Drug overdose resulting in quadriplegia

Teresa S. Wang · Betsy H. Grunch · Jessica R. Moreno ·
Carlos A. Bagley · Oren N. Gottfried

Received: 6 August 2011 / Revised: 15 November 2011 / Accepted: 30 December 2011 / Published online: 11 January 2012
© Springer-Verlag 2012

Abstract

Purpose To describe a case of cervical flexion myelopathy resulting from a drug overdose.

Methods A 56-year-old male presented to the emergency department unable to move his extremities following drug overdose. Neurological examination revealed him to be at C6 ASIA A spinal cord injury. The CT of his cervical spine revealed no fracture; however, an MRI revealed cord edema extending from C3 to C6 as well as posterior paraspinal signal abnormalities suggestive of ligamentous injury.

Results The patient underwent a posterior cervical laminectomy and fusion from C3 to C7. Neurologically he regained 3/5 bilateral tricep function and 2/5 grip; otherwise, he remained at ASIA A spinal cord injury at 6 months.

Conclusion Our patient suffered a spinal cord injury likely due to existing cervical stenosis, and in addition to an overdose of sedating medications, he likely sat in flexed neck position for prolonged period of time with the inability to modify his position. This likely resulted in cervical spine vascular and/or neurological compromise producing an irreversible spinal cord injury. Spinal cord injury is a rare finding in patients presenting with drug overdose. The lack of physical exam findings suggestive of trauma may delay prompt diagnosis and treatment, and thus

clinicians must have a high index of suspicion when evaluating patients in this setting.

Keywords Drug overdose · Suicide attempt · Quadriplegia · Cervical flexion myelopathy

Introduction

Flexion myelopathy, defined as a spinal cord injury caused by neck flexion, is due to a variety of conditions that include subluxation, excessive kyphosis, tight dural canal mechanism, and overstretching mechanism [1–3]. These proposed mechanisms consist of: (1) spinal cord compression by the anterior elements, including the vertebral bodies that provide contact pressure on the cord [4], (2) spinal cord stretching in the up-down direction, i.e., overstretching mechanism [3], and (3) spinal cord compression by a narrow dural canal due to the forward migration of the lower cervical posterior dura mater, i.e., tight dural canal mechanism [2, 5]. Upon flexion of the neck, the cervical spinal cord curves most markedly at the levels of C5–C6, contributing to the largest accumulation of pressure in this area [2, 6]. Neurological symptoms of cervical flexion myelopathy (CFM) include weakness and atrophy of the left forearm and hand muscles with fasciculation, but without definite sensory disturbances [7].

Cervical flexion myelopathy has also been implicated in cases of quadriplegia developing after posterior fossa surgery in either the sitting or prone positions [1, 8]. The sitting position in neurosurgery has been controversial because of its many operative advantages as well as some disastrous complications. For patients in the sitting position during posterior fossa surgery, the neck is placed in a hyperflexed position, generally with the chin almost

T. S. Wang · B. H. Grunch (✉) · J. R. Moreno ·
C. A. Bagley · O. N. Gottfried
Division of Neurosurgery,
Duke University Medical Center, 3807,
Durham, NC 27710, USA
e-mail: betsy.grunch@duke.edu

O. N. Gottfried
e-mail: oren.gottfried@duke.edu

Fig. 1 **a** Sagittal T2 weighted MRI reveals cord edema extending from C3 to C6 as well as posterior paraspinal signal abnormalities suggestive of ligamentous injury. **b** Axial T2 weighted image demonstrating a disc herniation causing cord compression. **c** Axial T2 weighted imaging demonstrating his preexisting cervical stenosis

touching the anterior chest [8]. It has been reported that hyperflexion of the neck during surgery plays an important role in causing quadriplegia by overstretching the cervical spinal cord and compromising its blood supply [1].

Case report

We report a case of quadriplegia following a drug overdose suicide attempt in a patient without any signs of external trauma. Potential mechanisms for the patient's spinal cord injury are discussed.

Clinical and radiographic findings

A 56-year-old male with a past medical history of depression was evaluated in the emergency department following a suicide attempt. The patient was found by a friend in the flexed position on his couch several hours after an overdose of oxycodone, xanax, and klonopin. He was taken to the emergency department by ambulance and was found to have sustained a high cervical spinal cord injury. His neurological examination included loss of all sensory modalities below the T2 dermatome, and motor examination revealed 1/5 deltoid, 4/5 bicep, and 3/5 wrist extensors bilaterally. All other motor groups were 0/5. All reflexes were absent. Computerized tomography (CT) revealed no fractures. An emergent magnetic resonance imaging (MRI) was performed that revealed T2 signal abnormalities and cord edema extending from C3 to C6, as well as posterior paraspinal signal abnormalities suggestive of ligamentous injury (Fig. 1a–c). He also had coexistent severe degenerative stenosis.

Operative technique and intraoperative findings

The patient was admitted to the neurointensive care unit, and given his acute spinal cord injury, his mean arterial pressures were kept above 85 using dopamine. He was taken to the operative room on the day after admission for a decompressive laminectomy and fusion from C3 to C7 (Fig. 2). On exposure, there was evidence of injury to posterior cervical musculature, as it was edematous and boggy. There was significant stenosis and with wide laminectomy and removal of hypertrophied ligamentum flavum, there was nice decompression of the thecal sac. There were no obvious fractures consistent with the pre-operative CT.

Results

Postoperatively, he underwent a tracheostomy and gastrostomy tube due to his cord injury. Four weeks postoperatively, he was able to be weaned off the ventilator and

Fig. 2 Postoperative lateral X-ray reveals posterior fusion from C3 to C7

his tricep strength had improved to 3/5 bilaterally. He still had no intrinsic hand function or lower extremity function. He was discharged to an acute rehabilitation center for further therapy needs. During rehabilitation, his intrinsic hand function improved to 2/5 strength. At 6 months, he remains at ASIA A spinal cord injury. Of note, the patient received comprehensive psychiatric evaluation and treatment.

Discussion

Cervical flexion myelopathy is due to a variety of causes, including subluxation, excessive kyphosis, overstretching, and tight dural canal [1–3]. While there have been many proposed mechanisms for the pathogenesis of CFM, the exact cause is still unknown. It has been hypothesized that hyperflexion of the neck causes a severe pressure increase at the levels C5–C6, which overstretches the spinal cord and compromises the blood supply [9]. CFM can also occur in patients who undergo posterior fossa surgery in either the sitting or prone positions [1, 8].

We are aware of two similar cases of CFM due to a drug overdose [10, 11]. One of the cases involved a 25-year-old woman who was admitted to the hospital after an overdose of valproic acid in a suicide attempt. She was found in the sitting position with her neck in extreme flexion for ~18 h. Upon awakening the patient was quadriplegic, and an MRI showed an enlarged cervical cord, maximally at C6, with prominent paraspinal soft tissue swelling [10]. She died from pulmonary embolism and pneumonia. The authors concluded that this prolonged neck hyperflexion, along with profound muscular relaxation due to her unconsciousness,

led to a severe myelopathy associated with compromise of the spinal cord's microcirculation. The second case involves a 34-year-old woman who developed cervical and thoracic flexion myelopathy with severe quadriparesis after an overdose of quetiapine fumarate, oxycodone/acetaminophen, and chloral hydrate [11]. The pathogenesis of the spinal cord injuries in drug overdose has two possible mechanisms: (1) vascular theory which suggests that acute stretching of the spinal cord altered the autoregulation of the vasculature [12] and (2) neuronal theory which suggests that the cord was damaged by a direct mechanical distortion through compression or stretching [6]. Our patient had existing cervical stenosis, and in addition to an overdose of sedating medications, he likely sat in flexed neck position for prolonged period of time with the inability to modify his position. This likely resulted in cervical spine vascular and/or neurological compromise producing an irreversible spinal cord injury.

We report an additional case of CFM resulting in acute quadriplegia due to drug overdose of narcotics and benzodiazepines. While acute spinal cord injury in “nontraumatic” drug overdose is rare, treating physicians must be cognizant of the entity as delay in diagnosis and treatment can be detrimental.

Conflict of interest None of the authors has any potential conflict of interest.

References

1. Rau CS, Liang CL, Lui CC, Lee TC, Lu K (2002) Quadriplegia in a patient who underwent posterior fossa surgery in the prone position. Case report. *J Neurosurg* 96:101–103
2. Iwasaki Y, Tashiro K, Kikuchi S, Kitagawa M, Isu T, Abe H (1987) Cervical flexion myelopathy: a “tight dural canal mechanism”. Case report. *J Neurosurg* 66:935–937
3. Mii K, Iida H, Tachibana S et al (1989) The overstretch syndrome: a new cervical myelopathy caused by the stretch mechanism of the spinal cord. *Spinal Surg* 3:137–141
4. Watanabe K, Hasegawa K, Hirano T, Endo N, Yamazaki A, Homma T (2005) Anterior spinal decompression and fusion for cervical flexion myelopathy in young patients. *J Neurosurg Spine* 3:86–91
5. Kikuchi S, Tashiro K, Kitagawa M et al (1987) A mechanism of juvenile muscular atrophy localized in the hand and forearm (Hirayama's disease)—flexion myelopathy with tight dural canal in flexion. *Clin Neurol* 27:412–419
6. Brieg A (1960) Biomechanics of the central nervous system. Stockholm, Sweden
7. Mii K, Shimizu S, Tanaka C, Matsumori K, Hasegawa K, Mizoi Y (1995) Thoracic flexion myelopathy. Case report. *J Neurosurg* 82:1059–1061
8. Haisa T, Kondo T (1996) Midcervical flexion myelopathy after posterior fossa surgery in the sitting position: case report. *Neurosurgery* 38:819–822
9. Fujimoto Y, Oka S, Tanaka N, Nishikawa K, Kawagoe H, Baba I (2002) Pathophysiology and treatment for cervical flexion myelopathy. *Eur Spine J* 11:276–285
10. Kaye KL, Ramsay D, Young GB (2001) Cervical flexion myelopathy after valproic acid overdose. *Spine* 26:E459–E462
11. Nielsen AS, Damek DM (2008) Window of opportunity: flexion myelopathy after drug overdose. *J Emerg Med* [Epub ahead of print]
12. Nelson E, Gertz SD, Rennels ML, Ducker TB, Blaumanis OR (1977) Spinal cord injury. The role of vascular damage in the pathogenesis of central hemorrhagic necrosis. *Arch Neurol* 34:332–333