Traumatic central cord syndrome (CCS) is believed to be due to damage to the corticospinal tracts of the spinal cord leading to marked axonal breakdown in the lateral white matter columns. Classically, patients present with significant weakness with preferential involvement of the hands and upper extremities. While the clinical presentation of most patients with CCS is similar, there is significant heterogeneity involved in this pathology. Often patients are older than 50 years of age and have significant preexisting cervical spondylosis; however, CCS also occurs in younger patients due to either bony instability leading to cord compression or an acute large central disc herniation.

Despite the relative frequency of CCS compared with other incomplete spinal cord injury (SCI) syndromes, its management is the most controversial. In the “Surgical Timing in Acute Spinal Cord Injury Study,” Fehlings et al. reported significantly improved neurological recovery after an early decompression (< 24 hours) in patients with an acute SCI. However, its applicability to patients with CCS is unclear. Since the syndrome was initially described by Schneider 70 years ago, the tendency for neurological recovery with only medical management has frequently been reported, and current literature on early surgical decompression in CCS has failed to consistently demonstrate clinical improvement after early decompression.
The discrepancy in the literature regarding the ideal treatment for CCS likely arises from injury heterogeneity. While numerous studies have been designed to evaluate different treatment variables, such as the presence and timing of surgery, often only a cursory analysis is performed to account for patient-specific variables. It is possible that patients with baseline chronic SCI have a different injury profile than patients with an acute injury. While not a perfect surrogate for spinal cord health, the presence of changes in the signal intensity of the spinal cord on MRI has been associated with both reversible injuries such as edema and ischemia, and permanent damage such as necrosis and myelomalacia. Furthermore, in patients undergoing surgery for cervical myelopathy, the changes in the signal intensity on MRI have been associated with a decreased functional recovery. While many studies have been performed looking at the effect of cord signal change in myelopathic patients, there is a paucity of literature on its effect on patients with CCS. The goal of this study is to compare both injury severity and early changes in the American Spinal Injury Association (ASIA) motor score (AMS) between CCS patients with and without increased T2 MRI signal intensity in the spinal cord.

Methods

A retrospective review of an SCI database identified all patients with CCS who presented to a regional spinal cord injury center (one of 14 centers designated by the National Institute on Disability and Rehabilitation Research) between January 1, 2004, and June 2009. While all patients underwent MRI, the diagnosis of CCS was determined based on the clinical impression of the attending surgeon. The daily progress notes of the Physical Medicine and Rehabilitation physicians were reviewed to determine the daily AMS for the 1st week after injury. The trauma database and hospital records were used to identify all important patient or treatment variables such as demographics, presenting neurological examination findings, imaging findings, timing of surgical procedures, and length of stay. The initial neurological presentation as well as changes in neurological status was measured using the AMS, as it allows for the scoring of the motor function of each limb individually. This allows for a more precise understanding of the patient’s neurological status than the ASIA grade and therefore is a better tool for following an evolving SCI.

Patients were stratified based on the presence or absence of increased signal intensity on axial and sagittal T2-weighted MRI sequences. As most patients did not have previous imaging available for review, no attempt was made to distinguish chronic versus acute signal change. The Student t-test for continuous variables and Fisher’s exact test for categorical variables was used to identify differences between the 2 groups. Type III ANOVA with Satterthwaite approximation for degrees of freedom was performed to determine if age, sex, congenital stenosis, the presenting Injury Severity Score (ISS), surgery within 24 hours or surgery in the initial hospitalization significantly affected the change in AMS. A p value of < 0.05 was accepted as significant. All statistical analyses were carried out in the statistical platform R 3.1.1 (R Foundation for Statistical Computing).

Results

Thirty-two patients with CCS and increased T2 signal intensity in their spinal cord were identified, and 43 patients with CCS without increased T2 signal intensity were identified. No difference in age, sex, or Glasgow Coma Scale score was identified in patients with or without an increase in T2 signal intensity (Table 1); however, patients with increased signal intensity had a more severe neurological injury on presentation than patients without an increased signal intensity (AMS 57.6 vs 75.3, respectively, p = 0.01), and an increased signal intensity was associated with a trend toward an elevated ISS (22.2 vs 16.8, p = 0.09). While the neurological injury was more severe in patients with an increased T2 signal intensity, there was a trend toward less severe mechanisms of injury (Table 2) in these patients. The mechanism of injury was a minor fall (defined as a fall from sitting or standing) in 42.9% of patients with an increased T2 signal intensity versus 27.5% of patients without an increase in T2 signal intensity (p = 0.15). Similarly, the mechanism of injury was a major fall (defined as any fall more severe than a simple fall from sitting or standing, such as a fall off a ladder, a fall down the stairs, or a fall out of a window) for 45.0% of patients without increase in T2 signal intensity, whereas as this was the mechanism of injury in 31.4% of patients with increased T2 signal intensity (p = 0.09).

Surgical treatment of both groups was similar, as 23.5% of patients with increased T2 signal intensity underwent surgery within 24 hours, compared with 25.6% of patients without increased T2 signal intensity (p = 0.99). Similarly, no significant difference in the number of patients who underwent surgical decompression during their initial hospitalization was identified (increased T2 signal intensity 93.8% vs no increased T2 signal intensity 79.5%, p = 0.10).

While the overall length of stay in the hospital was similar between patients with and without an increase in the T2 signal intensity (12.5 vs 10.3 days, respectively, p = 0.22), patients with an increased T2 signal intensity spent significantly more time in the ICU, and there was a strong trend toward an increased length of dependence on mechanical ventilation (Table 3). There was no difference in the average number of days the AMS was recorded for

<table>
<thead>
<tr>
<th>TABLE 1. Demographics of patients with CCS</th>
</tr>
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<tbody>
<tr>
<td>Variable</td>
</tr>
<tr>
<td>-----------------------------</td>
</tr>
<tr>
<td>Mean age</td>
</tr>
<tr>
<td>% Female</td>
</tr>
<tr>
<td>% Male</td>
</tr>
<tr>
<td>Mean GCS score</td>
</tr>
<tr>
<td>Mean ISS</td>
</tr>
<tr>
<td>Mean presenting AMS</td>
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</tbody>
</table>

GCS = Glasgow Coma Scale; SI = signal intensity.

* Values are presented as the mean ± SD unless otherwise specified. The value in boldface is statistically significant.
patients with and without an increased T2 signal intensity (6.4 ± 1.3 vs 5.8 ± 1.7, respectively, p = 0.13), but a trend was identified in the average change of AMS over the 1st week. Patients with an increased T2 signal intensity demonstrated a fairly stable neurological examination with the average AMS declining by 0.85 ± 17.9 in the 1st week, whereas patients without an increased T2 signal intensity had a decline in their AMS by 4.3 ± 15.5 over the 1st week (p = 0.07). Lastly, an ANOVA determined that age, sex, ISS, congenital stenosis, surgery within 24 hours and surgery during the 1st admission did not significantly affect the change in AMS over the 1st week (Table 4).

Discussion

The goal of the current study was to determine if the presence of T2 signal intensity change is correlated with a more severe neurological injury. We found that patients with CCS and increased T2 signal intensity have a more severe neurological injury (p = 0.01) but have minimal progression of the injury over the 1st week. Comparative-ly, patients without an increase in T2 signal intensity have a less severe initial injury but have continued neurological decline over the 1st week.

These results indicate that while the constellation of symptoms may be similar in all patients with CCS, the underlying neurological injury may be slightly different in patients with increased T2 signal intensity. An increase in T2 signal intensity is often associated with chronic compression of the spinal cord, and it is well established that chronic compression results in structural changes to the spinal cord. Persistent compression distorts the vascular architecture, leading to an overall decrease in the number of capillaries. Additionally, there is endothelial cell loss and dysfunction in the remaining vessels. These changes may make the spinal cord more sensitive to the initial injury, but with less blood flow there may be less hemorrhage, vasospasm, and production of free radicals limiting the secondary injury.

The finding that there is a difference between the neurological injury of patients with CCS with and without an elevated T2 signal intensity is of significant importance in future research, as it may explain the discrepancy in the literature regarding the ideal treatment for CCS. This variation in treatment of this injury can be seen in the current study by the fact that a similar number of patients in each cohort underwent surgery; despite the fact that patients with an increase in T2 signal intensity had a more severe neurological injury. Our institution is one of the largest SCI centers in North America, but the discrepancy in the literature still prohibits an agreed upon surgical algorithm for these patients. Because of this, the decision to perform surgical decompression in patients in this study was surgeon dependent. The results of the current study suggest that there is a difference between patients with CCS with and without an increase in T2 signal intensity, and it is possible that these patients should be treated differently. Unfortunately, because of the small number of patients in each cohort and the fact that the groups were treated similarly, the current study is severely underpowered to determine if surgical treatment leads to more improvement in patients with or without T2 signal intensity. It is our hope the this study will spur future investigators to perform high-level prospective studies to determine which patients with CCS will benefit from early surgical treatment and which can be managed conservatively.

While this is the first study designed to specifically compare patients with CCS with and without an increased T2 signal intensity, the results are consistent with existing literature. Multiple authors have demonstrated that a narrow midsagittal diameter of the spinal canal portends a poor neurological outcome; however, only one of these studies directly evaluated the appearance of the spinal cord. Aarabi et al. reported that patients with longer areas of increased signal intensity had worse manual dexterity after 12 months; however, because all the patients in the study had an increased T2 signal intensity, no comparison between patients with and without increased T2 signal intensity was possible. Furthermore, it is unclear if the worse long-term neurological function was the result of a more severe injury or less neurological recovery. Other recent literature on CCS also indicates that patient-

### Table 2. Mechanism of injury

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Increased T2 SI</th>
<th>No Increased T2 SI</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diving accident</td>
<td>5.7%</td>
<td>5.0%</td>
<td>0.99</td>
</tr>
<tr>
<td>Fall from height</td>
<td>31.4%</td>
<td>45.0%</td>
<td>0.09</td>
</tr>
<tr>
<td>Fall from either sitting or standing</td>
<td>42.9%</td>
<td>27.5%</td>
<td>0.15</td>
</tr>
<tr>
<td>Motor vehicle accident</td>
<td>20.0%</td>
<td>17.5%</td>
<td>0.99</td>
</tr>
<tr>
<td>Other</td>
<td>0.0%</td>
<td>2.5%</td>
<td>0.99</td>
</tr>
<tr>
<td>Sports injury</td>
<td>0.0%</td>
<td>2.5%</td>
<td>0.99</td>
</tr>
</tbody>
</table>

### Table 3. Outcomes of patients with and without an increased T2 signal intensity*

<table>
<thead>
<tr>
<th>Outcome Measure</th>
<th>Increased T2 SI</th>
<th>No Increased T2 SI</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean length of stay</td>
<td>12.5 ± 6.7</td>
<td>10.3 ± 7.9</td>
<td>0.22</td>
</tr>
<tr>
<td>Mean no. of days in the ICU</td>
<td>4.7 ± 0.3</td>
<td>2.0 ± 0.4</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean no. of days on mechanical ventilation</td>
<td>2.5 ± 0.3</td>
<td>1.2 ± 0.3</td>
<td>0.06</td>
</tr>
<tr>
<td>Mean change in AMS</td>
<td>−0.85 ± 17.9</td>
<td>−4.3 ± 15.5</td>
<td>0.07</td>
</tr>
<tr>
<td>Mortality</td>
<td>3.1%</td>
<td>2.6%</td>
<td>0.99</td>
</tr>
</tbody>
</table>

* Values are presented as the mean ± SD unless otherwise specified. The value in boldface is statistically significant.
specific variables may be one of the most critical factors in early neurological recovery. Thompson et al. reported that more than 80% of patients with CCS and a cervical spinal canal diameter of more than 8 mm had a functional Frankel grade (Frankel Grade D or E) when they were discharged from the hospital, and Schroeder et al. reported that patients with CCS and associated fracture had early neurological improvement, whereas similar patients without a fracture demonstrated neurological decline.

Significant limitations with our study exist, including those inherent to retrospective analysis and the short-term follow-up. However, this study was specifically designed to determine how T2 signal intensity affected the initial severity of the neurological injury and the neurological recovery during the hospital course. The significant difference (p = 0.01) in the severity of the initial neurological injury, in spite of a trend toward a less severe mechanism of injury (p = 0.09–0.14), indicates that the neurological injury in the primary injury may be slightly different in these patients. Furthermore, the nearly significant difference (p = 0.07) in the change in AMS over the 1st week in these 2 groups of patients suggests that there may also be a difference in the secondary injury mechanism. Because of the short follow-up and the fact that patients were treated similarly in both groups, the current study cannot be used to guide treatment. Rather, it allows for a physician to give evidenced-based expectations for the patient and family members. Currently only 2 studies in the literature allow surgeons to guide patients on what they can expect in the immediate aftermath of this devastating injury. Outcome stratification by patient-related and injury-related factors will be useful to physicians treating patients with CCS; evidence-based counseling on what patients and their families can expect over both short- and longer-term time spans is critical to set expectations and allow appropriate planning for future care. The need for long-term studies does not mitigate the importance of reporting early results. Lastly, the knowledge that a slight neurological decline in the first week is common in some patients with CCS may prevent significant consternation for both patient and physician when this occurs.

**Conclusions**

While patients with CCS are commonly grouped together, the current study indicates that both the primary and secondary neurological injury may be different in patients with and without an increase in spinal cord T2 signal intensity. Patients with increased T2 signal intensity are likely to have a more severe initial neurological deficit but will have relatively minimal early neurological deterioration. Comparatively, patients without an increase in T2 signal intensity will likely have a less severe initial injury but can expect to have a slight decline in neurological function in the 1st week.

**References**


Disclosures

Author Contributions
Conception and design: Schroeder, Weinstein, Kepler. Acquisition of data: Hjelm, Vaccaro, Kepler. Analysis and interpretation of data: Schroeder, Vaccaro, Kepler. Drafting the article: Schroeder, Vaccaro, Kepler. Critically revising the article: Schroeder, Vaccaro, Weinstein, Kepler. Reviewed submitted version of manuscript: Schroeder, Vaccaro, Weinstein, Kepler. Approved the final version of the manuscript on behalf of all authors: Schroeder. Statistical analysis: Kepler.

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