Conus medullaris syndrome without lower-extremity involvement in L-1 burst fractures

Report of four cases

Dean Chou, M.D., Roger Hartl, M.D., and Volker K. H. Sonntag, M.D.
Division of Neurological Surgery, Barrow Neurological Institute, St. Joseph's Hospital and Medical Center, Phoenix, Arizona

Conus medullaris syndrome has been described as affecting the lower sacral segments of the spinal cord. It usually is attributable to lesions at the conus medullaris. Adams and Victor described it as a constellation of signs and symptoms including the following: bowel and bladder dysfunction, sexual dysfunction, poor rectal tone, perianal sensory changes, and sometimes, lower-extremity weakness. Isolated conus medullaris syndrome has been related to intradural pathological conditions such as tumor or vascular lesions, but isolated conus medullaris injuries seldom occur without lower-extremity involvement from thoracolumbar burst fractures. As many as 50% or more of burst fractures at the thoracolumbar junction are associated with a neurological deficit. If bowel, bladder, and sexual dysfunction occur, there is usually concomitant motor weakness in the lower extremities. To our knowledge, only one case of an L-1 burst fracture causing conus medullaris syndrome without lower-extremity involvement has been reported. We report four cases of isolated conus medullaris syndrome, without lower-extremity involvement, which was caused by L-1 burst fractures.

Case Reports

Case 1

Examination. This 35-year-old man was skydiving and landed poorly. An L-1 burst fracture was diagnosed at an outside institution and the patient was transferred to our institution. On examination, the patient had Grade 5/5 muscle strength and no sensory changes in his lower extremities. Sensation around his scrotum and penis was slightly decreased, but his rectal tone was normal. His bladder was visibly distended, and postvoid residual catheterization revealed 1000 ml of retained urine.

Operation. Computerized tomography scanning demonstrated an L-1 burst fracture associated with 80% compromise of the spinal canal and 20% loss in the height of the vertebra. Within 24 hours of injury, the patient underwent an L-1 corpectomy, decompression, and reconstruction with an expandable cage and anterior plate from T-12 to L-2.

Postoperative Course. Postoperatively, the patient had full motor strength and no sensory changes in his lower extremities, but he did not regain vesicorectal function. At his 1-year follow-up examination, the patient was ambulating well but had slight urinary, bowel, and erectile dysfunctions.

Case 2

Examination. This 41-year-old man was riding his motorcycle in sand dunes when he encountered a 7-ft vertical drop. He landed on the ground on his buttocks. At an outside institution, an L-1 burst fracture was diagnosed. A Foley catheter was inserted, and the patient was transferred to our institution. On examination, the patient had a Grade 5/5 motor strength in both lower extremities but experienced perianal tingling. His sensation was slightly decreased around the scrotum, but his rectal tone was normal. A CT scan showed an L-1 burst fracture that had

Abbreviations used in this paper: CT = computerized tomography; MR = magnetic resonance.
resulted in approximately 60% compromise of the spinal canal. The vertebra had lost 10% of its height.

Operation. The patient underwent internal pedicle screw fixation from T-11 to L-3, with internal distraction to reduce the fragment away from the spinal canal. Postoperatively, the patient’s motor strength in his legs was intact, and the Foley catheter was removed. He was unable to void, however, and all narcotic agents were stopped. The patient continued to have urinary retention. A CT scan showed persistent fragments in the spinal canal, and the patient was returned to the operating room for an L-1 laminectomy. Postoperative MR imaging showed no compression of the conus medullaris after the laminectomy.

Postoperative Course. After decompression, the patient had full motor strength in both lower extremities and could walk without difficulty. At his 15-month follow-up examination, however, he continued to have urinary dysfunction, sexual dysfunction, and abnormal sphincter control. He was able to achieve an erection, but he could not ejaculate. Compared with his initial presentation, erectile function gradually improved, but it has not yet returned to normal.

Case 3

Examination. This 38-year-old man was trimming a palm tree when he fell approximately 25 ft to the ground. At an outside institution, an L-1 burst fracture was diagnosed and the patient was transferred to our institution for definitive care. On presentation, the patient had Grade 5/5 muscle strength in both lower extremities and no sensory changes in his hands. A formal voiding trial was not conducted, but his rectal tone was decreased. A CT scan showed 90% compromise of the spinal canal and 70% loss of vertebral height (Fig. 1A and B).

Operation and Postoperative Course. Within 24 hours of the injury, he underwent an L-1 corpectomy. The spinal canal was decompressed completely, and T-12 to L-2 was reconstructed with an expandable cage and anterior thoracolumbar plate (Fig. 1C and D).

Postoperative CT scanning confirmed good decompression of the spinal canal and appropriate positioning of the hardware. Immediately after surgery, the patient had full lower-extremity strength and no difficulty ambulating, but he did have urinary retention and bowel dysfunction. At his 1-month follow-up examination, he continued to have vesicorectal and ejaculatory dysfunction despite having Grade 5/5 muscle strength in both legs. He was subsequently lost to follow up.

Case 4

Examination. This 28-year-old man was riding an all-terrain vehicle and crashed. He subsequently ambulated but continued to have back pain. Seven days after his accident, he sought medical attention for severe back pain. He had difficulty starting his urinary stream and sensing when he was finished urinating. When he had bowel movements, they felt like “sandpaper.” Physical examination demonstrated 5/5 strength in all muscle groups in the lower extremities and no sensory changes. A CT scan showed an L-1 burst fracture associated with 15% compromise of the spinal canal and 10° of kyphosis.

Treatment and Follow Up. Despite the patient’s bowel and bladder abnormalities, he wished to pursue conservative therapy. Thus, a thoracolumbar orthosis was applied and he was discharged home. His bowel and bladder dysfunction persisted for 4 months after his initial injury and then returned to normal. At all times, his sexual function was normal. At his 6-month follow-up examination, the patient had normal bowel and bladder function, and his lower-extremity strength and sensation were intact.

Discussion

Classically, a pure conus medullaris syndrome has been described as signs consisting of paralytic bladder incontinence, bowel incontinence, impotence, perineal sensory changes, and absence of lower-extremity weakness. Isolated conus medullaris syndrome has been caused by various intradural pathological entities and even by diabetic neuropathy. However, it is extremely rare for a conus medullaris syndrome to be present without lower-extremity involvement associated with an extradural or traumatic process.

One case of isolated bowel and bladder dysfunction caused by an osteoporotic burst fracture has been reported. In 1993, Tanaka, et al. reported on a 73-year-old man who had fallen and in whom an L-1 compression fracture was initially diagnosed. The patient had no symptoms, but 2 months later he experienced urinary incontinence. At that time, radiographs showed an L-1 burst fracture. He had only bowel and bladder dysfunction; motor function in his lower extremities was normal. The patient underwent surgical decompression and regained vesicorectal functioning 1 year after surgery.

Dall and Stauf fer observed 14 patients with T-12 or L-1 burst fractures. At presentation, all had motor weakness except one patient, who had only bladder dysfunction. After undergoing decompression of the spinal canal, this patient regained bladder sphincter function and his lower-extremity strength remained intact. All of the other patients who presented with bladder dysfunction also had motor weakness.

Watanabe, et al. reported that the incidence of occult neurogenic bladder was as high as 41% in patients with intact neurological function of the lower extremities after a thoracolumbar fracture. The patients, whose neurological status American Spinal Injury Association Grade E (an otherwise completely intact neurological function), had occult neurogenic bladder based on formal urodynamic studies. The report was from a urological perspective. The authors did not focus on whether bowel function and sexual function were also affected. Although the most common level of injury was at L-1, it is unclear whether the level of injury correlated with the incidence of neurogenic bladder. Moreover, the authors did not report whether the patients were actually symptomatic. Rather, they diagnosed neurogenic bladder solely through urodynamic studies. Thus, while they examined 17 patients who were otherwise neurologically intact, the number of patients who complained of symptoms of bladder or bowel dysfunction is unclear.

Of our four patients, two underwent complete decompression within 24 hours of injury, one underwent com-
complete decompression within 72 hours, and one did not undergo surgery. The patient in Case 2 underwent partial decompression by posterior distraction within 24 hours of injury; subsequently, complete decompression was accomplished by laminectomy within 72 hours. Although laminectomy is a posterior decompressive approach to an anterior pathological process, a postoperative MR image confirmed that there was no further compression of the conus medullaris. Regardless of the time to decompression in our patients who underwent surgery and were available for follow-up review, their conus medullaris syndromes persisted and had not resolved at their 12- and 15-month follow-up examinations. After 4 months of conservative brace therapy, the one patient who chose non-surgical treatment regained normal bowel and bladder function; however, this patient also had the least amount of spinal canal compromise compared with the other three patients. One patient was lost to follow up. Motor strength was excellent in all four patients at injury, and they experienced no sensory changes in their lower extremities.

All four of these cases involved burst fractures at L-1. Moreover, the patient reported on by Tanaka, et al.,16 and the patient reported on by Dall and Stauffer2 both had injuries at L-1. Injury at this location usually corresponds to the conus medullaris. Huertas7 has stated that an extradural pathological entity concomitantly affects lower-extremity strength because the cauda equina is also involved with extradural compression. Intradural lesions located at the conus medullaris may exert little mass effect and, consequently, are less likely to compress the cauda equina. One potential explanation of this rare clinical profile associated with L-1 burst fractures is that the efferent neurons of the sacral voiding center located at the conus medullaris may be extremely sensitive to injury during the initial shock and retropulsion of the L-1 vertebral body. In contrast, the passing axons to the lower extremities may be sturdier and better able to withstand such a trauma; however, more studies are needed to elucidate the underlying pathophysiology.

Interestingly, the spinal canal in our one patient who recovered bowel and bladder function with conservative therapy was only 15% compromised. In contrast, the spin-

FIG. 1. Case 3. Preoperative axial (A) and sagittal (B) CT scans demonstrating a burst fracture of L-1. Axial (C) and sagittal (D) CT scans obtained after the patient underwent a corpectomy and reconstruction with an expandable cage and anterior plate.
cal canals of the two patients who underwent surgery but did not recover function after decompression were 60 and 80% compromised, respectively. Studies have evaluated the cross-sectional area of spinal canal compromise and the incidence of neurological deficit. Kilcoyne, et al., 5 and Gertzbein et al., 6 found no correlation between neurological impairment and the degree of spinal canal compromise demonstrated on CT scanning. Lindahl, et al., 12 Keene, et al., 8 and Dall and Stauffer 2 also found no relationship between spinal canal compromise and neurological injury. Limb, et al., 11 evaluated 20 patients with burst fractures. They postulated that the injury occurs at the time of trauma rather than resulting from pressure caused by fragments in the spinal canal. They found no correlation between the degree of spinal canal disruption and the degree of neurological compromise; however, the force of the traumatic injury and the degree or severity of neurological injury were correlated.

Denis, 3 however, found that compromise of the spinal canal was associated with neurological injury more frequently at the conus medullaris than at the cauda equina. Hashimoto, et al., 6 found that L-1 burst fractures that compromised more than 45% of the spinal canal were associated with neurological injury. Fontijne, et al., 4 evaluated 139 patients with burst fractures and correlated the probability of neurological injury with the level injured and the degree of neurological compromise; however, the force of the traumatic injury and the degree or severity of neurological injury were correlated.

Denis, 3 however, found that compromise of the spinal canal was associated with neurological injury more frequently at the conus medullaris than at the cauda equina. Hashimoto, et al., 6 found that L-1 burst fractures that compromised more than 45% of the spinal canal were associated with neurological injury. Fontijne, et al., 4 evaluated 139 patients with burst fractures and correlated the probability of neurological injury with the level injured and the degree of neurological compromise observed on CT scans. Trafton and Boyd 17 found that the risk of neurological injury associated with L-1 or T-12 burst fractures increased significantly when the spinal canal was compromised 50% or more. Kim, et al., 10 found a significant correlation between spinal canal compromise and neurological deficit. Patients who presented with a neurological deficit had sustained 49% impingement of the spinal canal at the conus medullaris, whereas those who presented without a neurological deficit had sustained only 34% impingement at the level of the conus. Thus, many studies have reported that neurological injury is associated with severity of burst fractures. However, authors of most of the studies reported motor weakness and bowel and bladder injury as combined injuries.

During examination of patients with conus medullaris syndrome, knowledge of the anatomical pathways in the lower sacral region is helpful. The sacral voiding center, located at the conus medullaris, is composed of autonomic and somatic components. Parasympathetic innervation provides the excitatory input to the bladder. Sympathetic innervation stimulates the smooth muscle of the urethra and base of the bladder but provides inhibitory input to the smooth muscle within the bladder wall itself. The S2–4 segments of the spinal cord give rise to both the parasympathetic and somatic efferent nerves to the external sphincter. The T11–L2 segments give rise to the sympathetic nerves in this region. The dorsolateral spinohalamic tracts are close to the autonomic tracts that supply the bladder. Thus, intact pinprick sensation in the lower sacral dermatomal distribution may portend a favorable prognosis for recovery of bladder function. The presence of the bulbocavernous reflex indicates that the arc between the pelvic afferents and the sacral cord efferents is intact, again portending a good prognosis. Formal urodynamic studies are important if there is a question of bladder dysfunction. 19

Although our sample is small, the course of these four patients shows that isolated conus medullaris syndrome without lower-extremity weakness can be associated with L-1 burst fractures. Unlike in the patient reported on by Tanaka, et al., 16 bowel and bladder function did not recover in our two patients who underwent surgery. It did, however, in our patient who chose conservative treatment. Isolated conus medullaris syndrome should be considered in patients with L-1 burst fractures and intact motor strength, adding another consideration to the surgical decision-making process. Underreporting of this clinical picture may decrease awareness of its existence. Because our numbers are small, it is unclear whether surgical intervention alters the outcome in patients with this pathological entity.

Conclusions

Conus medullaris syndrome without lower-extremity involvement can be associated with L-1 burst fractures. Despite immediate decompression, vesicorectal function may continue to be abnormal. Bowel and bladder function can recover spontaneously with conservative therapy. Even with surgical decompression, the prognosis is unclear. Further studies evaluating recovery from isolated conus medullaris syndrome are needed.

References

12. Lindahl S, Willen J, Irsland L: Computed tomography of bone
Conus medullaris syndrome in L-1 burst fractures


Manuscript received July 2, 2004. Accepted in final form December 21, 2005.

Address for Dr. Chou: University of California San Francisco, California.

Address for Dr. Hartl: Weill Cornell Medical College, New York, New York.

Address reprint requests to: Volker K. H. Sonntag, M.D., c/o Neuroscience Publications, Barrow Neurological Institute, 350 West Thomas Road, Phoenix, Arizona 85013; email: neuropub@chw.edu.