

Case Report

Cerebrospinal fluid drainage and blood pressure elevation to treat acute spinal cord infarct

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Abstract

Background: Current management of acute spinal cord infarction (SCI) is limited. Lumbar cerebrospinal fluid drainage (CSFD) with blood pressure augmentation is utilized in the thoracic/thoracoabdominal aortic repair and thoracic endovascular aortic repair (TEVAR) populations to increase spinal perfusion pressure.

Case description: We identified 3 patients who sustained acute SCI and underwent CSFD and maintenance of elevated mean arterial pressure (MAP) within 24 hours of injury. The first patient exhibited delayed-onset ischemia after a TEVAR. The second patient presented with an acute type B aortic intramural hematoma. The third patient developed spinal cord ischemia following bronchial artery embolization. There was significant improvement in the motor examination (e.g., ASIA impairment scale grade B or C) to grade D utilizing both blood pressure augmentation and CSFD.

Conclusions: Lumbar CSFD with MAP elevation benefited 3 patients with acute SCI of varying etiologies.

Key Words: Acute stroke therapy, intervention, ischemic stroke, treatment

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INTRODUCTION

Lumbar cerebrospinal fluid drainage (CSFD) helps prevent spinal cord injury for patients undergoing open or endoscopic thoracic or thoracoabdominal aortic aneurysm and thoracic endovascular aortic repair (TAA/TAAA/TEVAR) surgery.^[4,5] When combined with augmentation of the systemic blood pressure, CSFD reduces the risk of spinal cord infarction (SCI) by increasing the afferent spinal cord blood supply and perfusion pressure.^[8] Here we present three cases in which patients with different types of acute SCI neurologically improved [e.g., using the National Institutes of Health Stroke Scale (NIHSS) and American Spinal Injury Association (ASIA)] following elevation of the systemic blood pressure and placement of CSFD.

CASE REPORTS

Three patients from 2013 to 2016 who presented with acute SCI underwent the placement of CSFD combined with the elevation of systemic blood pressure

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within 24 hours of injury. Relevant demographic data and clinical variables were retrospectively collected. We obtained approval from an Ethical Standards Committee.

CASE DESCRIPTION

Case 1

A 77-year-old male with a history of hypertension (HTN), hyperlipidemia (HLD), focal segmental glomerulosclerosis with a kidney transplant, and a 15 pack-year smoking history underwent a TEVAR for TAA. Lumbar CSFD was discontinued after 48 h at which point the neurological examination was normal. Eight days postoperatively, he developed *Escherichia coli* sepsis with atrial fibrillation/rapid ventricular rate, and acute flaccid paraplegia [Table 1]. The thoracic magnetic resonance imaging (MRI) documented an acute SCI, and he underwent CSFD placement [Figure 1]. Within hours, his examination improved from ASIA (AIS) grade B to grade D. He was ambulatory at the time of discharge.

Case 2

A 61-year-old male with a history of uncontrolled HTN and HLD presented with 6 hours of chest pain, acute bilateral lower extremity weakness, a T4 sensory level, and urinary retention [Table 1]. The computed tomography angiogram revealed a type B intramural hematoma from the origin of the subclavian artery to the abdominal aorta [Figure 1]. A lumbar CSFD was placed and the patient improved from AIS grade C to grade D, at which point he regained normal baseline neurological function.

Case 3

A 61-year-old male with HTN, diabetes mellitus, and pulmonary *Mycobacterium avium intracellulare* was admitted for hemoptysis for which he underwent bronchial artery embolization. Immediately following the procedure, he developed acute right lower extremity weakness that exacerbated four days later when he also became hypotensive and tachycardic due to dehydration [Table 1]. When the thoracic MRI revealed an acute SCI, CSFD was initiated with improvement in AIS grade B to grade D [Figure 1].

DISCUSSION

For at-risk patients undergoing TAA/TAAA repair or TEVAR, guidelines recommend: (1) elevation of the mean arterial pressure (MAP) from 60 to 90–100 mmHg, and (2) lowering the intrathecal pressure to maintain spinal cord perfusion pressure (MAP – ITP).^[6] Surgical techniques such as aortic retraction and cross-clamping produce acute elevations in CSF pressure, which impairs venous outflow leading to ischemia.^[8] Such ischemia may be reversible in some cases by utilizing CSFD (e.g., to

Table 1: Patient characteristics and hospital course

Patient	Age (years)	Gender (M/F)	Etiology	PTA medications	Initial examination	MRI results	Time to CSFD (hours)	Lumbar drain location	CSFD duration (days)	Other therapy	Examination after drain removed	Length of hospitalization (days)	Discharge disposition
P1	77	M	Delayed paraplegia after TEVAR	Asa, atorvastatin, anti-htn, diuretic, low-dose prednisone, tacrolimus, mycophenolate	Flaccid paraplegia, NIHSS 8 (4 LLE, 4 RLE)	T12-L1 T2 hyperintense lesion	1	L4-L5	5	Vasopressors, IV abx, amiodarone, AC	BLE weakness, NIHSS 2 (1 LLE, 1 RLE)	18	AR
P2	61	M	Type B aortic dissection	Simvastatin, sertraline	Urinary retention, T4 sensory level (sparing dorsal columns), BLE weakness, NIHSS 7 (3 LLE, 3 RLE, 1 sensory)	C4-C6 cervical spondylosis	23	L3-L4	8	Asa, clevidipine, beta blocker	Baseline LLE weakness, NIHSS 0	14	AR
P3	61	M	Bronchial artery embolization	Rifampin, ethambutol, clarithromycin, amlodipine	Initially RLE weakness, NIHSS 3; worsened to urinary retention, patchy sensory deficit, BLE weakness, NIHSS 7 (2 LLE, 4 RLE, 1 sensory)	T2-T3 diffusion restriction, T3-T4 hyperintense lesion	20	L3-L4	8	Asa	RLE weakness, NIHSS 3	10	AR

TEVAR=Thoracic endovascular aortic repair, Asa=Acetylsalicylic acid, NIHSS=NIH stroke scale, PTA=Prior to admission, MRI=Magnetic resonance imaging, M=Male, F=Female, LLE=Left lower extremity, RLE=Right lower extremity, BLE=Bilateral lower extremities, abx=Antibiotics, AC=Anticoagulation, A=Acute rehabilitation

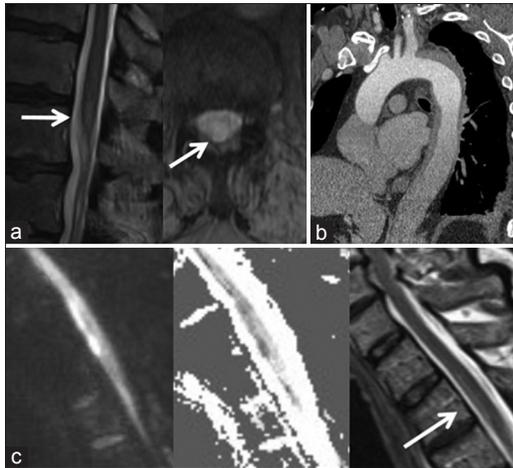


Figure 1: Radiographic findings in acute spinal cord infarction. (a) T2-weighted sagittal and axial MRI of the thoracic spine shows a hyperintense lesion at T12-L1. (b) CT angiogram of the aorta shows an intramural hematoma from the origin of the left subclavian artery to the proximal abdominal aorta. (c) Diffusion-weighted sagittal MRI shows abnormal diffusion restriction at T2-T3 which is confirmed by ADC maps, and is associated with a T2-weighted hyperintense lesion from T2-T4

be used for 36–48 hours up to 5–8 days) while also supporting systemic blood pressure.^[1-3,7]

Limitations to this approach include delay to diagnosis, lumbar/cranial (2.9%) hemorrhage in patients with anticoagulation/coagulopathy, infection, and procedure-related complications.^[10]

Here, all three patients improved their AIS grades to D prior to drain removal. In one study, 91% of SCI patients with mild (e.g., ASIA D) impairment achieved ambulation at follow-up.^[9] For acute SCI of variable etiology, therefore, CSFD with MAP elevation may prove beneficial in reversing the severity/extent of motor deficit.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have

given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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