Central cord syndrome (CCS)

Key concepts

● a disproportionately greater motor deficit in the upper extremities than lower

● usually results from hyperextension injury in the presence of osteophytes spurs

● surgery is often employed for ongoing compression, usually on a non-emergency basis except for rare cases of progressive deterioration

---

Originally described by Schneider et al [1)] in 1954. Central cord syndrome (CCS) is the most common type of incomplete spinal cord injury syndrome. Usually seen following acute hyperextension injury in an older patient with pre-existing acquired stenosis as a result of bony hypertrophy (anterior spurs) and infolding of redundant ligamentum flavum (posteriorly), sometimes superimposed on congenital spinal stenosis. The translational movement of one vertebra on another may also contribute. A blow to the upper face or forehead is often disclosed on history, or is suggested on the exam (e.g. lacerations or abrasions to face and/or forehead). This often occurs in relation to a motor vehicle accident or to a forward fall, often while intoxicated. Younger patients may also sustain CCS in sporting injuries; see burning hands syndrome. CCS may occur with or without cervical fracture or dislocation [2)]. CCS may be associated with acute traumatic cervical disc herniation. CCS may also occur in rheumatoid arthritis.

Young patients tend to have disc protrusion, subluxation, dislocation or fractures [3)] Older patients tend to have multi-segmental canal narrowing due to osteophytic bars, discs, and inbuckling of ligamentum flavum [4)].

Traumatic central cord syndrome (TCCS) is an incomplete spinal cord injury defined by greater weakness in upper versus lower extremities, variable sensory loss, and variable bladder dysfunction, bowel, and sexual dysfunction.

Acute cervical spinal cord injury (SCI), was initially described by Schneider and colleagues in 1954 [5)]. It is marked by a disproportionately greater impairment of motor function in the upper extremities than in the lower ones, as well as by bladder dysfunction and a variable amount of sensory loss below the level of injury [6)].

Epidemiology

Although CCS has been reported to occur with particular frequency among older persons with cervical spondylosis who sustain hyperextension injury, it can be found in persons of any age and can be associated with various etiologies, injury mechanisms, and predisposing factors.

It is the most common incomplete spinal cord injury.

United States The prevalence rate of central cord syndrome is 15.7-25%.

Mortality/Morbidity Central cord syndrome is generally associated with a favorable prognosis for the achievement of some degree of neurologic and functional recovery.

Sex Similar to all other SCIs, central cord syndrome predominantly affects males.

Age Central cord syndrome (CCS) has a bimodal distribution; in young persons, CCS tends to result from trauma, while in older individuals, it is typically caused by falls sustained by persons with preexisting spondylosis.

Classification

Spontaneous Central cord syndrome.

Traumatic Central cord syndrome

Etiology

The most common cause of central cord syndrome (CCS) is trauma. In older adults, premorbid cervical spondylosis is a significant risk factor. Accordingly, even minor falls may result in tetraplegia in populations with a narrowed spinal canal. In younger age groups, CCS results from major trauma, such as that associated with cervical fracture/subluxations.

In patients presenting with non-traumatic central cord syndrome, it is vital to identify risk factors for infection in a thoroughly obtained patient history, as well as to maintain a low threshold for diagnostic imaging [7)].

Pathophysiology

Central cord syndrome (CCS) most often occurs after a hyperextension injury in an individual with long-standing cervical spondylosis.

The most common mechanism of injury may be direct compression of the cervical spinal cord by buckling of the ligamenta flava into an already narrowed cervical spinal canal; this would explain the predominance of axonal injury in the white matter of the lateral columns [8)].

Historically, spinal cord damage was believed to originate from concussion or contusion of the cord with stasis of axoplasmic flow, causing edematous injury rather than destructive hematomyelia. Autopsy studies subsequently demonstrated that CCS may be caused by bleeding into the central part of the cord, portending a less favorable prognosis. Studies have also shown that CCS probably is associated with axonal disruption in the lateral columns at the level of the injury to the spinal cord, with relative preservation of the grey matter.

The syndrome also may be associated with fracture dislocation injury and compression fracture, especially in a congenitally narrowed spinal canal.

These anteroposterior compressive forces also distribute the greatest damaging effect on the central

mass of the cord substance.

CCS-related motor impairment results from the pattern of lamination of the corticospinal and spinothalamic tracts in the spinal cord. Sacral segments are the most lateral, with lumbar, thoracic, and cervical components arranged somatotopically, proceeding medially toward the central canal.

Symptoms

Symptoms of central cord syndrome occur following trauma (most commonly falls) and consist of upper and lower extremity weakness, with varying degrees of sensory loss. Pain and temperature sensations, as well as the sensation of light touch and of position sense, may be impaired below the level of injury. Neck pain and urinary retention are common.

Physical findings related to central cord syndrome are limited to the neurologic system and consist of upper motor neuron weakness in the upper and lower extremities. This impairment can be described as follows:

Impairment in the upper extremities is usually greater than in the lower extremities and is especially prevalent in the muscles of the hand. Sensory loss is variable, although sacral sensation is usually present. Anal wink, anal sphincter tone, and Babinski reflexes should be tested. Muscle stretch reflexes may initially be absent but will eventually return along with variable degrees of spasticity in affected muscles.

Diagnosis

In the setting of severe spinal cord injuries, such as central cord syndrome, T2 hyperintensity in MRI correlates with acute intramedullary hemorrhage [9].

The signal abnormality is often diffuse, spans several levels, and correlates with severe deficits.

Treatment

see Traumatic central cord syndrome treatment.

Aarabi B, Koltz M, Ibrahimi D. Hyperextension cervical spine injuries and traumatic central cord syndrome. Neurosurg Focus. 2008;25(5):E9. [Medline].

McKinley W, Santos K, Meade M, et al. Incidence and outcomes of spinal cord injury clinical syndromes. J Spinal Cord Med. 2007;30(3):215-24. [Medline]. [Full Text].

Aito S, D'Andrea M, Werhagen L, et al. Neurological and functional outcome in traumatic central cord syndrome. Spinal Cord. Apr 2007;45(4):292-7. [Medline].

Song J, Mizuno J, Inoue T, et al. Clinical evaluation of traumatic central cord syndrome: emphasis on clinical significance of prevertebral hyperintensity, cord compression, and intramedullary high-signal intensity on magnetic resonance imaging. Surg Neurol. Feb 2006;65(2):117-23. [Medline].

Dai L. Magnetic resonance imaging of acute central cord syndrome: correlation with prognosis. Chin Med Sci J. Jun 2001;16(2):107-10. [Medline].

Noonan VK, Kopec JA, Zhang H, et al. Impact of associated conditions resulting from spinal cord injury on health status and quality of life in people with traumatic central cord syndrome. Arch Phys Med Rehabil. Jun 2008;89(6):1074-82. [Medline].

Gil-Agudo A, Perez-Rizo E, Del Ama-Espinosa A, et al. Comparative biomechanical gait analysis of patients with central cord syndrome walking with one crutch and two crutches. Clin Biomech (Bristol, Avon). Aug 2009;24(7):551-7. [Medline].

Chen L, Yang H, Yang T, et al. Effectiveness of surgical treatment for traumatic central cord syndrome. J Neurosurg Spine. Jan 2009;10(1):3-8. [Medline].

Yoshihara H, Yoneoka D. Trends in the treatment for traumatic central cord syndrome without bone injury in the United States from 2000 to 2009. J Trauma Acute Care Surg. Sep 2013;75(3):453-8. [Medline].

Dahdaleh NS, Lawton CD, El Ahmadieh TY, et al. Evidence-based management of central cord syndrome. Neurosurg Focus. Jul 2013;35(1):E6. [Medline].

Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. N Engl J Med. May 17 1990;322(20):1405-11. [Medline].

Haller H, Leblhuber F, Trenkler J, et al. Treatment of chronic neuropathic pain after traumatic central cervical cord lesion with gabapentin. J Neural Transm. Sep 2003;110(9):977-81. [Medline].

Lenehan B, Street J, O'Toole P, et al. Central cord syndrome in Ireland: the effect of age on clinical outcome. Eur Spine J. Oct 2009;18(10):1458-63. [Medline].

Wirz M, Zorner B, Rupp R, et al. Outcome after incomplete spinal cord injury: central cord versus Brown-Sequard syndrome. Spinal Cord. Nov 10 2009;[Medline].

Hohl JB, Lee JY, Horton JA, et al. A Novel Classification System for Traumatic Central Cord Syndrome: The Central Cord Injury Scale (CCIS). Spine (Phila Pa 1976). Mar 11 2010;[Medline].

Anderson DG, Sayadipour A, Limthongkul W, Martin ND, Vaccaro A, Harrop JS. Traumatic central cord syndrome: neurologic recovery after surgical management. Am J Orthop (Belle Mead NJ). Aug 2012;41(8):E104-8. [Medline].

Miranda P, Gomez P, Alday R. Acute traumatic central cord syndrome: analysis of clinical and radiological correlations. J Neurosurg Sci. Dec 2008;52(4):107-12; discussion 112. [Medline].

Chen TY, Lee ST, Lui TN, et al. Efficacy of surgical treatment in traumatic central cord syndrome. Surg Neurol. Nov 1997;48(5):435-40; discussion 441. [Medline].

Maroon JC, Abla AA, Wilberger JI, et al. Central cord syndrome. Clin Neurosurg. 1991;37:612-21. [Medline].

Nath M, Wheeler JS Jr, Walter JS. Urologic aspects of traumatic central cord syndrome. J Am Paraplegia Soc. Jul 1993;16(3):160-4. [Medline].

Jump up ^ Behrman, Andrea, L.; Harkema, Susan J. (2007). “Physical Rehabilitation as an Agent for Recovery After Spinal Cord Injury”. Physical Medicine and Rehabilitation Clinics of North America 18 (2): 183–202. doi:10.1016/j.pmr.2007.02.002. PMID 17543768. Jump up ^ Behrman, A L; Harkema, S J. (2007). “Physical Rehabilitation as an Agent for Recovery After Spinal Cord Injury”. Physical Medicine and Rehabilitation Clinics od North America 18 (2): 183–202. doi:10.1016/j.pmr.2007.02.002. PMID 17543768. Jump up ^ Yadla, S; Klimo, J.; Harrop, J. S. (2010). “Traumatic Central Cord Syndrome: Etiology, Management, and Outcomes”. Topics in Spinal Cord Injury Rehabilitation 15 (3): 73–84. doi:10.1016/j.spinee.2006.04.006. PMID 17097539. Jump up ^ Yadla, Sanjay; Paul Klimo; James S. Harrop (2010). “Traumatic Central Cord Syndrome: Etiology, Management, and Outcomes”. Topics in Spinal Cord Injury Rehabilitation 15 (3): 73–84. doi:10.1310/sci1503-73.

Roth EJ, Lawler MH, Yarkony GM. Traumatic central cord syndrome: clinical features and functional outcomes. Arch Phys Med Rehabil. Jan 1990;71(1):18-23. [Medline].

Sidall PJ, Taylor DA, Mcclelland JM, et al. Pain report and the relationship of pain to physical factors in the first 6 months following spinal cord injury. Pain. May 1999;81(1-2):187-97. [Medline].

Tow AM, Kong KH. Central cord syndrome: functional outcome after rehabilitation. Spinal Cord. Mar 1998;36(3):156-60. [Medline].

Waters RL, Adkins RH, Sie IH, et al. Motor recovery following spinal cord injury associated with cervical spondylosis: a collaborative study. Spinal Cord. Dec 1996;34(12):711-5. [Medline].

Yamazaki T, Yanaka K, Fujita K, et al. Traumatic central cord syndrome: analysis of factors affecting the outcome. Surg Neurol. Feb 2005;63(2):95-9; discussion 99-100. [Medline].

Schneider RC, Cherry G, Pantek H (1954). “The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine”. J. Neurosurg. 11 (6): 546–77. doi:10.3171/jns.1954.11.6.0546. PMID 13222164. Jump up ^ Rich V, McCaslin E (2006). “Central Cord Syndrome in a High School Wrestler: A Case Report”. J Athl Train 41 (3): 341–4. PMC 1569555. PMID 17043705. Jump up ^ McKinley W, Santos K, Meade M, Brooke K (2007). “Incidence and Outcomes of Spinal Cord Injury Clinical Syndromes”. J Spinal Cord Med 30 (3): 215–24. PMC 2031952. PMID 17684887.

Case series

Motor unit number estimation (MUNE) was performed on both abductor pollicis brevis (APB) and extensor digitorum brevis (EDB) in 69 ATCCS patients (early vs. delayed surgical treatment: 29 vs. 35) and 42 healthy subjects. All patients were assessed by American spinal injury association and Medical Research Council scales. These examinations and disabilities of arm, shoulder, and hand © questionnaire were administered approximately 21 months after operation in 65 of these patients. Results: Preoperatively, MUNE values were lower in cervical-innervated muscles of ATCCS patients than in those of controls, and reduced motor units were observed in lumbosacral-innervated muscles in ATCCS patients with preoperative duration over 6 months ($P < 0.05$). Increased motor unit size without modification of MUNE values was found in delayed-surgical patients, whereas early-surgical patients mainly showed increased MUNE values in tested muscles between two assessments ($P < 0.05$). The postoperative follow-up analysis identified larger motor unit size and relatively fewer motor units in tested muscles, as well as higher DASH scores, in delayed-surgical patients than in early-surgical patients ($P < 0.05$).

Conclusion: ATCCS has adverse downstream effects on the LMNs distal to injury site. Surgical intervention within 2 weeks after injury in ATCCS patients may be beneficial in ameliorating dysfunction of spinal motor neurons at and distal to injury site, reducing secondary motor neuron loss, and eventually improving neurologic outcomes [10].

1)

Schneider RC, Cherry G, Pantek H. The Syndrome of Acute Central Cervical Spinal Cord Injury. J Neurosurg. 1954; 11:546–577

2)

Epstein N, Epstein JA, Benjamin V, et al. Traumatic Myelopathy in Patients With Cervical Spinal Stenosis Without Fracture or Dislocation: Methods of Diagnosis, Management, and Prognosis. Spine. 1980; 5:489–496

3) , 4)

Chen TY, Lee ST, Lui TN, et al. Efficacy of Surgical Treatment in Traumatic Central Cord Syndrome. Surg Neurol. 1997; 48:435–440

5)

SCHNEIDER RC, CHERRY G, PANTEK H. The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. J Neurosurg. 1954 Nov;11(6):546-77. PubMed PMID: 13222164.

6)

Massaro F, Lanotte M, Faccani G. Acute traumatic central cord syndrome. Acta Neurol (Napoli). 1993 Apr;15(2):97-105. PubMed PMID: 8328330.

7)

Dang T, Dong F, Fenati G, Rabiei M, Cerda M, Neeki MM. Subacute Presentation of Central Cord Syndrome Resulting from Vertebral Osteomyelitis and Discitis: A Case Report. Clin Pract Cases Emerg Med. 2020;4(2):267-271. Published 2020 Apr 23. doi:10.5811/cpcem.2019.8.44201
8)

Quencer RM, Bunge RP, Egnor M, Green BA, Puckett W, Naidich TP, Post MJ, Norenberg M. Acute traumatic central cord syndrome: MRI-pathological correlations. Neuroradiology. 1992;34(2):85-94. PubMed PMID: 1603319.
9)

Collignon F, Martin D, Lenelle J, Steneaert A. Acute traumatic central cord syndrome: magnetic resonance imaging and clinical observations. J Neurosurg. 2002;96(1 suppl):29-33.
10)

Zheng C, Yu Q, Shan X, et al. Early Surgical Decompression Ameliorates Dysfunction of Spinal Motor Neuron in Patients With Acute Traumatic Central Cord Syndrome: An Ambispective Cohort Analysis. Spine (Phila Pa 1976). 2020;45(14):E829-E838. doi:10.1097/BRS.0000000000003447