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Review Article

Central Cord Syndrome: A Review of Epidemiology, Treatment and Prognostic Factors

Schadler Paul, Shue Jennifer, and Girardi FP*

Spine Care Institute, Hospital for Special Surgery, USA

***Corresponding author**

Girardi FP, Spine Care Institute, Hospital for Special Surgery, 535 East 70th Street New York, NY 10021, USA, Tel: 212-606-1559; Email: girardif@hss.edu

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Abstract

Introduction: Traumatic central cord syndrome is the most common clinical syndrome encountered in the setting of incomplete spinal cord injury and is vaguely defined as by disproportionately more impairment in the upper than the lower extremities.

Methods: A MEDLINE database search was conducted to review the epidemiology, treatment and prognostic factors of this clinical entity.

Results and Discussion: Central cord syndrome typically occurs in white, middle-aged men. Symptoms can be mild to severe, ranging from burning of hands and feet to disproportionate weakness of the upper extremities, variable sensory loss and bladder or bowel dysfunction. The pathogenic mechanism can involve fractures, dislocation and disc herniation caused by hyperextension or flexion injuries of the cervical spine. The prognosis is benign in most cases and the majority of patients, particularly the young, have a high recovery rate. While there is a trend towards surgical intervention in the United States, the management of this syndrome remains controversial due to weak and conflicting evidence. Several prognostic factors have been described, including age and baseline neurologic function. Studies on blood tests and electrophysiological measurement show promising results in predicting a favorable outcome.

Conclusion: There are currently no evidence-based guidelines for surgical treatment or the timing of intervention. Future research, conducted as prospective trials of large populations, may provide further insight into this vaguely defined entity.

INTRODUCTION

Spinal cord injuries of the cervical spine can result in a spectrum of various clinical syndromes that can be classified as complete or incomplete spinal cord injuries [1]. These incomplete injuries can be further grouped into 6 syndromes by their clinical presentation: central cord syndrome, Brown-Squared syndrome, anterior cord syndrome, posterior cord syndrome, conus medullar is syndrome and cauda equine syndrome.

Traumatic central cord syndrome (TCCS) has a spectrum of mild to severe symptoms. It was defined in 1954 by Schneider et al. as "a syndrome that suggests central cervical spinal cord involvement" that is "characterized by disproportionately more motor impairment of the upper than of the lower extremities, bladder dysfunction, usually urinary retention, and varying degrees of sensory loss below the level of the lesion" [2,3].In its mild form, symptoms include burning of hands and feet as the only presentation [4]. This vague definition comprises a heterogeneous population of patients, however, and more accurate definitions are warranted for a better understanding and treatment of this entity. Based on the concept of disproportionate motor weakness, recent studies proposed new classification systems for central cord syndrome. Pouw et al., suggested to define central cord syndrome as a difference in upper and lower motor weakness of at least 10 points on the Medical Research Council motor score [5,6]. In a follow-up study, however, the authors concluded that the severity of the initial neurological deficit, expressed by the American Spinal Injury (ASIA) Impairment scale, has a stronger impact on the prognosis than the diagnosis of traumatic cord syndrome itself [7]. Hohl et al., based their classification system on predictive factors for the outcome measured with the Functional Independence Measurement at 1 year follow-up. Patients are graded as Central Cord Injury Scale 1, 2 or 3 based on ASIA motor score and abnormal MRI findings [8]. More accurate definitions will allow physicians and researchers to gain a better understanding of central cord syndrome.

The purpose of this article is to review the demographics, etiology, current treatment options, as well as outcomes and prognostic factors of central cord syndrome.

METHODS

We conducted a MEDLINE database search, using Pub Med, for English articles published between January 2000 and July 2016, on the topic of central cord syndrome. The search keywords or phrases included "central cord syndrome". In addition, the following keywords were added as search criteria to refine the query: "incidence", "prevalence", "epidemiology", "etiology", "treatment", "outcome", as well as "predictors". Case reports were excluded for this review. This yielded at total of 93 articles. The resulting abstracts were reviewed and sorted for relevance to this topic. The search was supplemented by indexed references of significant importance to this topic cited in articles obtained in the original query. Finally, a total of 62 articles were included in this review. The level of evidence was classified according to the recommendations of the Oxford Centre for Evidence-Based Medicine [9].

RESULTS AND DISCUSSION

Demographics

Incidence: The estimated incidence of spinal cord injury in the United States is 54 per million, or 17,000 new cases each year. The majority of patients are males involved in car accidents, falls or acts of violence. Incomplete injuries are currently the most frequent neurological category with about 60% [10]. Among incomplete cord injuries, central cord syndrome is the most common type, ranging from 40 to 70%of cases [11,12]. Central cord syndrome has been reported to be diagnosed in 4 to 35%of patients with spinal cord injury [12-16]. Recent studies show an increase in the proportion of patients with central cord syndrome among spinal cord injuries, over an 11 year course, from 25 to 37% [17].

Prevalence: In our search results, no study specifically addressed the prevalence of central cord syndrome. The number of people in the United States suffering from spinal cord injury in 2016 was estimated to range from 243,000 to 347,000 people [10].

Age, gender and race: A typical patient with central cord syndrome was described as a married, working, white man at a mean age of 45 years with an average educational level of 11 years by Roth et al.[18]. Men and Caucasians seem to be more susceptible to central cord syndrome: about 70 to 90% of patients were found to be male and about 70% of patients Caucasian [18-26]. In general, incomplete injuries were found to be more common with advancing age. This was particularly true for women [27]. The mean age reported in literature ranges from about 45 to 60 years. Studies reported a bimodal age distribution, however, with about 30% between 10 to 30 years old and 70% older than 40 years in one study [22].In another study assessing the demographics of spinal cord injuries, 55% of patients over 55 years were diagnosed with central cord syndrome, while only 19% of the younger had similar symptoms [17]. This study also found that patients with central cord syndrome were on average 15 years older than other individuals with traumatic cord injury. In contrast to previous studies, females were diagnosed with central cord syndrome in 33% of cases, while males only in 21% of cases in one study [16]. It is unclear, however, if this finding is related to the debated definition of this syndrome.

Pathophysiology and etiology

Injury pattern: Many traumatic and non-traumatic etiologies for central cord syndrome have been described. In this review we will focus on traumatic injuries of the spinal cord. Schneider et al., and Taylor et al., originally hypothesized that hyperextension of the cervical spine causes the ligamentum flavum to protrude anteriorly and compress the spinal cord in an already stenotic spinal canal [2,28]. In younger patients this can be facilitated in the setting of congenital stenosis. In older patients, this is usually caused by degenerative changes including cervical spondylosis or ossification of the posterior longitudinal ligament (OPLL) [22,29].In addition to this classic mechanism of central cord syndrome, several other injury pattern scan lead to central cord syndrome, including flexion injuries, as well as compressive and distractive forces [30-33].

Accidents associated with central cord syndrome: Central cord syndrome is most commonly caused by motor vehicle accidents, falls or sport injuries. These findings have been confirmed in many studies [8,18,23,30,32]. Thompson et al., reported falls as the etiology in 50% of cases in central cord syndrome, while this was reported in 29% of other traumatic spinal cord injuries [17].

Accidents by different age groups: Various patterns of injury have been observed in different age groups [22,24]: Aito et al., found that the mean age for falls was 58 years, while road traffic accidents occurred more commonly around the age of 50, and finally sport-related accidents at the mean age of 23 years. Similarly, Guest et al. reported that in adolescents and young adults (at the age of around 25 years) sport-related accidents were most common (60%), while adults from 26 to 64 years were victims of traffic accidents (60%) and the elderly tended to fall (75%) [34]. Other rare accidents have been described. Anderson et al. reported traumatic intubation (1%) as a cause of central cord syndrome [31]. Additionally, assault was reported as a cause in one patient in a series of 24 patients by Dai et al., [33].

Accidents and alcohol misuse: Several studies reported an association of alcohol misuse with traumatic injuries and central cord syndrome [8]. Lenehan et al., reported that 36% of patients were intoxicated at the time of injury, while Weingarden et al., showed that 68% of patients with central cord syndrome had consumed alcohol prior to their injury, compared to 53% of cases in other types of spinal cord injury [27,35].

Bony and soft tissue injuries associated with central cord syndrome: A multitude of bony and soft tissue injuries, including fracture, dislocation, acute disc herniation, or spinal cord injuries without radiographic abnormality (SCIWORA), are associated with central cord injury [18,19,22,24,25,29,31,32,34,36-42]. In a series of 50 patients, Lenehan et al., observed radiological abnormalities indicating trauma in 24% of patients, while 52%

had stenotic changes only [24]. Similar results were reported by Aito et al., who found canal stenosis in 58% of patients, while fracture occurred in 26%, fracture-dislocation in 13% and pure dislocation in 3% of patients [19]. Guest et al. found stenosis in 46% of patients, while disc herniation was demonstrated in 32%, fractures or dislocations in 20% of patients [34]. In a series of 127 patients, Kato et al. showed that 38% had evidence of stenosis, 47% had spondylosis or ossification of the posterior longitudinal ligament. These findings were more common in older patients. Furthermore, younger patients compared to older patients were more likely to exhibit no abnormal findings on imaging [29].

Level of injury

The lower cervical spine seems to be most commonly affected region. Bosch et al., found that the vertebral level of C5-C6 was most commonly involved in the spinal injury [11,19]. Dai et al., reported that herniation of intervertebral disc in the setting of central cord syndrome occurs most commonly at levels C5-C6, followed by C4-C5 [33]. Ishida et al. reported that C4-C5 was the neurologic level most commonly involved [22].

Bone injuries

Patients with central cord injury sustain fractures or dislocations in 35 to 65% of cases [18,25,31]. Based on the classification system of Allen et al., the most common type of injury was distractive extension injury (42%), followed by isolated posterior element fracture (35%), compression fracture (10%), burst fracture (5%), teardrop fracture (5%) and facet dislocation (3%) [31]. Similarly, Miranda et al. found fractures in 47% of patients, including odontoid fracture, atlantoaxial subluxation, subaxial compressive flexion fracture and complete ligament failure [36].

Spondylosis and stenosis of the spinal canal

Spondylotic changes were found in 8out of 15 (53%) patients with central cord syndrome in a study by Miranda et al. [36]. Ishida et al., reported that in patients without fracture, signs of congenital narrowing were found in 29% of young patients, while 93% of individuals older than 40 years had preexisting conditions, including spondylosis in 86%, developmental stenosis in 33% and OPLL in 13% [22]. OPLL was described in more detail by Gu et al., who reported that 18% of patients with acute cord injury caused by OPLL suffered from central cord syndrome [39]. Yamazaki found that 59% of patients had preexisting stenosis and 10% showed signs of OPLL [40].

Disc herniation

Studies reported that between 22% to 47% of patients suffered from acute disc herniations [32,38].

No radiographic abnormality and other rare findings: A study on spinal cord injury without radiographic abnormality (SCIWORA) demonstrated that 7 out of 59 (12%) patients suffered from central cord syndrome [43].Rarely,2 to 36 months after the initial trauma, the formation of a syrinx can cause incomplete lesions of the spinal cord, with incidences of post-traumatic syringomyelia ranging from 0.3 to 3.2% [44].

Cellular and histologic changes after injury

Schneider et al. proposed that necrosis and hemorrhage in the central area of the spinal cord was the cause for this clinical syndrome [2,3]. This theory was based on the assumption that the corticospinal tract has a somatotopical organization where the fibers for the lower extremity are found laterally, while those for the upper extremity medially. Thus, injury to the central portion of the spinal cord would lead to neurologic deficits of upper extremity more prominently. This hypothesis remains controversial, however. Studies rarely found hemorrhage on histopathologic or MRI examination of the spinal cord. Quencer et al., found no signal changes to suggest intramedullary blood, meningeal adhesions, or hemorrhage, in a series of 11 patients. Instead, focal edema and damage to the white matter of the corticospinal tracts was observed [44]. Similarly, Guest et al. found signal changes that suggested presence of intramedullary hemorrhage in 1 patient only [34]. Recently, Jimenezet al., reported that hand dysfunction in central cord syndrome results from injury to the large fibers of the lateral corticospinal tract. No evidence for motor neuron loss was found in the acute or sub acute phases (up to 5 weeks), but neuron loss was presentin chronic central cord injury [45].

Treatment and outcomes

The role of operative treatment and the timing of intervention remain to be a topic of investigation and debate. This section will first give an overview of current trends in treatment and the pattern of recovery. This review focuses on the scientific discussion on the benefits or disadvantages of surgical and conservative treatment, and does not discuss conservative management or surgical techniques in detail. The interested reader is referred to pertinent reviews.

Current trends in treatment: While most patients are treated conservatively, there is a trend towards increased surgical management of central cord syndrome in the United States. A recent study by Brodell et al., assessed trends in the treatment of 16,134 patients with central cord syndrome in the United States between 2003 and 2010. The study found that 60% of patients were treated non-operatively. Patients treated surgically underwent anterior cervical discectomy and fusion (ACDF) most commonly (19%), followed by posterior cervical discectomy and fusion (PCDF; 7%) and posterior cervical discectomy (PCD, 7%). The number of patients treated surgically increased from about 500 patients in 2003 to more than 2000 in 2013 [20]. Similarly, Yoshihara et al., assessing treatment trends in 19,451 patients with central cord syndrome without bony injury between 2000 and 2009in the United States, reported an increase of surgical treatment from 15 to 31% [46].

Pattern of recovery

The pattern of recovery was described in detail by Merriam et al. in a series of 77 patients of which 30 patients underwent cervical fusion surgery. Motor function first returned in the lower extremities, followed by bladder function, and motor power in the upper extremities. Sensory function did not follow this pattern, however. Normal bladder function was regained by 94% of patients and 86% of patients were independent walkers after 6 months. About 50% of patients had some hand function after 2 years. In this study, the effect of surgery was not examined. While some studies report a plateau in recovery after about 6 weeks, others show continued improvement after 2 years [11,22,42,47].

Comparison of conservative and surgical treatment

There are no evidence-based guidelines for the choice of treatment. Current literature and studies are confined to lower levels of evidence, specifically levels 3 and 4, and most studies are limited by small sample size, heterogeneous study population, and possible residual confounding. Systematic reviews are based on this weak evidence, and thus results must be interpreted with caution. In this section, we will review the current scientific evidence comparing surgical and conservative treatment. In order to make the reader aware of the conflicting evidence, studies are grouped based on whether the results are supportive of conservative or surgical treatment. For each group we will summarize the results of the studies.

Studies supporting conservative treatment

Schneider et al. initially proposed surgical decompression of the spinal cord in patients with central cord syndrome. Two patients described in the case series did not benefit from surgery, while the neurologic status of another patient decreased drastically after surgery. Because of this untoward outcome, the remaining patients were treated conservatively. Schneider et al., concluded that "surgical decompression of the spinal cord is contraindicated", because of the benign prognosis of the syndrome, explaining that "spontaneous improvement or complete recovery may occur" [2,3].

Several other descriptive studies have confirmed the benign prognosis. In a case series of60 conservative patients, Bosch et al., reported that at the time of admission only 19% of patients were independent ambulators, but at a mean follow-up of 5 years, 59% of patients were able to walk. Hand function increased for 26% of patients to 56% at the time of follow-up. Similar findings for bladder and bowel functions were reported: While 17% and 10% of patients had bladder and bowel control on admission, these numbers increased to 53% and 53%, respectively, by the time of follow-up. The late complication of increasing spasticity, stopping patients from being independent ambulators, was found in 24% of all 60 patients [11]. More recently, Ishida et al., described a study of 22 patients treated non-surgically with the aim of finding predictors of a good neurologic recovery. The study showed full motor recovery in 77% of patients, while the remaining had mild dysfunction of hands at 2-year follow-up. Full sensation was recovered in about 60% of patients. Both motor and sensory recovery occurred rapidly until 3 weeks and remained constant after approximately 6 weeks [22]. In agreement with these findings, Newey et al., reported that all patients younger than 50 years of age returned to independent ambulation and full bladder control, while only 69% and 88% of patients from 50 to 70 years recovered these functions, respectively, in a case series of 32 conservatively managed patients [42].

A few analytical studies comparing surgical and conservative treatment failed to show a benefit of surgical intervention. In 1997 Chen et al., retrospectively reviewed 114 patients of which 25% of patients were treated surgically. No significant difference between surgical and nonsurgical groups was found for functional and motor outcome, as well as bladder control. Regardless of treatment, bladder control and walking ability recovered on average after less than 6 days. Complications in surgical cases included loosening of screws, wound infection and additional stabilization surgery of the spine [30]. Similarly, in 1998 Chen et al., compared 16 operative patients to 21 non-operative patients. Neurologic improvement was higher in surgical groups at 6 months. No difference in neurologic outcomes between surgical and nonsurgical groups at 2-year follow-up was found, however. Surgical complications (25%) included neurologic deterioration, dysphasia and dysphagia, screw loosening, surgical site infection and pneumonia. Among nonsurgical patients, the complication rate was 38% and included pneumonia, pulmonary emboli and decubitus ulcer [49].

More recently, in 2007 Aito et al., described 82 patients that were treated surgically or conservatively and found no difference in short- or long-term outcomes between surgical and conservative treatment [19]. Similar results were found in 2015 by Schroeder et al., who retrospectively examined 80 patients with central cord syndrome. About the half of these patients underwent surgical treatment within 24 hours. Patients with fracture spent more days in intensive care unit and had increased length of stay. Surgery within 24 hours was not found to significantly affect changes in motor function, however [50].

Studies supporting surgical intervention

One descriptive study investigated the outcomes after surgical intervention. Uribe et al., described a series of 15 patients who were treated with open-door expansile cervical laminoplasty within 1 to 8 days. Complications rate was 13%, mortality rate 7%. After 3 months 71% of patients improved 1 ASIA grade. The authors concluded that this type of laminoplasty can be safely applied in the subset of patients with central cord syndrome without instability [26].

A few analytical studies found a benefit of surgical over conservative treatment. Dvorak et al. assessed the effect of surgery in 70 patients, of which 59% were treated operatively within 72 hours for instability, or with delayed surgery (after 72 hours) for neurologic deterioration. While regression analysis showed a significant improvement on the Functional Independence Measure Motor Scale, surgical treatment did not improve other outcomes, such as ASIA motor score [21].

Gu et al., compared 31 patients who underwent laminoplasty to 29 patients treated conservatively in 2014. All patients had spinal cord injury with signs of cord compression by OPLL and MRI signal changes. In the surgical group, 18% of patients had central cord syndrome, while this was found in 16% of cases in the non-surgical group. Mean hospital stay was shorter in the operative group. Motor scores were higher in the surgical group compared to the conservative group at 6 months and 3 years. Similarly, sensory scores were higher in the surgical group. Surgical complications included cystitis in 6% of patients, while complication rate in the nonsurgical group was 21% and included urinary tract infection, decubitus ulcer, pneumonia and deep vein thrombosis [39].

Stevens et al., conducted a retrospective review of 126 patients of which 67 patients were treated surgically and 59 non-surgically. Surgical procedures included anterior, posterior or combined decompression with or without instrumentation. Of the 67 surgical patients, 16 were treated within 24 hours, 34 patients after 24 hours, and 17 patients on readmission after a mean time of 137 days. Patients in the surgical group were significantly younger. The study demonstrated a significant difference in improvement in Frankel grade between surgical and non-surgical groups. No difference of neurologic outcome between the three surgical subgroups was found. Surgical treatment did not affect length of hospital or intensive care unit stay, or complication rate [25].

Finally, in two systematic reviews of case series and retrospective studies in 2013, Aarabi et al., and Dahdaleh et al., concluded that there was level 3 evidence to support the benefit of surgical treatment in patients with central cord syndrome [51,52].

Timing of surgical treatment

Controversy also exists over the optimal timing of surgical intervention. While some studies advocate early intervention, ideally within 24 hours, other studies support delayed surgery and some show no difference at all. This section will present studies grouped on their results, supporting early or late intervention, in order to emphasize the conflicting results.

Studies supporting early surgical intervention

A few analytical studies support early surgical intervention. Fehlings et al., conducted a prospective, multicenter study between 2002 and 2009, with 222 spinal cord injury patientswith a follow-up of 6 months. In multivariate analysis, the odds of improvement of at least 2 grades in ASIA Impairment Scale were 2.8 times higher in patients who underwent surgery within 24 hours. It is unclear if these findings can be applied to patients with central cord syndrome [53].

Guest et al., reported on 50 patients with central cord syndrome treated surgically. Surgery was performed via posterior or anterior approaches, within 24 hours in 16 patients, and after 24 hours in 34 patients. Patients with early surgery had a shorter stay in the intensive care unit and the hospital compared to late surgery. Patients with acute disc herniation, cervical fractures or dislocations had a bettermotor recovery than patients with spondylosis or stenosis, if the surgery was performed within 24 hours. After 36 months of follow-up, all patients with early intervention for disc herniation, fracture or dislocation were able to walk independently, while 88% of patients who underwent delayed surgery had the same outcome. Patients with spondylosis or stenosis required no assistance walking in 66% of early surgical cases and in 61% of delayed surgical cases [34].

Yamazaki et al., studied 47 patients with central cord syndrome of which 23 were treated with surgical decompression. The study showed that spinal canal diameter and interval between injury and surgery were significantly associated with a better recovery [40].

These results have been confirmed in a meta-analysis by La Rosa et al., Early decompression in patients with incomplete spinal cord injury resulted in significantly better outcome compared to late and conservative treatment [54]. In a systematic review in 2015, Anderson et al., reported low-level evidence suggesting that surgery within 24 hours significantly improved recovery of ASIA motor scores, while there was weak evidence that patients operated within 2 weeks have a higher recovery rate measured by the Japanese Orthopaedic Association score [55,56]. Similarly, Fehlings et al., conducted a systematic review in 2006 and concluded early decompressive surgery (within 24 hours) can be performed safely [57]. In a systematic review, Molliqaj et al., concluded that early decompression might be indicated in patients who exhibit progressive neurological deficits. Controversy persists, however, and no clear recommendation can be proposed, because current systematic reviews are based on low-level evidence [48].

Studies supporting delayed surgical intervention

One analytical study showed benefits in patients treated with delayed surgery. In 2015 Samuel et al. retrospectively reviewed 1060 patients with central cord syndrome treated surgically. Surgical timing was found to be associated with a 19% decrease in the odds of mortality with each 24-hour increase in time. Thus delayed surgery was associated with reduced odds of mortality. Time to surgery was also associated with a 7% increase in odds of minor adverse events with each 24-hour increase in time to surgery. Delayed surgery was not associated with more severe adverse events, however [23].

Studies showing no difference in surgical timing

A few analytical studies did not show a difference between early and delayed surgical intervention. In 69 patients treated surgically for spinal instability or declining neurologic status, Anderson et al. found an overall increase of 63 points on the American Spinal Injury Association (ASIA) motor score scale to 90 points. About 70% of patients improved 1 or more grades on the ASIA Impairment Scale. There was no difference in motor function outcome between early (less than 24 hours after injury), midrange (24 to 48 hours), and late (more than 48 hours) surgical treatment. There was also no difference in approach. A 25% surgical complication rate, with the most common complications being surgical site infections, urinary tract infection and dysphagia, was noted [31].

In 2009 Chen et al., assessed a study population of 49 patients who were treated surgically with an anterior or posterior approach within 4 days or after 4 days of admission. Significant improvement in ASIA scores was noted during the first 6 months after surgical intervention. No difference with regards to timing of surgical intervention was found. Full bladder control recovered from 56% to 80%, while ASIA motor scores improved from 50 and 60 to 99 and 90 in the early and delayed surgical group respectively. Spasticity and neuropathic pain were major factors leading to a poor quality of life as reported by patients [38].

In 2015 Kepler et al., reported of 68 patients who underwent surgery within 24 hours in 28% of cases, while the remaining 72% had delayed surgical intervention. Patients in the early intervention group were younger. The study showed no difference in length of stay in the hospital or intensive care unit, motor improvement, or morbidity and mortality after 7 days [58].

Finally, a systemic review by Aarabi et al., in 2013 concluded that most class III evidence did not show advantages of early versus late surgical treatment [52].

Prognostic factors for neurologic outcome

Demographic and medical factors: Several baseline prognostic factors have been established. Age was found to be an important factor for recovery in several studies. Neurologic improvement was higher in younger patients [14,19,22,30,30,33,38,58-60]. Furthermore, patients with a higher formal education were found to have a better neurologic outcome [21].

Injury-related and medical factors: Absence of comorbidities and anterior column fracture were associated with better outcomes in one study [21], and another study reported that patients with fracture or dislocation recovered more slowly than other patients [33]. Furthermore, studies indicated that the spinal canal diametermight correlated with the functional outcome [40,61].

Neurologic factors: The baseline neurologic status was found to be an important factor in recovery by several studies. Dvorak et al. found that ASIA motor score at admission and formal education predicted better recovery. Patients with a greater rate of motor recovery and higher scores at mean follow-up of 70 months were more likely to suffer from spasticity, however. Additionally, functional outcome was higher in young patients and lower in patients with spasticity [21]. In agreement with this Thompson et al. showed that an ASIA motor score of greater than 60 on admission correlated with an 80% chance of being able to walk at the time of discharge, while scores lower than 50 were correlated with an 80% chance of not being able to walk [61]. Several other studies reported a similar association of motor score and outcome [8,33,40]. Furthermore, the percent deficit improvement during the early period after injury was also a significant predictor of better outcome in one study [22].

Imaging

Magnetic resonance image plays an important prognostic role in patients with central cord syndrome. In the setting of a rehabilitation center, Hohl et al., showed that abnormal signal intensity on MRI at the time of injury was a significant predictor for the motor portion of the Functional Independence Measurement at 1-year follow-up [8]. Aarabi et al., [62] and other studies [22,40] reported similar findings of the association of T2-weighted signal intensity with poor neurologic status. While patients with increased T2-weighted signal intensity on MRI demonstrated a stable neurologic status within the first week, patients without this finding experienced a decline in their ASIA motor score of 4.3 points. Furthermore, patients with increased T2-weighted signal intensity spent more time in the intensive care unit. Aarabi et al., concluded that patients should be treated differently depending on T2-weighted MRI scan findings.

MRI in patients with SCIWORA

In patients with SCIWORA Machino et al., evaluated MRI findings within an average of 26 hours after injury for their predictive value of outcome. The study found that increased MRI signal intensity and prevertebral hyper-intensities correlated negatively with recovery rate [63]. Ouchida et al., described similar findings [64]. Liu et al., however, concluded that neurological outcome did not correlate with early MRI (within 24 hours) findings in patients with SCIWORA. Thus, a repeat MRI may be necessary [43].

Electrophysiological measurement

Intraoperative electrophysiologic measurement might be a valuable tool to predict neurologic recovery after spinal cord injury. Costa et al., conducted a prospective observational study on 55 patients undergoing posterior spine stabilization for traumatic spinal cord injury. The study reported that in central cord syndrome, electrophysiological measurement showed the presence of a clear D wave and absent low voltage muscular motor evoked potentials from hand muscle. Patients with this pattern recovered significantly [65].

Blood tests

Kuhle et al., examined serum neurofilament light chain levels in patients with central cord syndrome and other spinal cord injuries. All patients were treated with surgical decompression, while no patient received steroids. Baseline neurofilament levels were highest in patients with central cord syndrome, compared to other syndromes, and correlated with motor scores. Motor outcome plateaued after 3 months in all patients regardless of syndrome, and was highly correlated with baseline neurofilament levels. Similar correlations were seen for sensory recovery [47].

Prognostic factors for morbidity and mortality

Brodell et al., showed that mortality in patients with income of $41,000 to $50,999 was higher in comparison to other quartiles. Other factors increasing mortality were surgery at a rural hospital and congestive heart failure [20]. Similarly, Injury Severity Score and Charleson Comorbidity Index increased the risk of severe adverse events at an odds ratio of 1.04 and 1.25 in a study by Samuel et al.

CONCLUSION

Traumatic spinal cord injury is the most frequent incomplete spinal cord injury, most commonly caused by falls and motor vehicle accidents in middle-aged white males. The definition of this clinical syndrome is controversial as the patient population with this diagnosis suffers from a heterogeneous group of injury mechanisms and pathological findings. Similarly, surgical treatment and timing of intervention remain a topic of debate. No recommendation for the choice of treatment can be made based on current evidence. Prognostic factors, such as age and baseline neurologic function might help physicians in their treatment decision. Other prognostic tests, such as serum neurofilament or MRI studies seem promising. Future research should include analytical prospective trials and cohort studies to draw more definitive conclusions about treatment and timing of surgical intervention.

REFERENCES

1. American spinal injury association. Standard neurological classification of spinal cord injury worksheet. 2006.

2. 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. J Neurosurg. 1954; 11: 546-577.

3. SchneideR RC, Thompson JM, Bebin J. The syndrome of acute central cervical spinal cord injury. J Neurol Neurosurg Psychiatry. 1958; 21: 216-227.

4. Maroon JC. 'Burning hands' in football spinal cord injuries. JAMA. 1977; 238: 2049-2051.

5. Pouw MH, van Middendorp JJ, van Kampen A, Hirschfeld S, Veth RP, Curt A, et al. Diagnostic criteria of traumatic central cord syndrome. Part 1: a systematic review of clinical descriptors and scores. Spinal Cord. 2010; 48: 652-656.

6. van Middendorp JJ, Pouw MH, Hayes KC, Williams R, Chhabra HS, Putz C, et al. Diagnostic criteria of traumatic central cord syndrome. Part 2: a questionnaire survey among spine specialists. Spinal Cord. 2010; 48: 657-663.

7. Pouw MH, van Middendorp JJ, van Kampen A, Curt A, van de Meent H, Hosman AJ. Diagnostic criteria of traumatic central cord syndrome. Part 3: descriptive analyses of neurological and functional outcomes in a prospective cohort of traumatic motor incomplete tetraplegics. Spinal cord. 2011; 49: 614-622.

8. Hohl JB, Lee JY, Horton JA, Rihn JA. A novel classification system for traumatic central cord syndrome: the central cord injury scale (CSIS). Spine. 2010; 35: e238-243.

9. Ocebm levels of evidence working group, oxford centre for evidence-based medicine. 2016.

10. National spinal cord injury statistical center, spinal cord injury. Facts and figures at a glance.

11. Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia. A ten-year review. JAMA. 1971; 216: 473-478.

12. McKinley W, Santos K, Meade M, Brooke K. Incidence and outcomes of spinal cord injury clinical syndromes. J Spinal Cord Med. 2007; 30: 215-224.

13. Merriam WF, Taylor TK, Ruff SJ, McPhail MJ. A reappraisal of acute traumatic central cord syndrome. J Bone Joint Surg Br. 1986; 68: 708-713.

14. Pollard ME, Apple DF. Factors associated with improved neurologic outcomes in patients with incomplete tetraplegia. Spine (Phila Pa 1976). 2003; 28: 33-39.

15. Santos EA, Filho WJ, Possatti LL, Bittencourt LR, Fontoura EA, Botelho RV. Epidemiology of severe cervical spinal trauma in the north area of sao paulo city: a 10-year prospective study. Clinical article. J Neurosurg Spine. 2009; 11: 34-41.

16. Pickett GE, Campos-Benitez M, Keller JL, Duggal N. Epidemiology of traumatic spinal cord injury in Canada. Spine (Phila Pa 1976). 2006; 31: 799-805.

17. Thompson C, Mutch J, Parent S, Mac-Thiong JM. The changing demographics of traumatic spinal cord injury: An 11-year study of 831 patients. J Spinal Cord Med. 2015; 38: 214-223.

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

19. Aito S1, D'Andrea M, Werhagen L, Farsetti L, Cappelli S, Bandini B, et al. Neurological and functional outcome in traumatic central cord syndrome. Spinal Cord. 2007; 45: 292-297.

20. 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: 435-442.

21. 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: 2303-2311.

22. Ishida Y, Tominaga T. Predictors of neurologic recovery in acute central cervical cord injury with only upper extremity impairment. Spine. 2002; 1652-1658.

23. Samuel AM, Grant RA, Bohl DD, Basques BA, Webb ML, Lukasiewicz AM, et al. Delayed surgery after acute traumatic central cord syndrome is associated with reduced mortality. Spine (Phila Pa 1976). 2015; 140: 349-356.

24. Lenehan B, Street J, O'Toole P, Siddiqui A, Poynton A. Central cord syndrome in Ireland: the effect of age on clinical outcome. Eur Spine J. 2009;, 18: 1458-1463.

25. Stevens EA, Marsh R, Wilson JA, Sweasey TA, Branch CL, Powers AK. A review of surgical intervention in the setting of traumatic central cord syndrome. Spine J. 2010; 10: 874-880.

26. 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: 505-510.

27. Lenehan B, Street J, Kwon BK, Noonan V, Zhang H, Fisher CG, et al. The epidemiology of traumatic spinal cord injury in British Columbia, Canada. Spine (Phila Pa 1976). 2012; 37: 321-329.

28. Taylor AR, Blackwood W. Paraplegia in hyperextension cervical injuries with normal radiographic appearances. J Bone Joint Surg Br. 1948; 30B: 245-248.

29. Kato H, Kimura A, Sasaki R, Kaneko N, Takeda M, Hagiwara A, et al. Cervical spinal cord injury without bony injury: a multicenter retrospective study of emergency and critical care centers in Japan. J Trauma. 2008; 65: 373-379.

30. Chen TY, Lee ST, Lui TN, Wong CW, Yeh YS, Tzaan WC, et al. Efficacy of surgical treatment in traumatic central cord syndrome. Surg Neurol. 1997; 48: 435-440.

31. 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). 2012; 41: E104-108.

32. Rizzolo SJ, Piazza MR, Cotler JM, Balderston RA, Schaefer D, Flanders A. Intervertebral disc injury complicating cervical spine trauma. Spine (Phila Pa 1976). 1991; 16: S187-189.

33. Dai L, Jia L. Central cord injury complicating acute cervical disc herniation in trauma. Spine (Phila Pa 1976). 2000; 25: 331-335.

34. Guest J, Eleraky MA, Apostolides PJ, Dickman CA, Sonntag VK. Traumatic central cord syndrome: results of surgical management. J Neurosurg. 2002; 97: 25-32.

35. Weingarden SI, Graham PM. Falls resulting in spinal cord injury: patterns and outcomes in an older population. Paraplegia. 1989; 27: 423-427.

36. Miranda P, Gomez P, Alday R. Acute traumatic central cord syndrome: analysis of clinical and radiological correlations. J Neurosurg Sci. 2008; 52: 107-112.

37. Papadopoulos SM, Selden NR, Quint DJ, Patel N, Gillespie B, Grube S. Immediate spinal cord decompression for cervical spinal cord injury: feasibility and outcome. J Trauma. 2002; 52: 323-332.

38. Chen L, Yang H, Yang T, Xu Y, Bao Z, Tang T. Effectiveness of surgical treatment for traumatic central cord syndrome. J Neurosurg Spine. 2009; 10: 3-8.

39. 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: 344-352.

40. Yamazaki T, Yanaka K, Fujita K, Kamezaki T, Uemura K, Nose T. Traumatic central cord syndrome: analysis of factors affecting the outcome. Surg Neurol. 2005; 63: 95-99.

41. Boese CK, Nerlich M, Klein SM, Wirries A, Ruchholtz S, Lechler P. Early magnetic resonance imaging in spinal cord injury without radiological abnormality in adults: a retrospective study. J Trauma Acute Care Surg. 2013; 74: 845-848.

42. Newey ML, Sen PK, Fraser RD. The long-term outcome after central cord syndrome: a study of the natural history. J Bone Joint Surg Br. 2000; 82: 851-855.

43. Liu Q, Liu Q, Zhao J, Yu H, Ma X, Wang L. Early MRI finding in adult spinal cord injury without radiologic abnormalities does not correlate with the neurological outcome: a retrospective study. Spinal Cord. 2015; 53: 750-753.

44. Quencer RM, Bunge RP, Egnor M, Green BA, Puckett W, Naidich TP, et al. Acute traumatic central cord syndrome: MRI-pathological correlations. Neuroradiology. 1992; 34: 85-94.

45. Jimenez O, Marcillo A, Levi AD. A histopathological analysis of the human cervical spinal cord in patients with acute traumatic central cord syndrome. Spinal Cord. 2000; 38: 532-537.

46. 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. 2013; 75: 453-458.

47. Kuhle J, Gaiottino J, Leppert D, Petzold A, Bestwick JP, Malaspina A. Serum neurofilament light chain is a biomarker of human spinal cord injury severity and outcome. J Neurol Neurosurg Psychiatry. 2015; 86: 273-279.

48. Molliqaj G, Payer M, Schaller K, Tessitore E. Acute traumatic central cord syndrome: a comprehensive review. Neurochirurgie. 2014; 60: 5-11.

49. Chen TY, Dickman CA, Eleraky M, Sonntag VK. The role of decompression for acute incomplete cervical spinal cord injury in cervical spondylosis. Spine (Phila Pa 1976). 1998; 23: 2398-403.

50. Schroeder GD, Kepler CK, Hjelm N, Vaccaro AR, Weinstein MS. The effect of vertebral fracture on the early neurologic recovery in patients with central cord syndrome. Eur Spine J. 2015; 24: 985-989.

51. Aarabi B, Hadley MN, Dhall SS, Gelb DE, Hurlbert RJ, Rozzelle CJ, et al. Management of acute traumatic central cord syndrome (ATCCS). Neurosurgery. 2013; 72: 195-204.

52. Dahdaleh NS, Lawton CD, E Ahmadieh TY, Nixon AT, El Tecle NE, Oh S, et al. Evidence-based management of central cord syndrome. Neurosurg Focus. 2013; 35: E6.

53. Fehlings MG, Vaccaro A, Wilson JR, Singh A, Cadotte D W, Harrop JS, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). PLoS One. 2012; 7: e32037.

54. La Rosa G, Conti A, Cardali S, Cacciola F, Tomasello F. Does early decompression improve neurological outcome of spinal cord injured patients? Appraisal of the literature using a meta-analytical approach. Spinal Cord. 2004; 42: 503-512.

55. Anderson KK, Tetreault L, Shamji MF, Singh A, Vukas RR, Harrop JS, et al. Optimal Timing of Surgical Decompression for Acute Traumatic Central Cord Syndrome: A Systematic Review of the Literature. Neurosurgery. 2015; 77: S15-32.

56. 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: S180-186.

57. Fehlings MG, Perrin RG. The timing of surgical intervention in the treatment of spinal cord injury: a systematic review of recent clinical evidence. Spine (Phila Pa 1976). 2006; 31: S28-35.

58. Kepler CK, Kong C, Schroeder GD, Hjelm N, Sayadipour A, Vaccaro AR, et al. Early outcome and predictors of early outcome in patients treated surgically for central cord syndrome. J Neurosurg Spine. 2015; 23: 490-494.

59. Penrod LE, Hegde SK, Ditunno JF. Age effect on prognosis for functional recovery in acute, traumatic central cord syndrome. Arch Phys Med Rehabil. 1990; 71: 963-968.

60. Stevenson CM, Dargan DP, Warnock J, Sloan S, Espey R, Maguire S, et al. Traumatic central cord syndrome: neurological and functional outcome at 3 years. Spinal Cord. 2016.

61. Crispin Thompson, Jose Filipe Gonsalves, David Welsh. Hyperextension injury of the cervical spine with central cord syndrome. 2014; 24; 195-202.

62. 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: 122-130.

63. Machino M, Yukawa Y, Ito K, Nakashima H, Kanbara S, Morita D, et al. Can magnetic resonance imaging reflect the prognosis in patients of cervical spinal cord injury without radiographic abnormality? Spine (Phila Pa 1976). 2011; 36: E1568-1572.

64. Ouchida J, Yukawa Y, Ito K, Katayama Y, Matsumoto T, Machino M, et al. Delayed Magnetic Resonance Imaging in Patients With Cervical Spinal Cord Injury Without Radiographic Abnormality. Spine (Phila Pa 1976). 2016; 41: E981-986.

65. Costa P, Faccani G, Sala F, Montalenti E, Giobbe ML, Deletis V. Neurophysiological assessment of the injured spinal cord: an intraoperative approach. Spinal Cord. 2014; 52: 749-757.

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