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
MRI: Magnetic resonance imaging
SSEPs: Somatosensory evoked potentials

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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 cause. 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 neuroradiologic explanations.

METHODS

Permission was obtained from the institutional review board to review hospital

databases, medical records, and imaging studies. The authors have completed and maintain certification for the National Institutes of Health training course "Protecting Human Research Participants."

The inclusion criteria were as follows: acute onset of neurologic symptoms after elective cervical spine surgery; exclusion of reversible structural causes; follow-up clinical course consistent with spinal cord infarction; and findings on MRI, CT, or both consistent with spinal cord infarction. Five patients (three men and two women; mean age, 61.2 years) met the inclusion criteria and were evaluated during the 26-year interval from 1984–2010 by physicians at the authors' two institutions. Available hospital records were reviewed for type of cervical spine surgery, patient age, vascular risk factors, anesthesia monitoring, intraoperative somatosensory evoked potentials (SSEPs), presence or degree of hypotension, postoperative clinical course, and findings on postoperative imaging.

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

Table 1. Summary of Cervical Cord Infarction Presentation After Decompressive Surgery in Five Cases

Case Number	Age (years)/Sex	Type of Surgery	Hypotension Presentation	Intraoperative SSEPs	Length of Follow-up	Degree of Cervical Cord Infarction	Imaging Findings
1	76/M	C4-C7 decompression, lateral mass and posterolateral fusion	Postoperative day 4, hypotension single episode, MAP 50–70 mm Hg	Stable	6 months	Severe quadriparesis	Abnormal cord signal and increased AP cord dimension from C2 to T3, no displacement of surgical hardware
2	64/M	C3-C6 decompression and stabilization by lateral mass screws and fusion	Intraoperative single episode, MAP 50–70 mm Hg	Abnormal midway through surgery	24 months	Severe quadriparesis, sensation absent to pinprick below C6 on right and C7 on left	Abnormal cord signal at C3 and C4 without cord enlargement, no displacement of fusion hardware
3	45/F	Preoperative embolization, C6-7 corpectomy, strut graft, anterior instrumentation, and posterior fusion	None	Stable	2 weeks	Initially RLE weakness evolving into bilateral LE and hand weakness	Abnormal cord signal and increased AP cord dimension from top of C5 to mid-T1, no displacement of bone graft or hardware
4	68/M	C5-6 and C6-7 anterior cervical diskectomies	Intraoperative single episode, BP records unavailable	Not available	2 weeks	Severe quadriparesis	No cord compression, no deformity of thecal sac or spinal cord on cervical postmyelography CT
5	53/F	Laminectomies C3-C6 and lateral mass screw arthrodesis C4 and C5	Postoperative day 1 single episode, BP records unavailable	Not monitored	1.5 weeks	Severe quadriparesis	Abnormal cord signal and increased AP cord dimension from C2 to C7-T1, no displacement of hardware

SSEPs, somatosensory evoked potentials; M, male; MAP, mean arterial pressure; AP, anteroposterior; F, female; RLE, right lower extremity; LE, lower extremity; BP, blood pressure; CT, computed tomography.

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