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Cauda Equina Syndrome: Is It a Surgical Emergency?

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Introduction

Cauda equina syndrome is defined as the compression of the nerve roots distal to L1 secondary to acute disc herniation, bony fragments, tumor, infection, or postsurgical intervention. The result is a complex of symptoms consisting of low back pain, unilateral or bilateral sciatica, motor weakness of the lower extremities, sensory disturbances, and loss of bowel or bladder function. The literature is clear that the early diagnosis of this entity is crucial and may be difficult, particularly if the patient does not present with all of the aforementioned signs and symptoms. Conversely, the literature is unclear as to the optimal timing of intervention. Discussion in the literature ranges from the need for emergent early decompression to no adverse effects from delayed decompression. This discussion reviews the current literature and attempts to draw a conclusion about the optimal timing for surgical intervention in patients with cauda equina syndrome.

Anatomy

In the adult, the conus medullaris represents the termination of the spinal cord in the proximal lumbar spine. The conus is narrower than the more cephalad portion of the spinal cord and usually overlies the body of L1. The conus continues to taper to form the filum terminale. Proximal to the conus medullaris there is an enlargement of the lumbar spinal cord. It is this area where the lumbar nerves arise with the lumbar sympathetics. The sacral parasympathetic nerves and the sacral sensory nerves arise from the conus itself. The lumbar nerves join the sacral nerves to form the cauda equina or *horse's tail* [1,2].

The lumbar and sacral nerve roots contain sensory and motor function for the lower extremities, sensation to the perineum and genitals, and they also innervate the pelvic viscera. Voluntary and involuntary functions are also contained within the nerve roots of the cauda equina and are necessary for micturation, defecation, and sexual function. Compression of the cauda equina may involve all of the above functions, sensory only, motor only, or only those roots responsible for bowel and bladder function. Thus, the anatomy of the distal spinal cord and the cauda equina is responsible for the variability in presenting signs and symptoms.

Presentation

The lesions that produce cauda equina syndrome include fracture, tumor, pyogenic infection, spinal stenosis, and disc herniation. Rarely is there complete paralysis of all sensory and motor function of the pelvic viscera as well as the lower extremities. More often there is a varying degree of symptoms consisting of low back pain, unilateral or bilateral sciatica, motor weakness of the lower extremities, sensory disturbance, and loss of visceral function together with saddle anesthesia [2]. The presentation may be subtle with vague history of back pain and urinary retention. The classic minimal definition as defined by Scott [3] is bowel and bladder dysfunction caused by compression of the cauda equina but not the conus medullaris. Disc herniation affecting the lower sacral roots may present in this manner, with no sensory or motor changes in the lower extremities. To date, there is no correlation between the severity of the symptoms at onset and prognosis for outcome. Gleave and MacFarlane [4] postulated that the rapidity by which the compression occurs is of prognostic significance. However, this has not been supported by experimental models of cauda equina compression [9].

There is also no definite correlation between the size of herniated discal material and outcome. Kostuik et al. [8] postulated that of all prognostic indicators, the presence of a dense sensory deficit in a saddle distribution carried the poorest prognosis with respect to recovery of bowel and bladder function.

The incidence of cauda equina syndrome secondary to lumbar disc prolapse is reported in the literature to be between 2–6% [4]. Two populations of patient presentations have been defined in the literature. Several authors [5,6,8,10] have noted an acute type of presentation as well as a chronic type. The acute type usually presents in a younger patient with no previous history of symptoms. Symptoms develop relatively rapidly with sudden onset of back pain, sciatica, urinary retention or incontinence, and variable motor and sensory deficits (Fig. 1). The chronic variety usually presents with a more insidious onset. These patients are very often older and have a history of previous symptoms that include sciatica, neurogenic claudication,

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and intermittent retentive or incontinent episodes. It is not uncommon for these patients to have underlying spinal stenosis. Symptoms usually progressively worsen over a period of months to years. This second group can often pose more of a diagnostic challenge. Symptoms may be misinterpreted in this group as simply an exacerbation of previous back complaints or urinary changes secondary to aging.

Review of the Literature

Cauda equina syndrome is considered an absolute indication for decompression. However, the timing of this decompression, particularly in the presence of an acute disc herniation, is not clear in the literature and is a topic of considerable debate.

Many authors favor urgent and even emergent surgical intervention. Early studies recommended surgical decompression within 6 hours to maximize neurologic recovery. Dinning and Schaeffer [5] retrospectively reviewed 39 patients with acute cauda equina syndrome secondary to a herniated lumbar disc. Patients with stenosis or burst fracture were excluded. Of their 39 patients, 21 patients were operated on within 24 hours. Twenty-two patients presented with bowel or bladder complaints and 77% recovered normal function. The authors did not clarify or quantify the functional recovery in the early versus late group other than noting a "highly significant difference" in outcome of bladder function in most cases decompressed within 24 hours of paralysis compared to those operated on after 24 hours.

Shapiro [6] retrospectively evaluated 14 patients with cauda equina syndrome from herniated lumbar discs. Ninety-three percent of his patients developed or presented with bowel and/or bladder incontinence. The timing of surgical intervention ranged from less than 24 hours to more than 30 days. All (100%) of the patients with urinary or bowel incontinence who were decompressed within 48 hours regained bowel and bladder control. Only 33% of those operated on after 48 hours regained control. The author also noted a trend toward a decrease in chronic sciatic-type pain in those decompressed early. He recommends intervention within 24–48 hours after the onset of symptoms to obtain an improved outcome. Shapiro noted an overall improvement in outcome even if the decompression was performed late, although the degree of improvement was less than with early decompression.

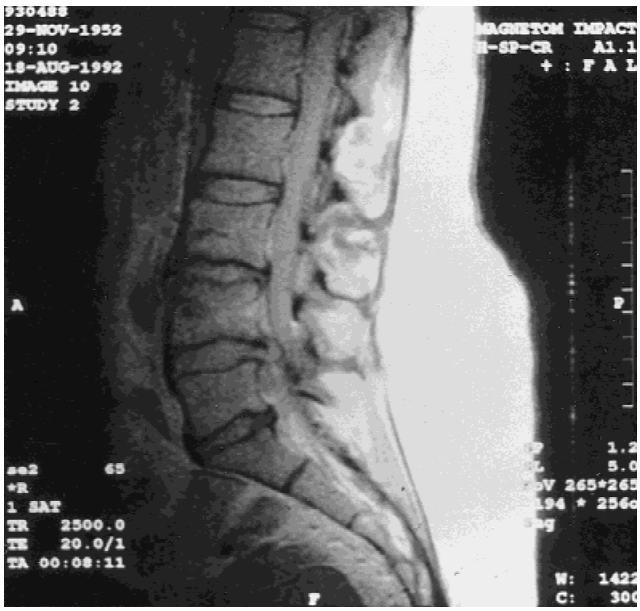

Fig. 1. MRI of a 46-year-old woman who presented with acute onset of sciatica, saddle anesthesia, and urinary retention for 24 hours. T1-weighted image shows L4-L5 disc herniation with compression of thecal elements. Patient underwent emergent decompression and complete recovery of neurologic function.

Mclaren and Bailey [7] retrospectively reviewed six cases of postdiscectomy cauda equina syndrome. Decompression was performed within 24 hours in four cases (three of the four were decompressed within 6 hours). Recovery of bowel and bladder function was noted in the four patients decompressed early whereas the late group did not regain function. The recovery of motor function was less clear but those with milder symptoms preoperatively tended to have greater improvement. Sensory recovery in both groups was noted to be good. The authors concluded that for optimal outcome, the decompression must be performed early before the motor deficit becomes too severe.

There is also evidence in the literature to support the fact that it may not make a difference in outcome if the decompression is done as an emergency or within the first few days. Kostuik et al. [8] retrospectively reviewed 31 patients with cauda equina syndrome secondary to lumbar disc herniation. They recognized two distinct populations of patients. One group had acute onset of sciatica, urinary retention, saddle parasthesias, and motor weakness with no antecedent pain. This group underwent decompression between 6 and 48 hours after the onset of symptoms. The second group had a more insidious onset of symptoms with gradual motor, sensory, and urinary abnormalities. Patients in this group reported intermittent bowel and bladder abnormalities for several months. The time to decompression for this group was between 1–5 days. The authors found no correlation between the length of time from the onset of symptoms to surgery and the extent of neurologic recovery. They found excellent recovery in 27 of 30 patients. They also noted no correlation between the severity of symptoms or clinical findings at the onset and the extent of neural and bladder function recovery. They concluded that although early decompression seems logical, they obtained excellent results with none of their patients being operated on before 6 hours.

Delamarter et al. [9] provided further evidence, in an animal model, that immediate decompression may not improve outcome. They developed a canine model for cauda equina syndrome and evaluated neurologic recovery following immediate (2–3 seconds), early (1 hour, 6 hours), and delayed (24 hours, 1 week) decompression. No significant changes in neurologic recovery were noted among the groups. There was no statistically significant change in histologic neuroanatomy or in recovery of somatosensory evoked potentials. The early neurologic recovery was vari-

able depending on the length of compression, although the eventual neurologic recovery at 6 weeks was identical. The authors concluded that based on their results, decompression of cauda equina syndrome is not a surgical emergency.

Interestingly, in a subsequent study, Delamarter et al. [10] evaluated the effect of timing in decompression of the spinal cord itself. In a canine model, they showed no neurologic recovery when the decompression was delayed longer than 6 hours. Significant recovery potential was noted if decompression was performed before 6 hours. Remyelination and axonal regeneration, which played a significant role for neurologic recovery of the cauda equina, did not provide neurologic recovery after cord decompression.

Rydevik et al. [11] developed a porcine model to evaluate the neurophysiologic changes in the cauda equina with increasing compressive pressures. They showed a threshold pressure (50–70 mmHg) below which full functional recovery could be expected. Pressures greater than 70 mmHg were consistently associated with residual neurologic deficit.

Discussion

The literature has shown that recovery after acute cauda equina syndrome cannot be clearly correlated to the rapidity of onset of the symptoms, the amount of protruded disc material, or to the severity of neurologic findings at presentation. Cauda equina syndrome presents in four general patient populations: (1) older patients with gradual onset of symptoms in whom imaging studies reveal severe stenosis, facet hypertrophy, infolding of the ligamentum flavum, and segmental translation; (2) younger patients with acute onset of symptoms related to herniated disc material or fracture fragments after trauma; (3) patients immediately postsurgery (24–72 hours) secondary to epidural hematoma; and rarely (4) patients with compression secondary to tumor or infection who will present with acute onset of symptoms. Clinical and experimental studies tend to support that the timing of the decompression is not the sole factor in gaining a favorable outcome since recovery has been documented by several authors with delayed decompression. The question then becomes: What factor or variable accounts for the fact that some patients do well with acute decompression while others have excellent recovery with delayed decompression? The variable proposed is the magnitude (amount) of the compression that the contents of the thecal sac can withstand.

Delamarter et al. [9] showed that at a constant pressure (75% constriction of cauda diameter), the time to decompression did not play a role in functional or histologic outcome. They did not evaluate the effect of graded compression on recovery. Rydevik et al.'s data [11], which showed a decrease in functional recovery once a certain threshold pressure was surpassed, may provide insight to the different recovery patterns seen clinically [4,6,8]. The concept that compressed neural tissue below a threshold pressure may retain the ability to recover indefinitely may describe the excellent recovery rates described in both clinical and experimental studies with delayed decompression.

The favorable effect of early decompression may reside in the fact that decompression prevents pressures from reaching the critical level where neurologic sequelae are irreversible. Conversely, patients who have neurologic recovery with delayed decompression may have never reached the critical threshold pressure. Extrapolating from experimental data, the optimal predictor for recovery may lie in intrathecal pressure monitoring. At the present time, it is not possible or feasible to monitor and interpret compressive pressures in patients with acute cauda equina syndrome. The practice at our institution is to decompress patients with acute cauda equina syndrome as soon as possible after diagnosis since the threshold for irreversible neurologic injury is unknown.

Determination of a compressive threshold in humans may provide clearer guidelines for decompression in the future. Further clinical studies regarding intrathecal pressure monitoring as well as development of a technique to monitor intrathecal pressures may prove useful.

Summary

The timing of decompression in cauda equina syndrome secondary to disc herniation is not clearly defined in the literature. A careful review of the literature reveals neurologic recovery with both early and late decompression. This may be secondary to patients having a spectrum of compressive pressures of the cauda equina.

Patients with gradual onset or subthreshold pressures may respond favorably to decompression whenever it is performed. However, once the critical pressure is surpassed, neurologic deficit is inevitable. We are currently unable to measure intrathecal pressures and thus cannot quantify them clinically. Patients presenting with signs and symptoms of acute cauda equina syndrome should have an urgent/emergent magnetic resonance imaging (MRI) or computed tomography (CT) myelogram to correctly identify the level of compression followed by decompression as early as possible, so as to prevent irreversible neurologic sequelae.

Patients with a more chronic presentation and less severe symptoms should also undergo imaging modalities to determine the level of compression. However, decompression should be performed when medically feasible. Delays in decompression for medical optimization are probably warranted and are less likely to contribute to irreversible neurologic changes than in acute cauda equina syndrome.

References

1. Countee RW, Vijayanathan T, Martin BF, et al.: The conus medullaris: Physiological, anatomical and clinical considerations. In: Camino M and O'Leary P (eds). *The Lumbar Spine.* New York: Raven Press, pp. 171–182, 1987.
2. McNeil T: Pelvic visceral dysfunction: Cauda equina syndrome. In: Wiesel S, Weinstein J, Herkowitz H, et al. (eds). *The Lumbar Spine: The International Society for the Study of the Lumbar Spine* (2nd ed.). Philadelphia: W.B. Saunders, pp 569–581, 1996.

3. Scott P: Bladder paralysis in cauda equina lesions from disc prolapse. *J Bone Joint Surg* 47B:224–235, 1965.
4. Gleave J and MacFarlane R: Prognosis for recovery of bladder function following lumbar central disc prolapse. *Br J Neurosurg* 4:205–210, 1990.
5. Dinning T and Schaeffer H: Discogenic compression of the cauda equina: A surgical emergency. *Aust N Z J Surg* 63:927–934. 1993.
6. Shapiro S: Cauda equina syndrome secondary to lumbar disc herniation. *Neurosurgery* 32:743–747, 1993.
7. Mclaren A and Bailey S: Cauda equina syndrome: A complication of lumbar discectomy. *CORR* 204:143–149. 1986.
8. Kostuik J, Harrington I, Alexander D, et al.: Cauda equina syndrome and lumbar disc herniation. *J Bone Joint Surg* 68A:386–391, 1986.
9. Delamarter R, Sherman J, Carr J: Cauda equina syndrome: Neurologic recovery following immediate, early or late decompression. *Spine* 16:1022–1029, 1991.
10. Delamarter R, Sherman J, Carr J: Pathophysiology of spinal cord injury: Recovery after immediate and delayed decompression. *J Bone Joint Surg* 77A:1042–1049, 1995.
11. Rydevik BL, Pedowitz RA, Hargens AR, et al.: Effects of acute, graded compression on spinal nerve root function and structure: An experimental study of the pig cauda equina. *Spine* 16:487–493, 1991.