Cervical Spinal Cord Infarction After Cervical Spine Decompressive Surgery

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Key words
- Cervical
- Decompression surgery
- Hypotension
- Imaging
- Infarction
- Spine

Abbreviations and Acronyms
CSF: Cerebrospinal fluid
CT: Computed tomography
MRA: Magnetic resonance angiography
SSEP: Somatosensory evoked potentials

OBJECTIVE: To report five patients who underwent cervical decompressive surgeries and developed persistent postoperative neurologic deficits compatible with spinal cord infarctions and evaluate causes for these rare complications.

METHODS: The clinical courses and imaging studies of five patients were retrospectively analyzed. Imaging findings, types of surgeries, vascular compromise or risk factors, hypotensive episodes, intraoperative somatosensory evoked potentials, concomitant brain infarctions, and clinical degree and radiographic extent of spinal cord infarction were studied. The presence of spinal cord infarction was determined by clinical course and imaging evaluation.

RESULTS: All five patients had antecedent cervical cord region vascular compromise or generalized vascular risk factors. Four patients developed hypotensive episodes, two intraoperatively and two postoperatively. None of the four patients with hypotensive episodes had imaging or clinical evidence of concomitant brain infarctions.

CONCLUSIONS: Neuroimaging evaluation of spinal cord infarction after decompressive surgery is done to exclude spinal cord compression, to ensure adequate surgical decompression, and to confirm infarction by imaging. Antecedent, unrecognized preoperative vascular compromise may be a significant contributor to spinal cord infarction by itself or in combination with hypotension.

INTRODUCTION

Although new-onset postoperative neurologic deficits after spinal surgery are uncommon, the consequences can be catastrophic for patients. Causes of such neurologic deterioration include direct surgical trauma or manipulation, vascular or perfusion compromise, inadequate decompression, spinal cord compression by epidural or subdural hematomas, or displacement of surgical constructs (3, 6, 7). In one comprehensive report, postoperative epidural hematomas and inadequate decompression were responsible for most new deficits (6). Postoperative cervical cord infarction is a rare case. Neuroimaging is pivotal to exclude reversible causes for such neurologic deficits and to confirm the presence of a spinal cord infarction.

It is difficult to accurately diagnose the cause of the postoperative neurologic deficit based only on physical examination. Postoperative spinal epidural hematomas are common and mostly asymptomatic, although 0.1% require surgical intervention (18). Decompressive operations and placement of fusion constructs are technically more demanding and increase the risk of neurologic complications as a result of hardware misplacement or displacement (6). Such problems can be identified on postoperative computed tomography (CT).

Only a few cases of cervical spinal cord infarction after decompressive surgery have been reported (2, 3, 7). In these studies, the proposed causes of ischemic events included intraoperative or postoperative hypotension or decreased venous return in conjunction with elevated vertebral venous pressures, which reduces arterial perfusion when patients undergo surgery in the prone position (3, 7).

Diagnosing an ischemic event involving the cervical spinal cord based on imaging is also challenging. Decompression is usually performed in conjunction with the placement of fusion hardware, which degrades magnetic resonance imaging (MRI) and CT scans, particularly diffusion-weighted sequences. Hemorrhagic products are often present in the postoperative bed, and the spinal cord may be swollen by extensive infarction that effaces cerebrospinal fluid (CSF) spaces and simulates compression.

We retrospectively evaluated five patients who presented after elective decompressive surgery with early and persistent postoperative neurologic deficits associated with abnormal imaging consistent with spinal cord infarctions. In this article, we discuss goals for postoperative spinal cord imaging, imaging findings of cord infarctions, and different vascular and neuroangiologic explanations.

METHODS

Permission was obtained from the institutional review board to review hospital data.
results

Demographics, imaging, surgical variables, anesthesia records, neurologic outcome, follow-up, and results are summarized in Table 1 for all five cases.

Case 1

A 76-year-old man with a 1-month history of painful "electric shocks" in his neck and radiating into his shoulder was admitted for a C4-C7 laminectomy with decompression and stabilization of the spinal cord using lateral mass screws and posterolateral fusion. His medical history included chronic obstructive pulmonary disease, prostate cancer, colon cancer, and hypertension. Preoperative motor power grades were 5/5 in all extremities. Preoperative MRI showed multilevel cervical degenerative changes most pronounced at C3-4, where there was mild spinal cord compression (Figure 1A). No prior magnetic resonance angiography was available, but retrospective review of preoperative MRI showed loss of the right vertebral flow void on cervical spine MRI. Subsequent medical evaluation showed that the patient's blood pressure before surgery was 120/71 mm Hg. The surgical procedure was performed with the patient placed in the prone position.
The surgery proceeded without complications. Blood pressure and SSEPs were within normal limits throughout the procedure. However, in the intensive care unit on postoperative day 4, the patient's condition began to deteriorate (upper and lower extremity weakness) after a hypotensive episode (mean arterial pressure, 80 mm Hg). T2-weighted MRI sequences showed an extensive area of increased signal intensity from C2 to T3 and effacement of CSF at the surgical site (Figure 1B). It was decided that the patient should undergo a second surgery to revise the hardware and exclude potential cord injury, expand the decompression, and place a lumbar drain. The second procedure proceeded without complications, although the patient remained severely quadriplegic thereafter with 0/5 sensation in the lower extremities. A follow-up vascular study (Figure 1C and D) showed proximal occlusion of the right vertebral artery and stenosis at the origin of the left vertebral artery, which had a tortuous course. Follow-up MRI showed the development of a thin, myelomalacic spinal cord from the bottom of C2 to the top of C5, consistent with evolution of a cord infarction.

Case 2
A 64-year-old man with a long history of progressive neck discomfort, numbness, and sharp burning pain in both extremities underwent a cervical laminectomy (C3-C6) with decompression of the spinal cord and nerve roots and stabilization using lateral mass screws with fusion. His medical history included chronic obstructive pulmonary disease, pancreatitis, cirrhosis, alcoholism, and gallstones. Preoperative motor power grades were 5/5 in all extremities. Preoperative MRI showed spinal cord compression from C3-4 through C5-6, with narrowing of the caliber of the spinal cord and increased signal intensity at C3-4 and C4-5. His blood pressure before surgery was 118/86 mm Hg, and his oxygen saturation was 98% on room air.

The surgical procedure was performed with the patient placed in prone position. After general anesthesia was induced, baseline SSEPs were obtained. Midway through the procedure, the SSEPs began to decline slowly concomitant with a decrease in the patient's blood pressure (mean arterial pressure, 50-70 mm Hg). The patient's blood pressure was immediately corrected, but his SSEPs did not return to baseline.

Postoperatively, the patient had right-sided weakness. Manual muscle testing showed the following: right biceps, 0/5; right wrist extension, 0/5; and right triceps, 0/5. Lower extremities had zero strength and tone with absent reflexes. Sensation was absent to pinprick below C6 on the right and below C7 on the left. On postoperative day 1, T2-weighted MRI showed an extensive area of hyperintense...
signal within the spinal cord (Figure 2A and B) and residual myelomalacia. The patient was placed on high-dose dexamethasone treatment and a rehabilitation program. By 6 months after surgery, he had partially recovered strength and sensation in the right upper extremity, but both lower extremities showed minimal improvement.

Case 3
A 45-year-old woman who had undergone surgery for a retroperitoneal paraganglioma and a recurrence 1 year earlier now presented with brain metastases. She had complained of neck pain and left C6 radicular symptoms for 1 month. Preoperative motor power grades were 5/5 in all extremities. MRI showed a mass at C6-C7 with epidural extension that was assumed to be a bony metastatic lesion. Preoperative embolization was performed with polyvinyl alcohol and coils (primarily supplied by the thyrrocervical trunk). The patient was neurologically intact and stable after embolization.

She underwent a C6-C7 corpectomy with placement of an allograft strut, anterior instrumentation at C5-T1, and a posterior fusion involving C3-T4. Immediately after surgery, she could move her legs. However, right lower extremity weakness developed the next morning and evolved to bilateral lower extremity weakness and hand weakness. Postoperative MRI showed a region of high signal intensity in the spinal cord at the C6-C7 level consistent with infarction. There was no compression of the spinal cord by hematoma or a displaced graft.

The patient was taken to a CT scan suite. A supine C1-2 puncture was performed with CT guidance, and myelographic contrast agent was injected for postmyelographic CT. This study excluded extrinsic mass effect on the spinal cord and the absence of focal dural or spinal cord abnormalities other than preexisting disease.

Case 4
A 68-year-old man with a history of radicular symptoms underwent a two-level anterior cervical discectomy. Preoperative myelography and CT confirmed significant stenosis at C5-6 and C6-7. Preoperative motor grades were 5/5 in all extremities. The patient had a history of hypertension and smoking. No preoperative or postoperative vascular studies were obtained. During surgery, he developed a hypertensive episode (record unavailable but mentioned in the operative report) but recovered with fluid resuscitation. In the recovery room, he was noted to have flaccid quadriplegia.

Case 5
A 53-year-old woman had undergone a transoral odontoidectomy and posterior fusion from the occiput to C3 to treat a plasmacytoma and myeloma 3 months earlier. She subsequently sought treatment for neck pain and moderate weakness in all extremities. MRI showed an epidural abscess anteriorly and posteriorly at C3-4 and C4-5. The patient was admitted and underwent a partial cervical laminectomy at C3, full laminectomy at C4 and C5, and partial laminectomy at C6 with lateral mass screw instrumented arthrodesis at C4 and C5. Prior neck magnetic resonance angiography images were unavailable. Her blood pressure was 134/89 mm Hg.

The surgical procedure was performed with the patient placed in the prone position...
position. SSEPs were not monitored. The surgery proceeded without complications, and her blood pressure was stable throughout the procedure. On postoperative day 1, the patient developed weakness associated with hypotension (record unavailable but mentioned in chart report). The weakness progressed to quadriplegia, and the patient was given fluid resuscitation. MRI showed increased signal intensity from C2 caudally to C7-T1 for which the patient was started on high-dose dexamethasone treatment and a rehabilitation program.

DISCUSSION

Ischemic infarctions of the cervical spinal cord are very rare compared with the prevalence of brain infarctions and constitute only 1% of all central nervous system infarctions (22). Based on a review of the literature and our series, there appear to be two general patterns of restricted arterial supply to the cervical spinal cord causing spinal cord infarctions as first published by Novy et al. (22). In the first pattern, larger vessels are involved with globally decreased perfusion to the spinal cord. This type may manifest with more extensive spinal cord involvement or with smaller border zone infarcts. The second pattern is caused by localized vascular disease and may show more focal involvement (22).

The first pattern associated with a globally reduced arterial supply to the cervical cord may be caused by vertebral artery compromise, hypotension, or both (case 1). There have been numerous reports of spinal cord ischemia from advanced atherosclerotic vertebral artery disease and vertebral artery dissections (11, 14, 25, 26). The second pattern reflects diverse vascular entities that locally or focally reduce the arterial supply to the cervical spinal cord (cases 3 and 5). Such causes include diabetes, vasculitis, emboli, spinal artery compression from herniated disks, epidural infections, syphilis, caisson disease, coagulopathies, sickle cell disease, tuberculosis, arteriovenous malformations, osteophytic narrowing of the neuroforamen at the same level as a radiculomedullary branch, cervical subluxation, and selective angiographic procedures with inadvertent catheterization and contrast agent injection of branches supplying the spinal cord (4, 16, 20, 22-24).

On conventional MRJ sequences, pencil-shaped signal changes on the superior and inferior margins are a feature of spinal cord infarctions (Figure 1B) (2, 27, 28). These changes reflect the greater effects on the central spinal cord from anterior spinal artery involvement and the susceptibility of the central spinal cord gray matter to ischemia. Approximately 50% of cases display abnormal signal intensity on T2-weighted sequences within the first 24 hours (2). However, diffusion-weighted sequences may show evidence of infarction within 8 hours if no hardware has been placed (1, 2, 10, 17).

Signal changes predominantly involve the central aspect of the spinal cord. A peripheral rim of tissue may be preserved by the collateral pial network and the posterior spinal arteries (28) and the propensity for greater involvement by the anterior spinal artery and central gray matter (Figure 3A-D). The complete transverse aspect of the spinal cord may be involved with extensive infarction, whereas the central portion of the spinal cord may be involved only at the superior and inferior edges. If the size of the infarct is significant, the spinal cord is swollen during the acute stage. During the subacute stage, contrast enhancement is visible and may persist for 3 weeks. The enhancement may reflect involvement of the central gray matter, and it may appear as a "snake-eye" on postcontrast axial T1-weighted and T2-weighted MRJ sequences. Associated increases in signal intensity may be present in the adjacent vertebral body (28).

Postoperative fusion associated with hardware placement diminishes the utility of diffusion imaging and degrades all cervical MRI sequences. In the acute postoperative setting, neuroimaging evaluation for spinal canal compression may have pitfalls. Metallic distortion of MRJ sequences and artifact on CT can complicate the evaluation for displaced instrumentation and epidural hematomas. A swollen spinal cord caused by extensive infarction with complete effacement of adjacent CSF spaces and the presence of postoperative blood can also complicate the evaluation for significant postoperative epidural hematoma (Figure 4). In patients without acute or persistent neurologic deficits, postoperative edema of the spinal cord can be difficult to differentiate from spinal cord infarction based solely on MRI findings. Lee et al. (19) reported six cases with postoperative edema of the white matter of the cervical spinal cord after laminectomy in patients with no symptoms of infarction. They theorized that the cause was a disturbance in venous circulation.

Our patients had preexisting significant vascular compromise or vascular risk factors that could have predisposed the cervical spinal cords to ischemic changes. Such changes may be caused solely by vascular disease (22). Suzuki et al. (26) reported a patient with decreased arterial perfusion and infarction of the cervical cord without prior surgery. The patient had identical vascular disease to our case 1 with an infarction at C3-C4, an occluded right vertebral artery, and stenosis of the left vertebral artery. There are several reports of cervical cord infarctions as a consequence of arterial dissection (11, 14, 25). The patient in case 3 underwent preoperative embolization and was neurologically intact after the procedure; however, regional perfusion to the spinal cord may have been diminished by the embolization. In case 5, an extensive epidural abscess may have restricted the arterial or venous vasculature, or both. Cases 2 and 4 both had significant vascular risk factors. The patient in case 2 had a small localized spinal cord infarction and likely had local or regional vascular disease. The patient in case 4 had a hypertensive episode and a presentation similar to case 1 with extensive and irreversible spinal cord infarction. This patient likely had an extensive infarction from globally reduced arterial restriction and hypotension similar to case 1.

Systemic hypotensive and reduced perfusion episodes in patients are common causes of ischemic changes in the brain and are rarely associated with spinal cord infarctions. Conversely, four of our patients had hypertensive episodes and spinal cord infarctions but no brain involvement based on clinical examination or diagnostic imaging. Two of the hypertensive episodes occurred during surgery, and two occurred during the postoperative period. Hypotension alone may not explain the pattern of isolated cervical cord infarcts without associated brain involvement.

New-onset neurologic deficits compatible with spinal cord infarction related to decompressive spinal surgery are rare. Only
Cervical spinal cord infarction and epidural blood effaces cerebrospinal fluid space in case 1. Extensive infarction with enlargement of the spinal cord and postoperative blood (arrows) simulate compression. (Used with permission from the Barrow Neurological Institute.)

Figure 4. Postoperative spinal cord infarction and epidural blood effaces cerebrospinal fluid space in case 1. Extensive infarction with enlargement of the spinal cord and postoperative blood (arrows) simulate compression. (Used with permission from the Barrow Neurological Institute.)

Figure 3. Cervical cord anterior spinal artery supply and cross-sectional cord infarction pattern are depicted. Posterior spinal artery supply is not shown because arteries are more numerous and highly variable. (A) Coronal illustration highlights the common patterns of blood supply to the cervical spinal cord to the anterior spinal artery. 1. The most common supply to the artery of the cervical enlargement is directly from the vertebral artery (red). 2. The anterior spinal artery originates from the distal vertebral arteries (red). 3. Midcervical branches from the vertebral artery may augment flow to the anterior spinal artery (blue). 4. Most common variant for origin of the artery of the cervical enlargement from the deep cervical branch of the costocervical artery (green). 5. Uncommon variant for blood supply to the artery of the cervical enlargement originates from the anterior cerebral branch of the thyrocervical artery (green). (B) Early phase computed tomography angiogram, coronally reformatted image, demonstrates the artery of the cervical enlargement (arrow). (C) Axial illustration delineating central and peripheral cervical spinal cord arterial vascular supply and ascending and descending spinal cord tracts. Arteries (right-side illustration): 1. posterior spinal arteries; 2. periperal pial supply; 3. central sulcal artery; 4. anterior spinal artery. Larger white matter tracts (left-side illustration), ascending in green and descending in yellow: 1. dorsal columns; 2. lateral corticospinal (pyramidal) tract; 3. posterior spinocerebellar tract; 4. lateral spinothalamic tract; 5. anterior spinocerebellar tract; 6. anterior spinothalamic tract; 7. anterior corticospinal tract. (D) Axial T2-weighted magnetic resonance imaging shows signal within the central cord with a preserved peripheral rim of tissue (arrow). (A) and (C) used with permission from Deborah Ravin. B and D used with permission from the Barrow Neurological Institute.)

a few pathophysiologic explanations have been suggested. Bhardwaj et al. (3) theorized that surgery in the prone position, reduced venous return, increased vertebral venous pressure, and decreased arterial pressure combined with hypotension were the primary causes. However, two of our patients developed spinal cord infarctions caused by hypotension after surgery, and three of the infarctions occurred in patients while in the supine position. Only two of our five patients met the conditions of Bhardwaj et al. (3) for cervical spinal cord infarctions associated with laminection. All of the patients reported by Cybulski and D'Angelo (7) developed infarctions during the postoperative period after hypotensive episodes.

One of the most frequently proposed causes of spinal cord infarction after spine surgery is hypotension. Bhardwaj et al. (3) reported four cases of new-onset neurologic deficits after cervical laminectomy. They identified a significant decrease in blood pressure during surgery as a contributing cause because there was no evidence of spinal compression in their cases. Similarly, profound intraoperative hypotension has resulted in anterior spinal artery syndrome after an anterior thoracic spinal fusion (2). Cybulski and D'Angelo (7) reported four cases of focal spinal cord injury associated with a postoperative hypotensive episode after cervical decompression surgery for spondylotic myeloradiculopathy. They
suggested the possibility of an ischemic origin related to hypotension because their patients were neurologically stable for >24 hours after surgery. Blood pressure was stable before the hypotensive event, and compressive mechanical factors were excluded by imaging. Likewise, Ebersold et al. (8) had two patients with new neurologic deficits. The first patient was treated via anterior decompression and fusion and developed weakness on postoperative day 3 after a sudden hypotensive episode. The second patient was treated via posterior laminectomy and developed neurologic deterioration after a hypotensive episode 64 hours after surgery.

An additional proposed cause of spine infarction correlates with reoxygenation-reperfusion injury to the spinal cord. After partial ischemia and subsequent reperfusion in animal models, spinal cord metabolism is affected secondary to the release of free fatty acids, to the increased intake of Ca++, and to enhanced vascular permeability (9, 12, 13, 15). In the clinical setting, C5 palsy after decompressive surgery is common. Some authors attribute reperfusion as the conditioning factor (5). However, the largest reported study to date failed to show any correlation between the incidence of C5 palsy and intraoperative SSEP changes (21). The most compelling clinical observation in our patients was the change in SSEPs during surgery. Changes in the symptoms of our patient who became quadriplegic postoperatively were temporally synchronous with the onset of hypotension. This case suggests that reduced arterial flow from vascular compromise, in conjunction with hypotension, was the predominant cause of the neurologic changes. Perfusion pressure breakdown would have shown less temporal correlation with the onset of hypotension.

We propose that preexisting, unrecognized arterial vascular compromise alone or in conjunction with hypotension may play a role in some patients who develop a spinal cord infarct after cervical spine surgery. Each year, a large number of cervical spine surgeries are performed, yet reports of postoperative spinal cord infarctions are exceedingly rare. Preoperative screening would require enormous investments of time and money, and even noninvasive screening vascular examinations are not completely risk-free. Although screening examinations may detect large-vascular abnormalities, smaller focal vascular disease would likely not be recognizable on most noninvasive vascular screening modalities. Conversely, patients with known preexisting vascular studies obtained for unrelated reasons with bilateral vertebral artery disease or significant compromise of multiple great vessels from the aortic arch may warrant greater vigilance for even minor degrees of hypotension during or after cervical spine surgery.

A limitation of our study is the small number of cases; however, the incidence of spinal cord infarctions is very rare even at large tertiary referral centers with significant case volumes. We observed an immediate correlation between the presence of a hypotensive event and the clinical presentation of cord infarction; blood pressure measurements were unavailable in two cases, and the duration of the hypotensive event was not accurately reported in all cases. Preoperative and postoperative vascular imaging studies were not available in all cases because the authors' centers do not routinely perform preoperative vascular screening. Focal or localized vascular disease involving moderate or small vessels may not be detected on vascular screening studies. Postoperative imaging was degraded by metallic artifact in most cases, and diffusion-weighted images were unavailable. Diffusion-weighted imaging of the cervical spine is not routinely performed, and none of our patients underwent diffusion imaging because of their metallic hardware and susceptibility artifact.

CONCLUSIONS

In the setting of postoperative decompression of the spinal cord and new onset of neurologic symptoms, neuroimaging is performed (i) to exclude spinal cord compression by a postoperative hematoma, displaced constructs, or misplaced hardware; (ii) to confirm adequate surgical decompression of the spinal cord; and (iii) to corroborate neuroimaging findings with spinal cord infarction. Our cases support an association between hypotension and postoperative spinal cord infarction in some patients. Antecedent, unrecognized preoperative vascular compromise may likely be a contributor to some postoperative spinal cord infarctions alone or in conjunction with hypotension.

REFERENCES


