Surgical Neurology International

SNI: Spine, a supplement to Surgical Neurology International

OPEN ACCESS

For entire Editorial Board visit : http://www.surgicalneurologyint.com

Diagnosis and management of traumatic cervical central spinal cord injury: A review

Nancy E. Epstein, Renee Hollingsworth

Department of NeuroScience, Winthrop University Hospital, Mineola, NY 11501, USA

E-mail: *Nancy E. Epstein - nancy.epsteinmd@gmail.com; Renee. Hollingsworth - rhollingsworth@winthrop.org
*Corresponding author

Received: 20 December 14 Accepted: 30 December 14 Published: 07 May 15

This article may be cited as:
Epstein NE, Hollingsworth R. Diagnosis and management of traumatic cervical central spinal cord injury: A review. Surg Neurol Int 2015;6:S140-53.
Available FREE in open access from: http://www.surgicalneurologyint.com/text.asp?2015/6/5/140/156552

Abstract

Background: The classical clinical presentation, neuroradiographic features, and conservative vs. surgical management of traumatic cervical central spinal cord (CSS) injury remain controversial.

Methods: CSS injuries, occurring in approximately 9.2% of all cord injuries, are usually attributed to significant hyperextension trauma combined with congenital/acquired cervical stenosis/spondylosis. Patients typically present with greater motor deficits in the upper vs. lower extremities accompanied by patchy sensory loss. T2-weighted magnetic resonance (MR) scans usually show hyperintense T2 intramedullary signals reflecting acute edema along with ligamentous injury, while noncontrast computed tomography (CT) studies typically show no attendant bony pathology (e.g. no fracture, dislocation).

Results: CSS constitute only a small percentage of all traumatic spinal cord injuries. Aarabi *et al*. found CSS patients averaged 58.3 years of age, 83% were male and 52.4% involved accidents/falls in patients with narrowed spinal canals (average 5.6 mm); their average American Spinal Injury Association (ASIA) motor score was 63.8, and most pathology was at the C3-C4 and C4-C5 levels (71%). Surgery was performed within 24 h (9 patients), 24–48 h (10 patients), or after 48 h (23 patients). In the Brodell *et al*. study of 16,134 patients with CSS, 39.7% had surgery. In the Gu *et al*. series, those with CSS and stenosis/ossification of the posterior longitudinal ligament (OPLL) exhibited better outcomes following laminoplasty.

Conclusions: Recognizing the unique features of CSS is critical, as the clinical, neuroradiological, and management strategies (e.g. conservative vs. surgical management: early vs. late) differ from those utilized for other spinal cord trauma. Increased T2-weighted MR images best document CSS, while CT studies confirm the absence of fracture/dislocation.

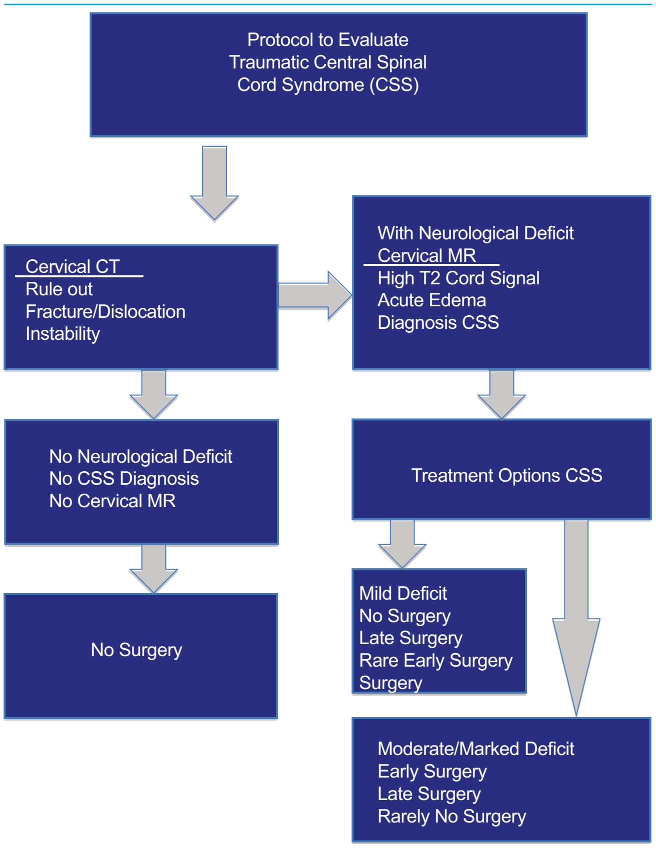
Key Words: Cervical, central cord injury, conservative therapy, spinal trauma, surgery

Access this article online
Website: www.surgicalneurologyint.com
DOI: 10.4103/2152-7806.156552
Quick Response Code:

INTRODUCTION

Traumatic cervical central cord spinal injuries (CSS) are now more readily recognized both clinically and on magnetic resonance (MR) scans. McKinley found that 9.2% of all spinal cord injuries were attributed to central spinal cord syndromes (CSSs; 77 of 839).[21] When presenting to emergency rooms, typically with greater upper vs. lower extremity neurological deficits, CSS patients first undergo computed tomography (CT) studies to rule out fracture/dislocation, and secondarily have MR examinations looking for classical central cord contusion/edema/hematomas, along with other factors (e.g. ligamentous injury, disc herniations) [Table 1]. The management of these injuries with or without surgery remains controversial [Table 1]. When surgery is chosen, there is continued debate regarding the optimal surgical approach (e.g., anterior, posterior, circumferential), and whether it should be performed early vs. late (e.g., the latter allows the patient's neurological status to plateau). Here we review the various clinical presentations, neuro-diagnostic findings, and therapeutic (nonsurgical vs. surgical) strategies for diagnosing and managing CSS [Tables 1 and 2].

Table 1: Protocol for CSS diagnosis/management



DEFINITION OF CENTRAL CORD SYNDROME

Central cord syndrome

Nowak *et al.* considered CSS to be the most common type of incomplete spinal cord injury that typically occurs following traumatic hyperextension events in older patients with underlying cervical spondylosis/stenosis [Table 2].[23] Neurological deficits typically involve the upper extremities more than the lower extremities, and are characterized by greater motor as compared with spotty/inconsistent sensory impairment. Deficits may vary from weakness in the hands/forearms with relative sensory preservation, to severe quadriparesis (with sacral sparing consistent with an incomplete injury). The optimal treatment remains controversial (nonsurgical vs. surgical performed early vs. late). Furthermore, if surgery is performed, as indicated based on preoperative MR findings of severe compression/stenosis, patients rarely fully recover.

Epidemiology of severe cervical spinal trauma in the north area of são paulo city

Santos *et al.* evaluated cervical spinal trauma (CST) data over a 10-year period, 1997–2006 [Table 2].[26] The 217 patients with CST (21.6 cases annually/1.8 cases monthly) averaged 36.75 years of age' 88% were male, and 174 (80.2%) had cervical subaxial lesions. Of the 2 patients presenting with initial American Spinal Injury Association (ASIA) Grade E scores that evolved to Grade D, one developed a central cord syndrome (CCS).

INJURY MECHANISMS AND DAMAGE AFTER ACUTE CENTRAL CORD SYNDROMES: ANIMAL MODEL

Acute central cord syndrome: Injury mechanisms

Li and Dai assessed the stress distribution to the cervical cord under different injury conditions to better understand the etiology of acute CCSs [Table 2].[20] Histopathologic studies typically revealed that CSS occurs due to predominant white matter injury. In their feline model, cervical cord injuries were simulated in hyperextension (pinch force anterior (A) or posterior (B); flexion injuries (C)), and vertical compression (pinch force anterior (D) or posterior (E)). Results were analyzed utilizing a representative cross-section analysis. They attributed upper extremity weakness to damage involving the corticospinal tracts and motor neurons in the anterior horn. Hyperpathia was due to damage to the anterior funiculus, posterior horn, and fasciculus cuneatus.

Table 2: Summary of sections

Title of Section	Summaries
INTRODUCTION	Summary: The management of these injuries with or without surgery remains controversial [Table 1]. Here we review the various clinical presentations, neuro-diagnostic findings, and therapeutic (nonsurgical vs. surgical) strategies for diagnosing and managing CSS [Tables 1 and 2]
DEFINITION OF CENTRAL CORD SYNDROME Central Cord Syndrome	Summary: Nowak *et al.* noted that central cord syndromes (CCSs) are the most common form of incomplete spinal cord injuries that typically occur following hyperextension trauma in older patients with underlying cervical spondylosis/stenosis.[23] Neurological deficits typically involve the upper moreso than the lower extremities, and are accompanied by greater motor than sensory impairment (the latter often spotty). The optimal treatment remains controversial (e.g., nonsurgical vs. surgical (early vs. late for those with MR findings of severe compression/stenosis
Epidemiology of Severe Cervical Spinal Trauma in the North Area of São Paulo City	Summary: When Santos *et al.* evaluated 217 patients with cervical spinal trauma (CST) data over a 10-year period 1997-2006, 174 (80.2%) had cervical subaxial lesions, and 1 of 2 patients presenting with initial ASIA Grade E improving to Grade D had a CCS.[26]
INJURY MECHANISMS AND DAMAGE AFTER ACUTE CENTRAL CORD SYNDROMES: ANIMAL MODEL Acute Central Cord Syndrome: Injury Mechanisms	Summary: Li and Dai's assessed the stress distribution to the cervical cord under different injury conditions to better understand the etiology of acute CCSs.[20] In a feline model, cervical cord injuries were simulated utilizing five traumatic conditions. They attributed upper extremity weakness to damage to the corticospinal tracts and motor neurons in the anterior horn, while hyperpathia was due to damage localized to the posterior horn, anterior funiculus, and fasciculus cuneatus
ETIOLOGY OF CENTRAL CORD INJURIES CSS: Traumatic Myelopathy in Patients with Stenosis Without Fracture/Dislocation	Summary: Epstein *et al.*, in 1980, published one of the first articles observing the incidence of traumatic myelopathy occurring in patients with cervical spinal stenosis without fracture or dislocation.[10] Their study looked at the subset of patients from a spinal cord injury study in which no specific lesion could be identified utilizing X-ray and CT studies (MR not yet available) to define the etiology of patient's CSS
Central Cord Injury: Pathophysiology, Management, and Outcomes	Summary: Harrop *et al.* evaluated the pathophysiology, management, and outcomes of acute traumatic central cord syndromes/injuries utilizing Medline to review articles regarding central cord injury (CSS).[15] They are the most frequent cause of incomplete traumatic cord injuries. Schneider *et al.* originally described a series of 15 patients with CSS injuries without fracture dislocation, noting the etiology to be acute disc herniations in the younger population, and hyperextension injures with stenosis in the older group. The pathoanatomic etiologies of these injuries are variously attributed to hematomyelia, vascular insult/ischemia (vertebral artery compression), or mechanical injury. On T2-weighted MR studies, hyperintense signals in the cord typically involves the lateral white matter tracts (coritcospinal); autopsy specimens further demonstrated axonal and myelin loss in these tracts
Acute Traumatic CSS; Clinical and Radiological Correlations	Summary: Miranda *et al.* correlated neurological impairment and radiological findings for 15 patients who sustained acute traumatic CSS.[22] On MR, cervical edema was typically noted, but a few patients also had hemorrhages. Notably, the length of cord edema "significantly correlated with the initial motor score," while the progressive decrease in T2-weighted hyperintensity in the 7 serial MR studies closely reflected the continued improvement of motor function in the upper extremities
Hyperextension Trauma Leads to Central Cord Syndrome	Summary: Aarabi *et al.* evaluated traumatic central cord syndromes (TCCS) characterized by incomplete spinal cord injuries.[1] In up to 50% of patients, they were due to hyperextension events in patients with congenital/degenerative spinal stenosis. Neurological findings were characterized by weakness of the upper vs. lower extremities, variable sensory loss, and bladder dysfunction, with a disproportionate loss of manual dexterity. Notably, the current level of evidence failed to demonstrate a clear correlation between the timing of surgical decompression and improvement in outcomes
Hyperextension CSS Injury of the Cervical Spine and Outcomes	Summary: Thompson *et al.* noted that traumatic hyperextension central cord syndromes (TCCS) are the most common form of an incomplete spinal cord injury especially in patients over 50 years of age with narrowed spinal canals (e.g., documented on plain X-rays and MR).[30] ASIA motor scores (AMS) of $\geq$60 on admission correlated with 80% rates of walking out of the hospital (e.g., canal diameter of $\geq$8 mm, 50% clinically improved, and 80% had functional outcomes). However, for AMS of $\leq$50, there was an 80% chance of not being ambulatory upon discharge, and the need to be transferred to spinal injury rehabilitation centers
Cervical Spine Injuries in Ocean Bathers: Wave-related Accidents	Summary: Robles evaluated spine injuries resulting from severe aquatic recreational activities; for example, hyperextension injuries resulting in CCSs.[25] The study involved 16 patients (1999 to May 2005) sustaining hyperextension injuries; they were more common for older patients (e.g., with stenosis/spondylosis), but resulted in greater neurological deficits in younger individuals
Pediatric Central Cord Injuries (Child Abuse)	Summary: Feldman *et al.* studied the mechanism of injury in five infants/toddlers who sustained cervical spinal cord injuries associated with brain injuries that resulting in paralysis and CCSs (e.g., decreased upper but with relatively preserved lower extremity function).[13] Typically MR studies most clearly documented the extent/severity of cord injury often without bony trauma (e.g., as in 4 of 5 children with cord injuries without radiological abnormalities)

Contd...

Table 2: Contd...

Title of Section	Summaries
CSS After Adequate Decompression for Cervical Spondylosis	Summary: Dickerman *et al.* presented a case in which a patient developed a CSS following a decompressive C3-C7 cervical laminectomy for cervical stenosis.[8] Following a postoperative fall at a rehabilitation facility, she developed the new-onset of a CSS. As follow-up studies showed no residual cord compression, no further surgery was warranted. Unfortunately, she failed to regain substantial neurological function
Central Cord Injury With Klippel-Feil Syndrome	Summary: Epstein *et al.* discussed the onset of traumatic myelopathy attributed to a hyperextension injury incurred when a 17-year-old male with a C2-C3 Klippel-Feil congenital fusion and stenosis was body surfing.[11] The plain X-rays and CT studies revealed no fracture/dislocation, and therefore, no surgical lesion. Despite his original quadriparesis, the patient fully recovered without surgical intervention
OTHER ETIOLOGIES OF CENTRAL CORD INJURIES CSS-related Status Epilepticus; A Case Report	Summary: In this case report, Lee *et al.* noted that a 25-year-old male sustained a CCS after status epilepticus.[18] Notably, the cervical MRI showed a hyperintense signal in the cord opposite the C1 level without other pathology, and the patient was managed nonsurgically. Theoretically the seizures produced the CSS injury due to (1) traction/stretch injuries with acute narrowing of the canal in hyperextension, (2) temporary bulging of the discs, and/or (3) transient vertebral subluxation
CSS After Total Hip Replacement	Summary: Buchowski *et al.* presented a case in which a patient sustained a CSS following a total hip replacement performed under general endotracheal anesthesia.[5] When the postoperative MR scan showed marked cervical cord compression the patient had an emergent cervical laminectomy. The authors concluded that it is important to evaluate the cervical spine and avoid cervical hyperextension during intubation in older patients who have a higher incidence of stenosis/spondylosis
Acute/subacute Spinal Epidural Hematoma Resulting in CSS	Summary: Yu *et al.* looked at the diagnosis/treatment of spinal epidural hematomas in 11 patients, 2 of who had clots in the cervical spine, and 2 at the cervico-thoracic junction.[31] Notably, three patients were quadriplegic, and one had a CCS. All four patients with cervical disease underwent emergent open-door laminoplasty accompanied by hematoma removal/drainage; all improved postoperatively
DIFFERENTIAL DIAGNOSES OF CENTRAL CORD SYNDROMES Devic's Syndrome: Initial Presentation of Systemic Lupus Erythematosus	Summary: Karim and Majithia defined Devic's syndrome/neuromyelitis optica (NMO) as an "inflammatory demyelinating disease of the central nervous system (CNS) associated with optic neuritis."[17] They reported a 22-year-old African American female, 22 weeks pregnant, who presented with quadriparesis, horizontal diplopia, temporal headache, and arthralgias. The brain, cervical and thoracic MR scans show abnormal intrinsic signals, and the diagnosis of Devic's syndrome with systemic lupus erythematosus (SLE) was established and managed with pulse IV solumedrol and plasmapharesis for 4 days, she improved
Primary Sjogren's Syndrome Involving the Central Nervous System	Summary: Alhomoud *et al.* described the clinical, laboratory, and radiological features of Primary Sjogren's syndrome (PSS) presenting within the CNS involvement.[3] For 12 females with PSS and CNS, averaging 40 years of age, 8 had myelopathy, 9 had optic neuritis, while spine MRIs demonstrated "multiple foci of hyperintensity on T2-weighted images (6 patients), and 2 showed long segments of hyperintensity in the cervical spinal cord
Multiple Sclerosis; MRI-defined Spinal Cord Involvement/Disability in MS	Summary: For MS patients, Cohen *et al.* correlated MRI-defined lesions, atrophy, and other pathology reflecting brain/spinal cord involvement with disability.[7] Cervical spinal cord contour volumes and cord T2 hyperintense lesions on MR were segmented; cervical spinal cord atrophy showed the highest correlation with physical disability in MS
RADIOGRAPHIC STUDIES CORRELATION WITH CSS Hyperintense T2 Intramedullary MR Signal Correlates with CSS	Summary: Song *et al.* assessed the clinical/prognostic value of dynamic X-rays and MR studies in 23 patients with CCS without fracture/dislocation.[28] They evaluated multiple X-rays and MR factors including prevertebral hyperintensity (HI), cord compression, intramedullary high-signal intensity (IMHSI), with/without instability.[28] Of interest, prevertebral HI was noted in 17 patients (11 with instability; 19 with cord compression). Neurological deficits also closely correlated with the level of instability (100%), and on MR, IMHSI (95%), and cord compression (87%)
Cervical Spine Trauma Clearance: Are Multidetector CT Scans Sufficient?	Summary: Chew *et al.* observed that clearing the cervical spine following trauma is critical.[6] Although the neurological exam alone may be sufficient in intact patients without neck pain, those with neck pain, altered levels of cognition/consciousness, require further assessment. Of 1004 patients (614 males averaging 47 years of age), 662 MR studies were performed for neck pain, 467 for altered mental status, and 157 for neurological signs or symptoms. The authors concluded that MR scans documented clinically irrelevant ligamentous injury when MDCT were normal (rate of 97-100%); notably, none of these patients required surgery or a halo bracing. Of interest, 39 patients required surgery (e.g., 29 anterior, 10 posterior) at some point, and 5 of the 39 with CSS underwent delayed operations
Acute CSS: Rare Anterior Spinal Artery Syndromes on CT Angiography	Summary: Zhang *et al.* studied the incidence of anterior spinal artery (ASA) rupture following 20 SCI largely attributed to blunt injuries (except 1 stab wound): 10 with CCS, four with Brown-Sequard syndromes (BSS) and six quadriplegic patients (ASIA A).[32] Computed Tomographic Angiography (CTA) revealed; no ASA in CCS/BSS patients, and only one ASA in a patient following a stab-wound
SCORING SYSTEM FOR SPINAL CORD INJURY Cervical Injury Scoring Using a Subaxial Injury Classification System	Summary: Joaquim *et al.* noted the Subaxial Injury Classification (SLIC) system and severity score facilitated the management of subaxial cervical spine injuries.[16] SLIC scores of 1-3 points were managed without surgery, SLIC scores of 4 points including those with central cord injuries with incomplete neurologic deficits were treated conservatively or operatively, while SLIC scores of 5-10 points were largely managed surgically

Contd...

Table 2: Contd...

Title of Section	Summaries
MEDICAL COMPLICATIONS OF CENTRAL CORD SYNDROME Bilateral Upper-extremity Deep Vein Thrombosis After CSS	Summary: Onmez *et al.* presented the case of a 51-year-old female with a central cord injury who developed bilateral upper-extremity DVT; she was successfully medically managed, and the bilateral thrombus regressed[24]
CONSERVATIVE VS. SURGICAL MANAGEMENT OF CSS Conservative Treatment vs. Surgery for Traumatic Central Cord Syndrome	Summary: Stevens *et al.* reviewed the recommendations for conservative treatment (59 patients) vs. surgery (67 patients), timing of surgery (16<24 h, 34>24 h, or 17 delayed 2nd admission), outcomes (Frankel Grade), and length of stay (LOS; intensive care unit) for 126 patients who sustained TCSSs between 1985 and 2006.[29] Patients were followed an average of 32 months (1-210 months). Those managed surgically improved an average of one Frankel grade vs. those treated medically, but the timing of surgery did not appear to correlate with any significant difference/improvement in neurologic outcomes (Frankel Grades). The authors concluded a "prospective randomized controlled trial is needed to definitively compare surgical versus medical management and/or early versus delayed surgical treatment in the setting of traumatic CCS"
Trends in Management of Central Cord Syndrome in 16,134 Patients	Summary: Brodell *et al.* evaluated the treatment (surgical/nonsurgical) for 16,134 patients obtained from the Nationwide Inpatient Sample (NIS) 2003-2010) following traumatic cervical CCS[4] Utilizing ICD9-CM codes, 39.7% of patients (6351) had surgery; anterior cervical decompression and fusion (19.4% ACDF), posterior cervical decompression and fusion (PCDF 7.4%), and posterior cervical decompression (6.8% PCD). Notably over the 7-year course of this study, the frequency of surgical treatment increased an average of 40% per year. Of interest, in-patient mortality (2.6%) was variously attributed to increased age, a greater number of comorbidities, lower surgical rates, treatment in more rural hospitals, and lower patient incomes
Laminoplasty vs. Conservative Treatment of CSS Due to OPLL	Summary: Gu *et al.* evaluated how patients with OPLL who sustained spinal cord injuries exhibit better outcomes following laminoplasty vs. conservative treatment.[14] Surgical patients exhibited shorter LOS, better motor/ sensory outcomes, fewer complications, increased canal diameters/reduced occupation ratios/reduced high intrinsic cord signals on T2-weighted MR images at 6 months, without loss of lordosis or range of motion vs. those managed nonsurgically[14]
Acute Traumatic CSS Treated with Open-door Expansile Cervical Laminoplasty	Summary: Uribe *et al.* evaluated the efficacy of open-door expansile cervical laminoplasty (ODECL) in the management of CSS attributed to multilevel cervical spondylotic myelopathy.[29] Over 3 years, 29 of 69 patients with cervical spinal cord injuries had acute traumatic cord syndromes (ATCCS). For the 15 undergoing expansile cervical laminoplasty (within an average of 3 days following trauma), surgery proved to be safe and effective, resulted in no postoperative deterioration, and in fact, a 71.4% (of incidence of improvement 1 ASIA grade (e.g., within 3 postoperative months)
Surgery for Acute Subaxial Traumatic CSS Without Fracture or Dislocation	Summary: Song *et al.* evaluated 22 patients with subaxial acute traumatic CCS without fracture or dislocation who required spinal surgery.[27] Pathology (dynamic cervical X-rays and MR studies) included; cervical cord compression (22 patients), instability (11 patients), disc herniations (7 patients), cervical spondylosis (11 patients), and OPLL (4 patients). None deteriorated following anterior decompression/ fusion (12 patients), posterior decompression/fusion (11 patients), and 1 re-operation was performed an average of 8 days following the injury
TIMING OF SURGERY AND OUTCOMES FOR CSS Outcomes for Acute Traumatic Central Cord Syndrome Due to Spinal Stenosis	Summary: Aarabi *et al.* evaluated 1-year outcomes of surgery performed in 42 of 59 patients with acute traumatic central cord syndromes (ATCCS) attributed to spinal stenosis.[2] For these patients, compression was most severe at the C3-4 and C4-5 levels (71%; canal measured 5.6 mm). Additionally, the maximal canal compromise (MCC) was 50.5%, and the length of parenchymal damage (T2-weighted MR) was typically 29.4 mm. The intervals between injury and surgery varied from 24 h (9 patients) to 24-48 h (10 patients), and >48 h (23 patients). Major factors determining the quality of long-term outcomes included admission AMS, mid-sagittal canal diameter, (% of MCC), the length of the intrinsic high cord signal (MR), and age
Urgency of Early Surgical Decompression in Acute CSS with Stenosis	Summary: Lenehan *et al.* evaluated whether urgent surgical decompression facilitates neurologic recovery following acute central cord injuries without attendant fracture/dislocation.[19] They found that early surgery "resulted in a 6.31 point greater improvement in total motor score than did the late surgery group." They concluded that it is "reasonable and safe to consider early surgical decompression in patients with profound neurologic deficit (ASIA=C) and persistent spinal cord compression due to developmental cervical spinal canal stenosis without fracture or instability." Alternatively, for those with lesser deficits (ASIA=D) initial treatment may be nonsurgical, but surgery may be considered at a later date
Early Surgery for Spinal Cord Injury, Especially for Incomplete CSS	Summary: Fehlings *et al.* utilized a 20-question survey of 971 spinal surgeons (orthopedic and neurosurgeons) worldwide regarding the optimal timing of surgery (early 24 h vs. later decompression) for acute SCI.[12] They recommended that both complete and incomplete cervical SCI be decompressed within 6 h (46.2% and 72.9%, respectively), except for patients with CCSs
Incidence and Outcomes of Acute CSS and Other Clinical Syndromes	Summary: McKinley *et al.* examined outcomes for patients sustaining spinal cord injury associated with CSS, Brown-Sequard (BSS), anterior cord (ACS), posterior cord (PCS), cauda equina (CES), and conus medullaris (CMS) syndromes. They evaluated 175 patients (20.9%) who had SCI clinical syndromes; CCS (44%) was most commonly noted; followed by CES (25.1%), and BSS (17.1%). Of interest, CSS patients were typically in the older age group and exhibited the poorest functional levels/outcomes[21]

Contd...

Table 2: Contd...

Title of Section	Summaries
Factors Predicting Motor Recovery and Outcome After Traumatic CSS	Summary: Dvorak *et al.* prospectively assessed the improvement in AMS (at 72 h and follow-up), functional status (FIM), and generic health-related quality of life (HRQoL) for patients who sustained traumatic CSS.[9] AMS improved from 58.7 to 92.3; 81% of patients exhibited residual incontinence, and 86% ambulated without assistance
SUMMARY	Although the treatment strategies for CSS remain controversial, establishing the diagnosis of CSS has been greatly facilitated with the ready availability of CT and MR studies [Tables 1 and 2]. Decisions regarding nonsurgical vs. surgical options depend upon multiple factors; the severity of the neurological deficit, whether the deficit has spontaneous improved or it is has plateaued, and the severity of cord compromise

ETIOLOGY OF CENTRAL CORD INJURIES

CSS: Traumatic myelopathy in patients with stenosis without fracture/dislocation

Epstein *et al*., in 1980, published one of the first articles observing the incidence of traumatic myelopathy occurring in patients with cervical spinal stenosis without fracture or dislocation [Table 2].[10] Their study looked at the subset of patients from a spinal cord injury (SCI) study in which no specific lesion could be identified utilizing X-ray and CT studies (MR not yet available) to define the etiology of patient's CSS [Figures 1-6].

Central cord injury: Pathophysiology, management, and outcomes

Harrop *et al*. utilized Medline to evaluate the pathophysiology, management, and outcomes of acute traumatic central cord syndromes (ATCCSs)/injuries (CSS) [Table 2].[15] ATCCS are the most frequent cause of incomplete traumatic cord injuries. Schneider *et al*. originally defined this syndrome in 1954, citing the predominant hyperextension mechanism in the absence of fracture/dislocation following various traumatic events (e.g. motor vehicle accidents, falls, and diving injuries). His definition included the greater upper vs. lower extremity motor deficits, the presence of bladder dysfunction, and variable sensory below the locus of the trauma. His study included a review of 15 patients who presented with minor ormajor cervical injuries, in patients ranging in age from 18 to 85. Most injuries were attributed to acute mechanical compression attributed to hyperextension, resulting in the "pincer compression" of the cord between the spondylotic osteophytes anteriorly and acute inward buckling of the yellow ligament posteriorly. However, in younger patients, their deficits could be attributed to acute disc herniations without the stenotic findings.

Pathoanatomic etiologies of ATCCS injuries were variously attributed to: Hematomyelia (worse injury/poor prognostic finding), vascular insult/ischemia (vertebral compression), or mechanical injury. T2-weighted MR studies typically demonstrate hyperintense signals in the cord tissues involving the lateral white matter tracts (corticospinal) with accompanying axonal and myelin loss (the latter confirmed on autopsy).

Acute traumatic CSS; clinical and radiological correlations

Miranda *et al*. correlated neurological impairment and radiological findings for 15 patients who sustained acute traumatic CSS [Table 2].[22] Global motor scores were determined for the upper extremities on admission, and an average of 16 months later (6 months to 4 years). Radiographic studies included; plain films, cervical CT and cervical MR; seven had serial MR scans performed. On MR, cervical edema was typically noted, but a few patients also had hemorrhages. Notably, the length of cord edema "significantly correlated with the initial motor score," while the progressive decrease in T2-weighted hyperintensity in the 7 serial MR studies closely reflected the continued improvement of motor function in the upper extremities.

Hyperextension trauma leads to central cord syndrome

Aarabi *et al*. evaluated traumatic central cord syndromes (TCCSs) characterized by incomplete spinal cord injuries [Table 2].[1] In up to 50% of patients, they are due to hyperextension events in those with underlying congenital/degenerative spinal stenosis. Patients exhibited disproportionate weakness of the upper vs. lower extremities, accompanied by variable sensory loss, and bladder dysfunction; over half demonstrated spontaneous improvement of motor deficits, but showed a continued lack of manual dexterity. Notably, the current level of evidence failed to demonstrate a clear correlation between the timing of surgical decompression and improvement in outcomes.

Hyperextension CSS injury of the cervical spine and outcomes

Thompson *et al*. noted that TCCS is the most common form of an incomplete SCI [Table 2].[30] It is most frequently encountered in patients over the age of 50 with narrow spinal canals, and is typically correlated with acute hyperextension trauma. Between 2004 and 2008, patients with traumatic hyperextension injuries resulting in CCSs were analyzed utilizing plain radiographs and MR studies.

Figure 1: Midline sagittal stir T2-weighted MR image following CSS. following a hyperextension injury in a 75-year-old male, this midline sagittal stir T2-weighted MR image readily demonstrated marked intrinsic signal changes within the spinal cord opposite the C4 and C5 vertebral bodies. Note the maximal cord compression opposite the disc space of C4-C5, followed by C5-C6. At the C6-C7 level, there is thecal sac intrusion without significant cord compression

Figure 2: Midline sagittal soft-tissue window CT study following CSS. In the same 75-year-old male, following a hyperextension injury, the midline sagittal soft-tissue window CT study confirmed the absence of fracture or dislocation following the hyperextension traumatic event that led to the CSS. Note the presence of spinal stenosis/spondyosis documented by ventral osteophytes at multiple levels (e.g., most marked at C4-C5- and C5-C6, followed by C6-C7 and C3-C4) and the accompanying dorsolateral shingling of the C4, C5, C6 laminae resulting in the greatest canal compromise at the C4-C5 and C5-C6 levels

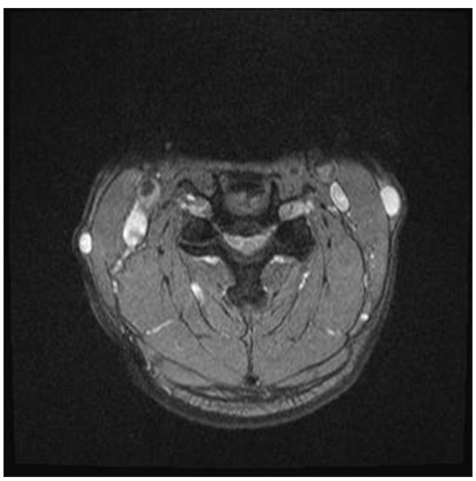


Figure 3: Axial T2-weighted STIR MR image at the C4-C5 disc space following the CSS. On the axial T2 weighted STIR MR image at the C4-C5 disc space level following the CSS one can see the marked reduction in the anterior-posterior diameter of the spinal canal to 5--6 mm secondary to ventral osteophyte formation vs. disc, and dorsal laminar shingling. On this STIR study, there also appears to be an increased signal in the cord at this level

Figure 4: Axial CT image at the C4-C5 disc space following the CSS. This axial noncontrast CT study demonstrated marked stenosis/spondylosis at the C4-C5 level narrowing down the spinal canal to 5–6 mm. Ventrally the hyperdense soft-tissue appears more spondylotic vs. soft disc, while posteriorly the compression was attributed to inward shingling of the C5 lamina. Certainly, both the MR and CT studies help document why this patient, with such severe stenosis at the C4-C5 level, was so susceptible to the CSS cord injury

ASIA motor scores (AMS) of ≥60 on admission correlated with 80% rates of walking out of the hospital. For those with cervical spinal canal diameters of ≥8 mm, 50% demonstrated clinical improvement and 80% exhibited functional outcomes. Alternatively, AMS of ≤50 correlated with an 80% chance of not being able to ambulate at the time of discharge, and correlated with having to be transferred to spinal injury rehabilitation centers.

Cervical spine injuries in ocean bathers: Wave-related accidents

Robles evaluated spine injuries resulting from severe aquatic recreational activities; for example, hyperextension injuries resulting in CCSs [Table 2].[25] His study involved 16 patients (1999 to May 2005) who sustained hyperextension injuries; they were more common in older patients (e.g., with stenosis/spondylosis), but resulted in greater neurological deficits in younger individuals.

Pediatric central cord injuries (child abuse)

Feldman *et al.* studied the mechanism of injury in five infants/toddlers who sustained cervical spinal cord

Figure 5: Ossification of the posterior longitudinal ligament (OPLL) contributing to cervical Stenosis. Patient with severe OPLL and markedly narrowed cervical spinal canals are very susceptible to CSS injury following even minor hyperextension traumatic events. Note the segmental/continuous forms of OPLL extending from the C4-C7 vertebral levels that lead to a reduced AP diameter of the canal to less than 5 mm at multiple foci

injuries (CSSs) associated with brain injuries resulting in paralysis [Table 2].[13] Their deficits were characterized by decreased upper, but relatively unimpeded lower extremity function. Typically, the performance of cervical MR studies was delayed by life support efforts. When obtained, MR scans very clearly documented the extent/severity of SCI that often occurred without bony trauma. Of interest, in this series, four of five children had cord injuries without radiological abnormalities.

CSS after adequate decompression for cervical spondylosis

Dickerman *et al.* presented a case in which a patient developed a CSS following a decompressive C3-C7 cervical laminectomy for cervical stenosis [Table 2].[8] Postoperatively, the patient's spasticity/gait improved, and she was discharged to an inpatient rehabilitation center to treat her residual upper extremity weakness. Following a fall, however, she developed the new onset of a CSS. However, since her studies showed adequate canal decompression, no further surgery was performed, and she failed to demonstrate any substantial improvement.

Central cord injury with Klippel–Feil syndrome

Epstein *et al.* discussed the onset of traumatic myelopathy attributed to a hyperextension injury incurred when a 17-year-old male with a C2-C3 Klippel–Feil (KL) congenital fusion/stenosis was body surfing [Table 2].[11] The plain X-rays and CT studies revealed no fracture/dislocation, and therefore, no surgical lesion. Despite his original quadriparesis, the patient fully recovered without surgical intervention.

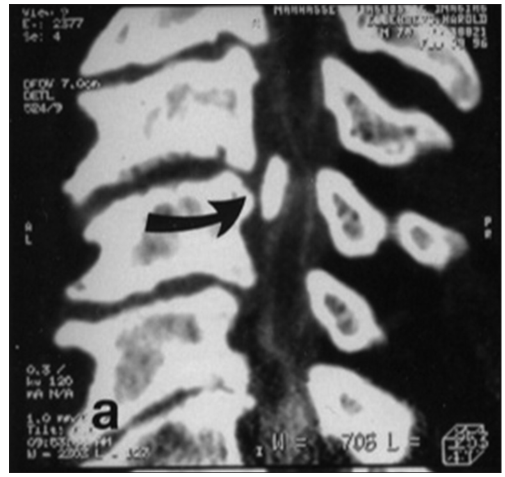


Figure 6: Midline sagittal Myelo-CT documenting early OPLL compressing the cord at the C3-C4 and C4-C5 Levels. Patient with marked cord compression due to early OPLL are also extremely susceptible to CSS injury following even minor hyperextension trauma. Early OPLL is defined by hypertrophied posterior longitudinal ligament (HPLL) combined with progressive punctate ossification centers. In this midline sagittal Myelo-CT study, marked ventral compression at the C3-C4 level is attribute to a coalescence of punctate ossification centers within HPLL (e.g., pearl of ossification). At the C4-C5 level below, HPLL itself without such ossification contributes to marked cord compromise

OTHER ETIOLOGIES OF CENTRAL CORD INJURIES

CSS-related status epilepticus: A case report

In this case report, Lee *et al.* noted that a 25-year-old male sustained a CCS after status epilepticus [Table 2].[18] Notably, the cervical MRI showed a hyperintense signal in the cord opposite the C1 level without other pathology; the patient, managed nonsurgically, improved on the AMS and regained bladder function. The CSS injury was attributed to muscle contractions occurring during generalized seizures. Theoretically the seizures produced (i) traction/stretch injuries with acute narrowing of the canal in hyperextension, (ii) temporary bulging of the discs, and/or (iii) transient vertebral subluxation.

CSS after total hip replacement

Buchowski *et al.* presented a case in which a patient sustained a CSS following a total hip replacement performed under general endotracheal anesthesia [Table 2].[5] When the postoperative MR scan showed marked cervical cord compression, the patient had an emergent cervical laminectomy. The authors concluded that it is important to evaluate the cervical spine and avoid cervical hyperextension during intubation in older patients who have a higher incidence of stenosis/spondylosis.

Acute/subacute spinal epidural hematoma resulting in CSS

Yu *et al*. looked at the diagnosis/treatment of spinal epidural hematomas in 11 patients; 2 had clots in the cervical spine, and 2 at the cervico-thoracic junction [Table 2].[31] Notably, three patients were quadriplegic, and one had a CCS. MRI studies showed equal intensity/hyperintensity on T1-weighted images, and mixed hyperintensity on T2-weighted images. All four patients underwent emergent open-door laminoplasty accompanied by hematoma removal/drainage; all improved postoperatively. The authors concluded that since the physical and MRI findings correlated with acute/subacute spinal epidural hematomas, emergent surgical intervention to facilitate maximal recovery was warranted.

DIFFERENTIAL DIAGNOSES OF CENTRAL CORD SYNDROMES

Devic's syndrome: Initial presentation of systemic lupus erythematosus

Karim and Majithia defined Devic's syndrome/neuromyelitis optica (NMO) as an "inflammatory demyelinating disease of the central nervous system (CNS) associated with optic neuritis."[17] In this report, a 22-year-old African American female, 22 weeks pregnant, presented with the onset of quadriparesis, horizontal diplopia, temporal headache, and arthralgias [Table 2]. She exhibited a discoid rash behind the left ear, with motor function of 3/5 in the upper, and 0/5 in the lower extremities. MR scans showed an abnormal signal within the brain, cervical, and thoracic spine. The diagnosis of Devic's syndrome likely associated with systemic lupus erythematosus (SLE) was established; after treatment with pulse IV solumedrol and plasmapharesis for 4 days, she improved.

Primary sjogren's syndrome involving the central nervous system

Alhomoud *et al*. described the clinical, laboratory, and radiological features of primary Sjogren's syndrome (PSS) presenting within the CNS [Table 2].[3] In this retrospective analysis of 12 females with PSS and CNS, patients averaged 40 years of age (range 16–58 years), 8 (66%) were myelopathic, 9 (75%) had optic neuritis, 7 (58%) had positive immunological tests (anti-Sjogren's syndrome A and anti-Sjogren's syndrome B), and 11 had positive salivary gland biopsies demonstrating inflammatory cell infiltrates. Furthermore, the spine MRIs demonstrated "multiple foci of hyperintensity on T2-weighted image in six patients, and long segments of hyperintensity in the cervical spinal cord in two patients."

Multiple sclerosis: Mri-defined spinal cord involvement/disability in ms

For multiple sclerosis (MS) patients, Cohen *et al*. correlated MRI-defined lesions, atrophy, and other pathology reflecting brain/spinal cord involvement with disability [Table 2].[7] This study involved performing 3T MRIs of the brain/spinal cord in 21 MS patients (18 relapsing-remitting, 1 secondary progressive, 1 primary progressive, and 1 clinically isolated syndrome). Spinal cord contour volumes and cord T2-hyperintense lesions were segmented. They determined that with MR, "only upper cervical spinal cord volumes significantly correlated with Expanded Disability Status Scale scores ($r = -0.515$, $P = 0.020$)." Therefore, cervical spinal cord atrophy showed the highest correlation with physical disability in MS.

RADIOGRAPHIC STUDIES CORRELATION WITH CSS

Hyperintense T2 intramedullary MR signal correlates with CSS

Song *et al*. assessed the clinical/prognostic value of dynamic X-rays and MR studies in 23 patients with CCS without fracture/dislocation [Table 2].[28] They evaluated multiple X-rays and MR factors including; prevertebral hyperintensity (HI), cord compression, intramedullary high-signal intensity (IMHSI), with/without instability.[28] Patients' outcomes were correlated with their preoperative and postoperative ASIA scores. Surgical intervention included 12 anterior decompressions/fusions (with 1- or 2-level lesions), and 11 posterior decompressions (with multilevel lesions). Of interest, prevertebral HI was noted in 17 patients (11 with instability; 19 with cord compression). Neurological deficits also closely correlated with the level of instability (100%), and on MR, IMHSI (95%), and cord compression (87%).

Cervical spine trauma clearance: Are multidetector CT scans sufficient?

Chew *et al*. observed that clearing the cervical spine following trauma is critical.[6] Although the neurological examination alone may be sufficient in intact patients without neck pain, those with neck pain or altered levels of cognition/consciousness require further assessment [Table 2]. Here, the optimal diagnostic management becomes controversial. Do you first order a multidetector CT (MDCT) scan alone, or additionally request an MR to rule out ligamentous/other injuries? In this retrospective analysis from a Level I trauma center, the authors evaluated all patients admitted from January 2004 to June 2011 who underwent MDCT scans (showing no acute traumatic injury) plus cervical MRIs obtained "during the same hospitalization." Multiple variables were studied; patient demographics, the type of injury, Glasgow Coma Scale (GCS), when/why an MRI was performed, and whether cervical spine surgery was performed. Of 1004 patients (614 males averaging 47 years of age), 662 MR studies were performed for neck pain, 467 for altered mental status, and 157 for neurological signs or symptoms. MR studies were normal in 645 patients or showed only ligamentous injury in 125 patients; the remainder exhibited only mild degenerative changes. Interestingly, 52.8% were cleared on a clinical basis only (29 on clinical exam and 39 with flexion extension X-rays). The authors concluded that MR scans documented clinically irrelevant ligamentous injury when MDCT were normal (rate of 97–100%); notably, none of these patients required surgery or halo bracing. Of interest, 39 patients required surgery (e.g., 29 anterior, 10 posterior) at some point, and 5 of the 39 with CSS underwent delayed operations.

Acute CSS: Rare anterior spinal artery syndromes on CT angiography

Zhang *et al*. observed that with acute traumatic SCI significant vascular damage may be associated with spinal contusions, fractures, and dislocations [Table 2].[33] This study specifically evaluated the incidence of anterior spinal artery (ASA) rupture following 20 SCI largely attributed to blunt injuries (except 1 stab wound). Neurological deficits included; 10 with CCS, 4 with Brown–Sequard syndrome (BSS), and 6 with quadriplegia (ASIA A). Computed tomographic angiography (CTA) looking for ASA rupture revealed none in the CCS/BSS patients, and only one ASA in a patient following a stab-wound. The authors concluded CTA readily documents ASA following SCI, but that ASA rupture is rare following acute blunt cervical SCI.

SCORING SYSTEM FOR SPINAL CORD INJURY

Cervical injury scoring using a subaxial injury classification system

Joaquim *et al*. noted that the subaxial injury classification (SLIC) system and severity score facilitates the management of subaxial cervical spine injuries [Table 2].[16] Utilizing a PubMed database (2007–2014), they correlated the scoring of spinal cord injuries and their ultimate outcomes (e.g. system score). Those with an SLIC score of 1–3 points with spinous process, laminar, or small facet fractures, or those with compression/burst fractures but who were neurologically intact were all conservatively managed (e.g. without surgery). Those with SLIC of 4 points had either incomplete spinal cord injuries (e.g., CCS or compression injuries) with incomplete neurologic deficits, or burst fractures with complete neurologic deficits; these patients were either managed conservatively or with surgery. However, those with SLIC of 5–10 points with distraction/rotational injuries, traumatic disc herniations (with deficits), and burst fractures with incomplete neurologic deficits were largely managed surgically.

MEDICAL COMPLICATIONS OF CENTRAL CORD SYNDROME

Bilateral upper-extremity deep vein thrombosis after CSS

Onmez *et al*. evaluated the incidence of deep vein thrombosis (DVT) involving the upper extremities in patients who sustained SCI characterized by CCS [Table 2].[24] In this case study, a 51-year-old female with a central cord injury developed bilateral upper-extremity DVT; she was successfully medically managed, and the bilateral thrombus regressed. The authors concluded that with this type of SCI, DVT may arise in the upper extremities, and, therefore, prophylactic treatment should be considered.

CONSERVATIVE VS. SURGICAL MANAGEMENT OF CSS

Conservative treatment vs. Surgery for traumatic central cord syndrome

Stevens *et al*. reviewed the recommendations for conservative treatment (59 patients) vs. surgery (67 patients), timing of surgery (<24 h, >24 h, or delayed second admission), outcomes (Frankel Grade), and length of stay (LOS; intensive care unit) for 126 patients who sustained TCSSs between 1985 and 2006 [Tables 1 and 2].[29] Surgery was performed in 16 patients within 24 h, in 34 patients 24 h after the injury (average 6.7 days later), while 17 had surgery during second hospital admissions (mean interval of 137 days between injury and surgery (range 3–209 days)). Patients were followed an average of 32 months (1–210 months). Although those managed surgically improved an average of one Frankel grade vs. those treated medically, the timing of surgery itself did not correlate with any significant difference/improvement in neurologic outcomes (Frankel Grades). They did, however, observe a "trend toward decreased LOS" particularly for those undergoing surgery during the first hospital stay. Despite similar complication rates for both the surgical/non surgical groups, a "trend toward fewer complications and deaths" for those undergoing surgery within the first 24 h or during the first hospital stay" was observed. The authors concluded that a "prospective randomized controlled trial is needed to definitively compare surgical versus medical management and/or early versus delayed surgical treatment in the setting of traumatic CCS."

Trends in management of central cord syndrome in 16,134 patients

Brodell *et al*. evaluated the treatment (surgical vs. nonsurgical) for 16,134 patients following traumatic cervical CCSs [Table 2].[4] They utilized a retrospective cohort from the Nationwide Inpatient Sample (NIS) 2003–2010 database, and assessed clinical variables, mortality in the hospital, and surgical procedures performed. Utilizing ICD9-CM codes, 39.7% of patients (6351) had surgery; anterior cervical decompression and fusion (ACDF; 19.4%), posterior cervical decompression and fusion (PCDF; 7.4%), and posterior cervical decompression (PCD 6.8%) alone [Figures 7-9]. Notably over the 7-year course of this study, the frequency of surgical treatment increased an average of 40% per year. Of interest, in-patient mortality (2.6%) was variously attributed to increased age, a greater number of comorbidities, lower surgical rates, treatment in more rural hospitals, and lower patient incomes. Notably, surgery for CSS was more common performed in the south, and was more frequently performed at larger institutions.

Laminoplasty vs. Conservative treatment of CSS due to OPLL

Gu *et al*. evaluated how patients who sustained acute spinal cord injuries following minor trauma (falls, motor vehicle accidents, sports) with underlying ossification of the posterior longitudinal ligament (OPLL: Mostly with mixed and segmental variants) were managed conservatively (C group; 29 patients) or surgically (L group – lamionplasty; 31 patients) [Table 2].[14] Those undergoing laminoplasty exhibited shorter lengths of stay (LOS), better motor/sensory outcomes, fewer complications, and had increased canal diameters/reduced occupation ratios/reduced high intrinsic cord signals on T2-weighted MR images at 6 months vs. those managed conservatively. Nevertheless, the extent of lordosis and range of motion (ROM) were the same for both groups. The authors concluded laminoplasty offers OPLL patients with SCI following minor trauma (e.g. often with CSS) more satisfactory outcomes.

Acute traumatic CSS treated with open-door expansile cervical laminoplasty

Uribe *et al*. evaluated the efficacy of open-door expansile cervical laminoplasty (ODECL) in the management of CSS attributed to multilevel cervical spondylotic myelopathy [Table 2].[29] Over 3 years, 29 of 69 cervical spinal cord injuries were due to central cord syndromes (ATCCS) secondary to hyperextension injuries attributed to cervical spondylosis/stenosis without fracture/dislocation, or instability. Expansile cervical laminoplasty was performed in 15 patients (averaging 56 years of age) typically within 3 days of injury; it proved not only to be safe and effective, resulting in no postoperative neurological deterioration, but also 71.4% improved 1 ASIA grade within 3 postoperative

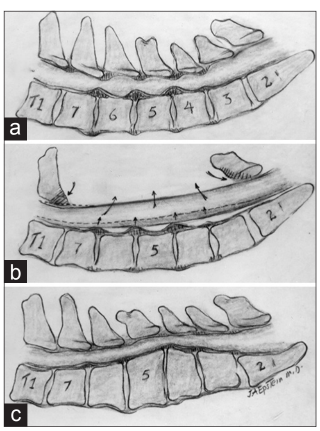


Figure 7: Patients with cervical spinal stenosis are susceptible to CSS and may require posterior or anterior decompressive surgery (a) This midline lateral illustration of cervical stenosis demonstrates diffuse ventral spondylotic intrusions at the C4-C5, C5-C6, and C6-C7 levels and dorsolateral compression attributed to multilevel inward buckling of hypertrophied/ossified yellow ligament (OYL) and inward shingling of the lamina. (b) When the cervical lordotic curvature is adequately preserved, a dorsal decompression with/without fusion may sufficiently decompress the spinal cord. Illustrated here is a laminectomy involving resection of the C3-C7 laminae. (c) In this instance, there reversal of the lordosis with substantial kyphosis warranting a multilevel anterior decompressive procedure (e.g., corpectomies)

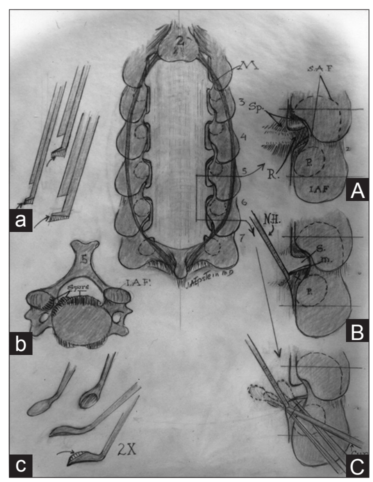


Figure 8: Multilevel laminectomy with medial facetectomy/foraminotomy. CENTRAL IMAGE: This illustrates the posterior view of a C3-C7 laminectomy accompanied by medial facetectomy/foraminotomy at the C23, C34, C45, C56, C67 C7T1 levels. (a) Illustration of filed-down Kerrison Rongeur utilized to perform medial facetectomy/foraminotomy and resect hypertrophied/ossified yellow ligament to decompress the nerve roots in the foramina. (b) Axial illustration of the ventral/foraminal osteophytes, which often require resection with a down-biting curette. (c) Illustration of the down-biting curette employed to remove foraminal disc/spur/osteophytes. (A) Illustration of nerve root exiting into the foramen following performance of a medial facetectomy/foraminotomy. (B) In order to resect foraminal disc or spur, utilizing a micro nerve hook allows for mobilization of the foraminally exiting nerve root. (C) The down-biting curette is then introduced ventral to the root and lateral aspect of the thecal sac to remove disc/spur

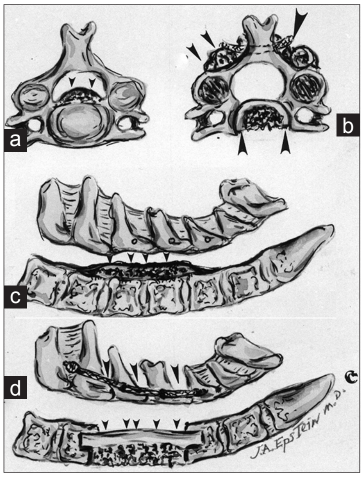


Figure 9: Illustrations of multilevel OPLL before after multilevel anterior cervical corpectomy/fusion.(a) Axial image of ventral OPLL filling the midline spinal canal (small arrows).(b) Axial image following anterior corpectomy and fusion (ventral graft in place) with posterior wires/spinous process fusion (single arrow) and accompanying bone graft (double arrows).(c) Midline sagittal illustration of multilevel continuous OPLL starting behind the inferior C4 and extending to the superior aspect of the C7 vertebral body.(d) Illustration of an anterior fibular strut graft extending from C4-C7 with posterior wiring and fusion involving the same levels

months. Alternatively, 14 other CSS patients without radiographic evidence of sagittal instability were managed conservatively (e.g. without surgery).

Surgery for acute subaxial traumatic CSS without fracture or dislocation

Song *et al*. evaluated 22 patients with subaxial acute traumatic CCS without fracture or dislocation who required spinal surgery [Table 2].[27] The 13 males and 9 females averaged 61.2 years of age, and had mostly sustained falls (68%) or motor vehicle accidents (32%). Pathology, documented on dynamic cervical X-rays and MR studies included cervical cord compression (22 patients), instability (11 patients), disc herniations (7 patients), cervical spondylosis (11 patients), and OPLL (4 patients). Anterior decompression/fusion (12 patients; 1- or 2-level lesions), posterior decompression/fusion (11 patients; multilevel lesions), and 1 re-operation were performed an average of 8 days following the injury (range 1–37 days). Postoperatively, all patients improved, and none exhibited further deterioration.

TIMING OF SURGERY AND OUTCOMES FOR CSS

Outcomes for acute traumatic central cord syndrome due to spinal stenosis

Aarabi *et al*. evaluated multiple variables impacting the 1-year postoperative outcomes for patients sustaining ATCCSs attributed to spinal stenosis [Table 2].[2] Variables studied included clinical data, an evaluation of injury severity measures (e.g. imaging studies), AMS, functional independence measure (FIM), manual dexterity, and dysesthetic pain scores. Over 100 months, out of 211 patients with ATCCS, 59 with spinal stenosis underwent surgical decompressions; 5 died, 2 were lost to follow-up, 10 were not eligible for the study, while the remaining 42 were followed for at least 12 months. Patients averaged 58.3 years of age, 83% were male, and 52.4% of accidents involved falls. The mean admission AMS was 63.8 (upper extremities 25.8, lower extremities 39.8). Most frequently, cord compression was documented at the C3-4 and C4-5 (71%) levels, and the canal typically measured only 5.6 mm. Additionally, the following measurements were obtained; the extent of maximum canal compromise (MCC) of 50.5%, maximum spinal cord compression of 16.5%, and length of parenchymal damage (T2-weighted MR) of 29.4 mm. The intervals between injury and surgery varied; within 24 h (9 patients), 24–48 h (10 patients), and >48 h (23 patients). One year postoperatively, the mean AMS was 94.1 (upper extremities 45.7, lower extremities 47.6), FIM (111.1), manual dexterity (64.4% of baseline), and pain (3.5). The major factors determining the quality of long-term outcomes included admission AMS, mid-sagittal canal diameter, percentage of MCC, length of intrinsic high cord signal (MR), and the patient's age.

Urgency of early surgical decompression in acute CSS with stenosis

Lenehan *et al*. evaluated whether urgent surgical decompression facilitates neurologic recovery following acute central cord injuries without attendant fracture/dislocation [Tables 1 and 2].[19] They reviewed whether the following variables impacted outcome; demographic data, mechanism of injury, comorbidities, neurologic status, and surgery. Outcomes (obtained on admission, discharge, 6 and 12 months later) utilized the following measures: AMS, ASIA Grade, FIM Score, SF-36, Sphincter Disturbance, and Ambulatory status. They concluded that early surgery "resulted in a 6.31 point greater improvement in total motor score than did the late surgery group." At 6 months, early surgery (within 24 h; 7.79 U more improvement in FIM total Score) showed greater improvement in the ASIA Grade vs. late surgery; this increased further at 12 months. The authors concluded that it is "reasonable and safe to consider early surgical decompression in patients with profound neurologic deficit (ASIA = C) and persistent spinal cord compression due to developmental cervical spinal canal stenosis without fracture or instability." Alternatively, patients with lesser deficits (ASIA = D) may be initially followed conservatively, reserving surgery for later if indicated.

Early surgery for spinal cord injury, especially for incomplete CSS

Fehlings *et al*. evaluated the optimal timing of surgery (early vs. later decompression) for acute SCI [Table 2].[12] They utilized a 20-question survey evaluated by 971 orthopedic and neurosurgical spine surgeons worldwide. They found that spine surgeons recommended that both complete and incomplete cervical SCI should be decompressed within 6 h (46.2% and 72.9%, respectively). "In almost every clinical scenario, with the exception of CCS, the majority of respondents (≥80%) preferred to decompress the spinal cord within 24 h"; in fact, many favored decompression within 12 h especially for patients with incomplete deficits except for those with CCSs [Table 1].

Incidence and outcomes of acute CSS and other clinical syndromes

McKinley *et al*. examined the factors correlating with outcomes for patients sustaining SCI associated with CSS, BSS, anterior cord syndrome (ACS), posterior cord syndrome (PCS), cauda equina syndrome (CES), and conus medullaris syndrome (CMS) [Table 2].[21] They evaluated 175 patients who had SCI clinical syndromes; CSS (44%) was most commonly noted, followed by CES (25.1%), and BSS (17.1%). Notably, those with acute CSS were typically in the older age group and exhibited the poorest functional outcomes.

Factors predicting motor recovery and outcome after traumatic CSS

For patients who sustained traumatic CSS, Dvorak *et al*. prospectively assessed improvement in AMS (at 72 h and follow-up), generic health-related quality of life (HRQoL), and functional status [Table 2].[9] AMS improved from 58.7 to 92.3; although 81% of patients exhibited residual incontinence, 86% ambulated without assistance. Better functional status (FIM) and improved outcomes (HRQoL) reflected higher initial AMS scores, higher levels of formal education, the lack of comorbidities, and younger age.

SUMMARY

Although the treatment strategies for CSS remain controversial, establishing the diagnosis of CSS has been greatly facilitated with the ready availability of CT and MR studies [Tables 1 and 2]. After first obtaining negative CT scans (demonstrating no fracture, dislocation, or instability), MR studies now readily demonstrate hyperintense T2 signals in the cord indicative of edema and more rarely hematomyelia with or without ongoing neural compression [Table 1]. Decisions regarding nonsurgical vs. surgical options depend upon multiple factors; the severity of the neurological deficit, whether the deficit has spontaneous improved or it is has plateaued, and the severity of cord compromise. For those with mild deficits or where deficits have substantially improved/plateaued, without significant cord compromise, nonsurgical options may be chosen, followed choices for delayed surgery or rarely, acute surgery. Alternatively, for those with moderate/severe deficits who have not improved in the presence of marked cord compromise, operative choices typically factor acute surgery, followed by delayed surgery, and, only rarely, no surgery (e.g. other extenuating factors).

REFERENCES

1. Aarabi B, Koltz M, Ibrahimi D. Hyperextension cervical spine injuries and traumatic central cord syndrome. Neurosurg Focus 2008;25:E9.
2. Aarabi B, Alexander M, Mirvis SE, Shanmuganathan K, Chesler D, Maulucci C, *et al*. Predictors of outcome in acute traumatic central cord syndrome due to spinal stenosis. J Neurosurg Spine 2011;14:122-30.
3. Alhomoud IA, Bohlega SA, Alkawi MZ, Alsemari AM, Omer SM, Alsenani FM. Primary Sjogren's syndrome with central nervous system involvement. Saudi Med J 2009;30:1067-72.
4. Brodell DW, Jain A, Elfar JC, Mesfin A. National Trends in the Management of Central Cord Syndrome: An Analysis of 16,134 patients. Spine J 2015;15:435-42.
5. Buchowski JM, Kebaish KM, Suk KS, Kostuik JP. Central cord syndrome after total hip arthroplasty: A patient report. Spine (Phila Pa 1976), 2005;30:E103-5.
6. Chew BG, Swartz C, Quigley MR, Altman DT, Daffner RH, Wilberger JE. Cervical spine clearance in the traumatically injured patient: Is multidetector CT scanning sufficient alone? Clinical article. J Neurosurg Spine 2013;19:576-81.
7. Cohen AB, Neema M, Arora A, Dell'oglio E, Benedict RH, Tauhid S, *et al*. The relationships among MRI-defined spinal cord involvement, brain involvement, and disability in multiple sclerosis. J Neuroimaging 2012;22:122-8.
8. Dickerman RD, Lefkowitz M, Epstein JA. A traumatic central cord syndrome occurring after adequate decompression for cervical spondylosis: Biomechanics of injury: Case report. Spine (Phila Pa 1976) 2005;30:E611-3.
9. Dvorak MF, Fisher CG, Hoekema J, Boyd M, Noonan V, Wing PC, *et al*. Factors predicting motor recovery and functional outcome after traumatic central cord syndrome: A long-term follow-up. Spine (Phila Pa 1976) 2005;30:2303-11.
10. Epstein NE, Epstein JA, Benjamin V, Ransohoff J. Traumatic myelopathy in patients with cervical spinal stenosis without fracture or dislocation: Methods of diagnosis, management, and prognosis. Spine 1980;6:489-96.
11. Epstein NE, Epstein JA, Zilkha A. Traumatic myelopathy in a seventeen-year-old child with cervical spinal stenosis (without fracture or dislocation) and a C2-C3 Klippel-Feil fusion. A case report. Spine (Phila Pa 1976) 1984;9:344-7.
12. Fehlings MG, Rabin D, Sears W, Cadotte DW, Aarabi B. Current practice in the timing of surgical intervention in spinal cord injury. Spine (Phila Pa 1976) 2010;35 (21 Suppl):S166-73.
13. Feldman KW, Avellino AM, Sugar NF, Ellenbogen RG. Cervical spinal cord injury in abused children. Pediatr Emerg Care 2008;24:222-7.
14. Gu Y, Chen L, Dong RB, Feng Y, Yang HL, Tang TS. Laminoplasty versus conservative treatment for acute cervical spinal cord injury caused by ossification of the posterior longitudinal ligament after minor trauma. Spine J 2014;14:344-52.
15. Harrop JS, Sharan A, Ratliff J. Central cord injury: Pathophysiology, management, and outcomes. Spine J 2006;6 (6 Suppl):198-206S.
16. Joaquim AF, Patel AA, Vaccaro AR. Joaquim AF, Patel AA, Vaccaro AR. Cervical injuries scored according to the Subaxial Injury Classification system: An analysis of the literature. J Craniovertebr Junction Spine 2014;5:65-70.
17. Karim S, Majithia V. Devic's syndrome as initial presentation of systemic lupus erythematosus. Am J Med Sci 2009;338:245-7.
18. Lee S, Lee JE, Yang S, Chang H. A case of central cord syndrome related status epilepticus-a case report. Ann Rehabil Med 2011;35:574-8.
19. Lenehan B, Fisher CG, Vaccaro A, Fehlings M, Aarabi B, Dvorak MF. The urgency of surgical decompression in acute central cord injuries with spondylosis and without instability. Spine (Phila Pa 1976) 2010;35 (21 Suppl):S180-6.
20. Li XF, Dai LY. Acute central cord syndrome: Injury mechanisms and stress features. Spine (Phila Pa 1976) 2010;35:E955-64.

21. McKinley W, Santos K, Meade M, Brooke K. Incidence and outcomes of spinal cord injury clinical syndromes. J Spinal Cord Med 2007;30:215-24.
22. Miranda P, Gomez P, Alday R. Acute traumatic central cord syndrome: Analysis of clinical and radiological correlations. J Neurosurg Sci 2008;52:107-12.
23. Nowak DD, Lee JK, Gelb DE, Poelstra KA, Ludwig SC. Central cord syndrome. J Am Acad Orthop Surg 2009;17:756-65.
24. Onmez H, Cingoz HT, Kucuksen S, Anliacık E, Yaşar O, Yilmaz H, *et al*. Bilateral upper-extremity deep vein thrombosis following central cord syndrome. J Spinal Cord Med 2013;36:243-6.
25. Robles LA. Cervical spine injuries in ocean bathers: Wave-related accidents. Neurosurgery 2006;58:920-3.
26. Santos EA, Filho WJ, Possatti LL, Bittencourt LR, Fontoura EA, Botelho RV. Epidemiology of severe cervical spinal trauma in the north area of São Paulo City: A 10-year prospective study. Clinical article. J Neurosurg Spine 2009;11:34-41.
27. Song J, Mizuno J, Nakagawa H, Inoue T. Surgery for acute subaxial traumatic central cord syndrome without fracture or dislocation. J Clin Neurosci 2005;12:438-43.
28. Song J, Mizuno J, Inoue T, Nakagawa H. 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 2006;65:117-23.
29. Stevens EA, Marsh R, Wilson JA, Sweasey TA, Branch CL Jr, Powers AK. A review of surgical intervention in the setting of traumatic central cord syndrome. Spine J 2010;10:874-80.
30. Thompson C, Gonsalves JF, Welsh D. Hyperextension injury of the cervical spine with central cord syndrome. Eur Spine J 2015;24:195-202.
31. Uribe J, Green BA, Vanni S, Moza K, Guest JD, Levi AD. Acute traumatic central cord syndrome-experience using surgical decompression with open-door expansile cervical laminoplasty. Surg Neurol 2005;63:505-10.
32. Yu HP, Fan SW, Yang HL, Tang TS, Zhou F, Zhao X. Early diagnosis and treatment of acute or subacute spinal epidural hematoma. Chin Med J (Engl) 2007;120:1303-8.
33. Zhang Z, Wang H, Zhou Y, Wang J. Computed tomographic angiography of anterior spinal artery in acute cervical spinal cord injury. Spinal Cord 2013;51:442-7.