Central Cord Syndrome

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KEYWORDS

• Central cord syndrome • Spinal cord injury • Management • Pathophysiology

KEY POINTS

- Central cord syndrome (CCS) is an injury to the cervical spinal cord that causes arm greater than leg weakness, mixed modalities of sensory impairment, and bladder dysfunction.
- CCS has a good prognosis, although factors, such as older age and more severe neurologic injury at presentation, are associated with lower likelihood for neurologic recovery.
- Conservative treatment remains the most common treatment of CCS. The role and timing of surgical treatment of CCS remains controversial because there is limited evidence to support any particular treatment. Patients who have had a high-energy mechanism, evidence of spinal instability, or ongoing spinal cord compression should be considered for early surgery.

INTRODUCTION

Central cord syndrome (CCS) is most commonly caused by blunt trauma. Schneider and colleagues[1] initially described this syndrome in the 1950s, and its clinical description has changed very little since that time. CCS results in weakness of the arms with relative preservation of leg strength. Thus, CCS has been given the colloquial name of man in a barrel syndrome.[2] Mixed modalities of sensory impairment below the level of the lesion can occur. Bladder dysfunction in the form of urinary retention can also be seen in this syndrome. CCS often occurs in patients with underlying cervical stenosis and is prevalent in the elderly.[3] However, CCS occurs more frequently in younger patient populations and is more likely to be associated with cervical spine fractures or traumatic disc herniation in this group.[4–6] The management of patients with CCS is variable as there is no high-level evidence to guide treatment recommendations.[7]

The goal of this review is to provide the reader with a broad understanding of CCS from pathophysiology to management. Care must be taken to use the surgeon's training, experience, and clinical results to help select the appropriate treatments for each patient.

INCIDENCE/PREVALENCE

CCS represents about 9.0% of adult spinal cord injuries and 6.6% of pediatric spinal cord injuries.[3] The distribution of affected ages tends to be bimodal, with a young group of patients and an older group of patients that develop CCS.[5] Patients with CCS have similar neurologic presentations; but the underlying traumatic cause is heterogeneous and seems to be age related, with an age cutoff around 45 to 50 years old (depending on the study). In patients less than about 45 to 50 years old, the cause of CCS includes high-energy events: high-speed motor vehicle crashes (MVC), falls, athletic injuries/diving, gunshot wounds, and assault. In patients greater than 45 to 50 years old, the cause of CCS is more likely to be low-energy events: low-speed MVC and falls.[5] The variation in injury patterns is probably not secondary to age but due to morphologic and biomechanical differences between young versus old patients in the degree of

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cervical spondylosis, baseline cervical stenosis, and spinal flexibility.

ANATOMY AND PATHOPHYSIOLOGY

CCS was originally described as a clinical syndrome. Schneider originally proposed that mechanical compression of the spinal cord caused injury to the central region of the spinal cord, causing central cord edema and occasionally hematoma formation, leading to the eventual dysfunction of the medial portion of the lateral corticospinal tract.[1] However, more recent autopsy studies by Quencer and colleagues[8] suggest that the injury and axonal breakdown is localized to the white matter of the lateral corticospinal tracts with sparing of the central gray matter. Although previously reported as a classic component of CCS, hemorrhage is a rare finding in subsequent imaging and autopsy studies. Further pathologic findings demonstrate that the axons are diffusely injured in the lateral corticospinal tract.[8] The pathophysiologic mechanism of weakness remains poorly understood, although recent study of cadaveric specimens revealed that there does not seem to be axon loss at the level of injury but rather Wallerian degeneration of the axons adjacent to the epicenter of the injury that is the likely cause of persistent neurologic findings.[9]

MECHANISM OF INJURY

The mechanism of injury is secondary to trauma in most cases, but the subsequent injury morphologies are heterogeneous. Schneider and colleagues[10] initially described this in 1958 and subsequently has been supported by more recent studies.[5,10–26] The original proposed mechanism is secondary to cervical degenerative disease with subsequent hyperextension, which causes buckling of the ligamentum flavum (**Fig. 1**).[1] This mechanism was initially demonstrated in cadavers

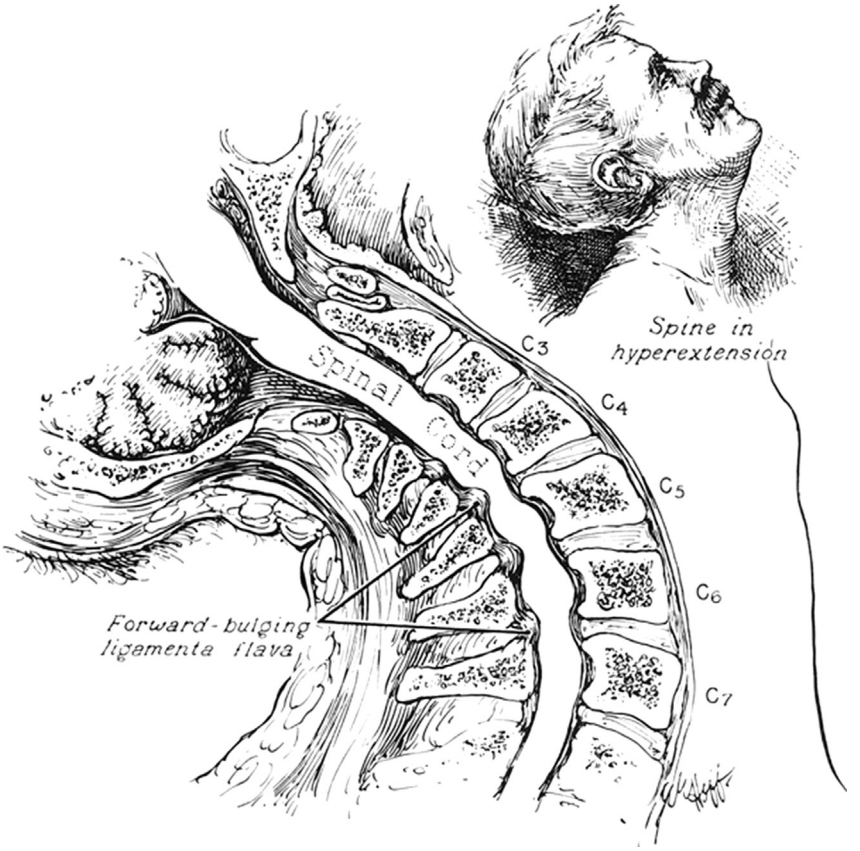

Fig. 1. In older patients, aged 45 years or greater, CCS is most often caused by low-energy cervical hyperextension in the setting of chronic cervical spondylosis and stenosis. Younger patients tend to have CCS secondary to high-energy fracture dislocation or acute disc herniations. (*From* Schneider 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;11(6):552; with permission.)