or patent foramen ovale. Ultrasound of the lower extremities revealed an acute-on-chronic deep venous thrombosis of the right popliteal vein. Follow-up imaging with CTA of the chest showed multiple acute emboli, with early pulmonary infarction in the periphery of the right mid-lung, without evidence of right heart strain. Given the contraindication to therapeutic anticoagulation, an inferior vena cava filter was placed. Hematology and coagulation studies showed a normal international normalized ratio, partial thromboplastin time, and bleeding time, with no evidence of an underlying heritable coagulopathy.

The patient was observed overnight in the neurosciences intensive care unit, where he remained clinically and neurologically stable. Warfarin was restarted on day 2. The patient was ultimately transferred to the stroke neurology service, followed by acute inpatient rehabilitation. At the time of discharge, the patient had persistent left facial droop, left arm paresis with minimal activation, and left hip flexion antigravity without distal activation.

DISCUSSION

In this case report, we present a perioperative ischemic stroke 4 days following an uncomplicated ACDF in a patient with multiple risk factors, including bilateral carotid stenosis, resulting in arterial thrombosis and ischemia ipsilateral to the surgical approach. This is the first case of such a complication occurring beyond the immediate perioperative period, and only the fifth reported cerebral infarct attributable to any etiology after ACDF without corpectomy.[1,2,9,11]

The patient history demonstrated many risk factors for perioperative stroke of various etiologies, including hypertension, male gender, new onset atrial fibrillation, and history of unprovoked PE requiring anticoagulation. In addition, his warfarin was stopped perioperatively, and during the early postoperative period, he developed dehydration due to systemic illness. We hypothesize that, in this patient with significant underlying atherosclerotic disease, surgical retraction sufficiently disturbed the carotid plaque to create turbulent flow and local hemostasis. In the setting of acute dehydration, compounded by an untreated hypercoagulable state, flow through the carotid artery was critically diminished, precipitating thrombosis and right hemispheric watershed infarct.

Although it is difficult to say with certainty that surgical retraction contributed to this complication, we find the circumstantial evidence compelling. The proposed mechanism finds further support from physiologic studies suggesting that prolonged retraction of an atherosclerotic carotid artery may induce flow-limiting stenosis or precipitate arterial embolic events, via disruption or dislodgement of the plaque by the retractor system.[8] In addition, the patient was also observed to have significant contralateral carotid disease, yet no left-sided infarcts were observed, implicating the surgical intervention, and arguing against a strictly hypotensive etiology.

From the unique clinical history, a number of alternative mechanisms can be imagined, invoking the patient's history of new atrial fibrillation and previous unprovoked PE. Cessation of anticoagulation in nonvalvular atrial fibrillation is a rare, but not an impossible cause of perioperative stroke, with an estimated incidence of 2 events in 10000 procedures.[6] Indeed, an embolic source could underlie such an infarct; however, the watershed infarct pattern argues strongly in favor of a thrombotic phenomenon. In addition, no evidence of cardiac thrombus or patent foramen ovale was observed on echocardiogram, making the likelihood of a cardiac or paradoxical source less likely—particularly in the setting of new arrhythmia, and the more probable thrombotic mechanism of surgical manipulation coupled to dehydration.

Similarly, in the setting of a potential iatrogenic carotid injury, arterial dissection precipitating thrombosis warrants consideration although our literature review found this to be an even rarer complication of ACDF than carotid thrombosis.[4,7] Nevertheless, in the present case, this possibility was ruled out on CTA and MRA during the acute stroke work-up; however, it should remain an important item on the differential in parallel cases.

In retrospect, some consideration is due to whether the elective procedure at the heart of the present case was warranted, or if the preoperative risk factors for thrombosis and other related complications may have outweighed the potential benefit. With knowledge of the outcome in mind, one can assemble a compelling list of arguments in opposition to the intervention, or at minimum, a more thorough preoperative assessment: a history of coagulopathy and clinically significant thrombotic events requiring anticoagulation, substantial atherosclerotic burden, and other related medical comorbidities. In parallel, our review of the literature affirms the scarcity of thrombotic complications attributable to arterial injury after ACDF; taken together with the high incidence of asymptomatic carotid stenosis—up to 3.1% in the general population—and the frequency with which ACDF is performed, it is difficult to compellingly argue against neurosurgical intervention in patients with symptomatic radiculopathy or myelopathy and vascular risk factors alone. Taking these considerations in concert, a more extensive preoperative work-up may provide the optimal balance in high risk patients in need of symptomatic relief.

Central to our recommendations is an individually-tailored patient care plan; however, certain key elements should be weighed in each assessment. These may include consultation with Medicine or Cardiology to better define preoperative risk factors, or carotid artery imaging in appropriately selected individuals.

The question of an ideal surgical approach in the present case is impossible to answer without adequate preoperative vascular imaging; however, based on the postoperative outcome, we can offer some limited insight regarding the viability of either a contralateral anterior or a posterior approach. Postoperative vascular imaging suggests that the patient's severe carotid disease was in fact bilateral, even though potentially less pronounced on the left, highlighting one potentially safer alternative surgical corridor [Figures 3 and 5]. However, given the patient's risk factors for thrombosis, complete avoidance of an anterior operation may have been advised, had these abnormalities been detected. In parallel, although the central C5-C6 disk fragment would not have been accessible from an anterior approach, the patient's symptoms localized more prominently to the C6-C7 pathology; correspondingly, a C5–C7 laminectomy with a right C6-C7 foraminotomy, and resection of the herniated fragment may have provided yet another alternative, and one with the capacity to treat the patient's symptoms, decompress the stenosis at both levels, and avoid carotid manipulation altogether.

CONCLUSION

The implications of this case are challenging to interpret, particularly in the light of the limited evidence regarding carotid injury following ACDF. On the one hand, universal screening for carotid disease with preoperative imaging would be highly cost-ineffective, particularly in

an exceedingly low-incidence entity. However, the present case and related reports highlight the impoverished knowledge base surrounding cerebral ischemia as a potential complication of ACDF, inviting speculation that the true incidence may be underestimated by current reports—particularly in the delayed setting.

With this possibility in mind, we recommend a focused review of the medical history for atherosclerosis risk factors in all patients prior to ACDF, with a low threshold for thorough preoperative assessment in patients with significant vascular risk factors and consideration for alternative surgical approaches. In cases with indeterminate histories, physical examination with carotid auscultation may provide an essential data point informing the decision to image, although the low cost and risk of carotid ultrasound should bias toward imaging in unclear circumstances. Similarly, a role may be defined for postoperative carotid imaging to confirm vessel patency and laminar flow among patients with known atherosclerotic burden. In appropriately selected patients, this could open the door to intervene before complications arise.

Finally, we believe that our case argues compellingly in favor of early reinitiation of anticoagulation or antiplatelet therapy, which in the case of an elective ACDF that was not complicated by significant intraoperative hemorrhage may be started within 48 hours of surgery, potentially within the first 12 hours following the operation in patients at highest risk of thrombotic complications. At present, clinical evidence regarding resumption of postoperative anticoagulation is heterogeneous, and the lack of specific information on post-ACDF populations makes it challenging to generalize the limited evidence that does exist, in particular given the rarity of the complication in question.[3,5,6,10] Notwithstanding, thoughtful management is requisite, and the full range of potential anticoagulation agents and schemes—ranging from early institution of prophylactic or therapeutic modalities, to bridging therapies, to delayed resumption of preoperative regimens, and other strategies—are likely to play an important role in individualized patient care.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Chin KR, Seale J, Butron V, Cumming V. Postoperative cervical haematoma complicated by ipsilateral carotid thrombosis and aphasia after anterior cervical fusion: A case report. Case Rep Med 2013;2013:590639.
2. Drummond JC, Englander RN, Gallo CJ. Cerebral ischemia as an apparent complication of anterior cervical discectomy in a patient with an incomplete circle of Willis. Anesth Analg 2006;102:896-9.
3. Guha D, Coyne S, Macdonald RL. Timing of the resumption of antithrombotic agents following surgical evacuation of chronic subdural hematomas: A retrospective cohort study. J Neurosurg 2016;124:750-9.
4. Halbach VV, Higashida RT, Dowd CF, Fraser KW, Smith TP, Teitelbaum GP, et al. Endovascular treatment of vertebral artery dissections and pseudoaneurysms. J Neurosurg 1993;79:183-91.
5. Hirsh J, Dalen J, Anderson DR, Poller L, Bussey H, Ansell J, et al. Oral anticoagulants: Mechanism of action, clinical effectiveness, and optimal therapeutic range. Chest 2001;119 (1 Suppl):8S-21S.
6. Kearon C, Hirsh J. Management of anticoagulation before and after elective surgery. N Engl J Med 1997;336:1506-11.
7. Loret JE, Francois P, Papagiannaki C, Cottier JP, Terrier LM, Zemmoura I. Internal carotid artery dissection after anterior cervical disc replacement: First case report and literature review of vascular complications of the approach. Eur J Orthop Surg Traumatol 2013;23:107-10.
8. Pollard ME, Little PW. Changes in carotid artery blood flow during anterior cervical spine surgery. Spine 2002;27:152-5.
9. Radhakrishnan M, Bansal S, Srihari GS, Sampath S, Rao GS. Perioperative stroke following anterior cervical discectomy. Br J Neurosurg 2010;24:592-4.
10. Uribe J, Moza K, Jimenez O, Green B, Levi AD. Delayed postoperative spinal epidural hematomas. Spine J 2003;3:125-9.
11. Yeh YC, Sun WZ, Lin CP, Hui CK, Huang IR, Lee TS. Prolonged retraction on the normal common carotid artery induced lethal stroke after cervical spine surgery. Spine 2004;29:E431-4.