

## CLINICAL VIGNETTE

# Acute Traumatic Central Cord Syndrome

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Traumatic central cord syndrome is a condition of acute neurological impairment resulting from cervical spine trauma causing spinal cord damage and edema. The neurological impairment is characterized by a disproportionate weakness of the upper greater than lower extremities and may also include bladder dysfunction and varying degrees of sensory loss. The mechanism and pathophysiology of the spinal cord injury are well-described. The natural history of the syndrome varies, with some patients experiencing complete spontaneous recovery and others having persistent neurological deficits. The role of surgical intervention remains controversial. We present an elderly patient who had a ground-level fall that led to central cord syndrome.

### *Case Report*

An 84-year-old woman with no significant previous medical problems was walking on the sidewalk while shopping, slipped on a banana peel, fell onto the left side of her body, and struck the left side of her head on the ground. The fall was determined to be mechanical in nature as the patient denied palpitations, dizziness, chest pain, headache, visual changes, preceding muscle weakness, bowel or bladder incontinence, or loss of consciousness. After the fall, the patient was unable to stand up. She reported right-arm weakness and numbness in her left hand and fingers and both feet and toes. Bystanders called the paramedics and the patient was brought to the Emergency Department for further evaluation.

Initial physical examination was remarkable for normal vital signs other than a blood pressure of 166/73 mmHg. Cardiopulmonary examination was unremarkable. Neurological assessment was notable: normal mental status, memory, and concentration. Speech was fluent and the cranial nerve examination was normal. Motor examination revealed normal strength (5/5) in the left upper extremity and bilateral lower extremities proximally and distally in all muscle groups. However, in the right upper

extremity, proximal strength (deltoid 0/5, triceps 2/5, biceps 2/5) and distal strength (wrist 2/5, fingers and grip 3/5) was markedly reduced (2/5). Sensation to light, touch, temperature, and pain was intact. Deep tendon reflexes were normal in the left upper extremity and bilateral lower extremities but were absent throughout the right upper extremity.

Laboratory testing revealed a white blood cell count (WBC) of 20,000/ $\mu$ l and pyuria consistent with a urinary tract infection. The remainder of the complete blood count and basic chemistry panel was within normal limits. Chest radiograph and electrocardiogram were unremarkable. Computed tomography (CT) scan of the brain without contrast showed multiple old small lacunar infarcts, but no evidence of recent infarction. In addition, a left parietal scalp hematoma and mild chronic microvascular ischemic changes were seen. There was no evidence of an underlying skull fracture or intracranial bleeding. CT scan of the cervical spine showed no evidence of acute bony fracture or subluxation. However, there was severe degenerative disc disease and cervical spondylosis from C4-C5 through the C6-C7 disc levels, causing spinal stenosis at these levels.

Due to concern for an acute stroke causing the patient's neurological deficits, magnetic resonance imaging (MRI) of the brain was performed, which showed no evidence of acute ischemia or intracranial hemorrhage. A left posterior scalp fluid collection consistent with a hematoma, small old lacunes in the deep nuclei, and mild microvascular ischemic changes of the cerebral white matter were again noted. Subsequently, Neurology consultation was obtained and MRI of the cervical spine was performed, which showed severe multilevel degenerative disc disease from C4-C5 through C6-C7, resulting in spinal canal stenosis. In addition, extensive T2-weighted signal abnormality was noted within the spinal cord from C3-C4 through C6, consistent with edema (**Figure 1**). There were no T1-weighted signal changes to suggest hemorrhage identified within the cord. There was also a significant amount of edema of the prevertebral soft tissues. These findings were compatible with recent trauma and the patient was diagnosed with 'traumatic central cord syndrome'.



**Figure 1.** MRI of the cervical spine showed multilevel degenerative disk disease at C4-5, C5-6, C6-7, and C7-T1 resulting in spinal canal stenosis at C4-5 through C6-7. There is extensive T2-weighted signal abnormality within the spinal cord consistent with edema, most prominent at the level of C4-5, but extending from approximately the level of C3-4 through the level of C6. There is no T1-weighted signal change to suggest hemorrhage identified within the cord.

On the following day, the numbness in the patient's left hand and bilateral feet had improved. Her right-arm motor strength remained severely impaired. Due to the findings on the cervical spine MRI and the patient's persistent symptoms, a Neurosurgery consultation was obtained. The neurosurgeon advised that the patient was at risk for quadriplegia if she had another fall or other traumatic injury to her neck. Furthermore, it was recommended that the patient wear an aspen collar at all times and undergo cervical decompression and fusion surgery in the near future when the spinal cord edema subsided. After being fully informed, the patient elected not to undergo surgery and was subsequently discharged to a rehab facility for physical and occupational therapy. At a follow-up appointment several weeks later, the patient had persistent severe right-arm weakness and continued to decline surgical treatment. She was subsequently lost to follow-up.

### **Discussion**

Traumatic central cord syndrome was recognized as

early as the late 1880s.<sup>1</sup> Schneider et al first described the syndrome in detail in 1954.<sup>2</sup> The original description noted that the syndrome was "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".<sup>2</sup> This classic description of central cord syndrome is not present in all patients and varying levels of neurological compromise can be seen. Some patients may present with only upper extremity weakness and variable sensory impairments without lower extremity weakness or bladder dysfunction.<sup>3,4</sup>

Traumatic central cord syndrome typically occurs in elderly individuals with significant cervical degenerative disc disease causing cervical stenosis. The mechanism of injury is believed to be hyperextension of the neck with pinching of the spinal cord between a thickened ligamentum flavum and a protruding anterior disc or osteophyte.<sup>2,5</sup> A ground level fall in an elderly patient can produce this mechanism of injury. Young

people without degenerative disc disease are also susceptible to this type of injury if the force of the trauma is great enough. Central cord syndrome is described resulting from high-force injuries from motor vehicle accidents, diving accidents, skiing or snowboarding accidents, and weight lifting. The mechanism in these situations has been described as hyperextension/flexion, cervical fracture, or herniated disc.<sup>6-9</sup>

When central cord syndrome was originally described it was believed that hematomyelia (intramedullary bleeding) was responsible for the neurological impairments.<sup>2</sup> A better understanding of the pathophysiology of the spinal cord injury leading to central cord syndrome has occurred with the advancements in medical technology including the availability of MRI. This has improved understanding of the disproportionate loss of upper extremity strength that is characteristic of central cord syndrome. Collignon et al evaluated 18 cases of acute traumatic central cord syndrome with MRI where none of the patients had evidence of intramedullary blood based on MRI signal changes (lack of T1-weighted signal changes).<sup>10</sup> A subsequent study also demonstrated that traumatic central cord injury patients did not have hematomyelia, but rather developed edema (T2-weighted signal abnormality) of the cord from the trauma with subsequent degeneration.<sup>11</sup>

In addition to evidence from MRI studies, post-mortem histopathological examination was performed on 3 patients who died between 3 days and 7 months after the initial injury and showed no evidence of intramedullary hematoma. These exams noted spinal cord edema 3 days after the initial injury with separation of axon-myelin units and some developing myelin breakdown. At 6 weeks after the injury, marked axonal breakdown was noted. At 7 months after the injury, loss of myelin and axons was seen with the loss preferentially confined to larger-diameter axons and sparing the smaller-diameter axons. This axonal loss was seen in the white matter lateral corticospinal tracts and not in the central gray matter. The medial part of the lateral corticospinal tracts was most affected.<sup>11</sup> This finding explains why the upper extremities are more affected in central cord syndrome as the medial part of the lateral corticospinal tracts innervates the upper extremities whereas the lateral aspect innervates the lower

extremities. A subsequent histopathological study involving 5 patients confirmed these findings.<sup>12</sup>

The natural history of central cord syndrome is quite variable. Some patients will experience spontaneous complete neurological recovery whereas others have persistent deficits. Recovery typically follows a pattern with lower extremity motor function returning first, followed by recovery of bladder function, and finally return of motor function of the upper extremities.<sup>6</sup> Motor function of the hand is typically last to return. Recovery of sensory function varies and may not be concurrent with the return of motor function. Studies have shown that overall between 50% and 80% of patients with central cord syndrome will recover spontaneously over time.<sup>13</sup> Several risk factors that portend a poor prognosis for complete

**Table 1. Risk Factors for a Poor Prognosis for Recovery from Central Cord Syndrome.**

1. Age greater than 50 years old
2. More severe neurological deficit at the time of initial presentation
3. Evidence of cord edema on cervical MRI
4. Cervical stenosis with cervical spine canal size of less than 14 mm
5. Multiple-level injury
6. Lack of recovery of function during the first few days following injury

recovery have been described (**Table 1**).<sup>5,13-15</sup>

Treatment for traumatic central cord syndrome remains controversial. Traditionally, it was not recommended to undergo surgical intervention as available evidence showed that patients who underwent surgical cervical decompression had worse neurological outcomes than those who did not undergo operations.<sup>2</sup> However, subsequent studies have demonstrated that surgical decompression may actually improve neurological symptoms in some patients. Brodkey et al performed surgical decompression on 7 patients with central cord syndrome whose recovery had plateaued or worsened after conservative treatment. These patients were documented to have ongoing stenosis of the cervical spine. Despite persistence of some neurological deficits, it was shown that surgical decompression for these patients improved symptoms overall. Furthermore, 3 of the 7 patients returned to normal function.<sup>16</sup>

In a subsequent study, Chen et al performed a prospective analysis of 37 patients with cervical spondylosis and incomplete traumatic cord injury.<sup>17</sup> Sixteen patients were treated with cervical decompression surgery and 21 patients were treated non-operatively. Compared to conservative treatment, it was shown that the group undergoing surgical decompression had faster recovery of neurologic function, earlier mobilization, briefer hospital stays, improved long-term neurological outcome, and fewer complications related to prolonged bed confinement. The differences in recovery were statistically significant in favor of the surgical group at 1 month and 6 months follow-up ( $P < 0.002$  and  $P = 0.005$ , respectively). However, at 2-year follow-up, there was no statistical difference in neurological recovery between the two groups ( $P = 0.06$ ).<sup>17</sup> This study illustrated that surgical intervention may expedite neurological recovery but that long-term recovery at 2 years appears unaffected compared to conservative management.

Other investigators performed a retrospective analysis of early (= 24 hours after injury) versus late (> 24 hours after injury) surgery on patients with traumatic central cord syndrome. In the early treatment group, there was a shorter ICU stay and total length of hospital stay. Also, those patients with acute disc herniation or fracture/dislocation showed better overall motor improvement when having early surgical treatment ( $P = 0.04$ ). However, in patients with spinal stenosis or spondylosis, early surgical treatment did not improve motor outcome compared to late surgical treatment ( $P = 0.51$ ).<sup>18</sup> Based upon the available evidence, surgical intervention for traumatic cord syndrome remains controversial and it is important to evaluate patients on an individual basis with the assistance of a spinal surgeon to determine the most appropriate treatment plan.

The patient we described above had multiple risk factors indicating a poor prognosis for neurological recovery including advanced age, multilevel cervical stenosis and cord edema on MRI, severe neurological impairment on presentation, and limited recovery over the first few days following the injury. Not surprisingly, her neurological deficits remained significant at follow-up several weeks after the initial injury. Surgical decompression and fusion was recommended and it would likely offer protection in

the event of another traumatic injury but it is unclear if it would have improved her neurological deficits.

### Conclusion

In conclusion, traumatic central cord syndrome should be suspected when patients present with disproportionate weakness of the upper extremities greater than lower extremities. The injury often occurs in elderly patients with significant degenerative disc disease, causing cervical stenosis after a simple fall. Younger people with high-force injuries to the cervical spine can also present with central cord syndrome in the absence of underlying cervical degenerative disc disease. Pathophysiologically, the injury involves myelin loss and axonal damage, mainly in lateral corticospinal tracts. Many patients will have spontaneous neurological improvement over time but certain risk factors are known to signify a poor prognosis. The need for and timing of surgical decompression is controversial. Surgery may expedite neurological recovery in some patients and may provide additional neurological recovery when clinical improvement has plateaued or worsened. However, surgical intervention has not been shown to consistently improve long-term neurological outcomes. We recommend evaluating patients on an individual basis with the assistance of a spinal surgeon to determine the most appropriate treatment plan.

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Submitted on December 7, 2008