A Review Paper

Cauda Equina Syndrome: A Comprehensive Review

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Abstract

Cauda equina syndrome (CES) is a rare syndrome that has been described as a complex of symptoms and signs—low back pain, unilateral or bilateral sciatica, motor weakness of lower extremities, sensory disturbance in saddle area, and loss of visceral function—resulting from compression of the cauda equina. CES occurs in approximately 2% of cases of herniated lumbar discs and is one of the few spinal surgical emergencies.

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Anatomy

The vertebral column consists of 7 cervical vertebrae, 12 thoracic vertebrae, 5 lumbar vertebrae, 5 sacral vertebrae, and 4 coccygeal vertebrae. These vertebrae provide the bony structure that protects the spinal elements.[1-4] The spinal cord is housed by these bony elements and specifically is bordered anteriorly by the intervertebral discs and the anterior longitudinal ligament; posteriorly by the ligamentum flavum, lamina, and facet joints; and laterally by the pedicles.[4-7] The mean anterior-posterior diameter of the spinal canal is 12 mm.[3,8]

The meninges and the cerebrospinal fluid provide additional protection for the spinal canal.[9,10] The dura mater, the most superficial meninge, transforms into epineurium at the level of the dorsal root ganglion. In addition, the subarachnoid space ends at the dorsal root ganglion. Ligaments of Hoffman connect the dura mater and nerve roots to the posterior longitudinal ligament. The next layer of meninge, the arachnoid layer, lines the dural sac.[10-12] The deepest and microscopic layer is the pia mater, which closely invests the brain, spinal cord, and nerve roots and ultimately forms the filum terminale, which arises from the conus medullaris and anchors the spinal cord to the coccyx.[9-12]

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The cauda equina consists of nerve roots distal to the conus medullaris.[2,5] These nerve roots have both a dorsal root and a ventral root. The dorsal root consists of afferent fibers for transmission of sensation, and the ventral root provides motor fibers for the efferent pathway.[5-7] Orientation within the cauda equina is unique and specific. The most posterior neural elements within the sac are the fifth sacral nerve roots. In a cranial direction, the fifth sacral nerve roots progress anteriorly from the fourth through the first sacral vertebra. The most anterior element at the fifth lumbar and first sacral disc level is the first sacral nerve root.[3-5] The fifth lumbar nerve root enters anterolaterally between the fourth and fifth lumbar vertebrae—displacing the first sacral nerve root more posteriorly. Each subsequent nerve root continues this displacement, with one root added in the cephalad direction at each disc level. The motor fiber components are anteromedial, and the larger sensory components are posterolateral.[3-7]

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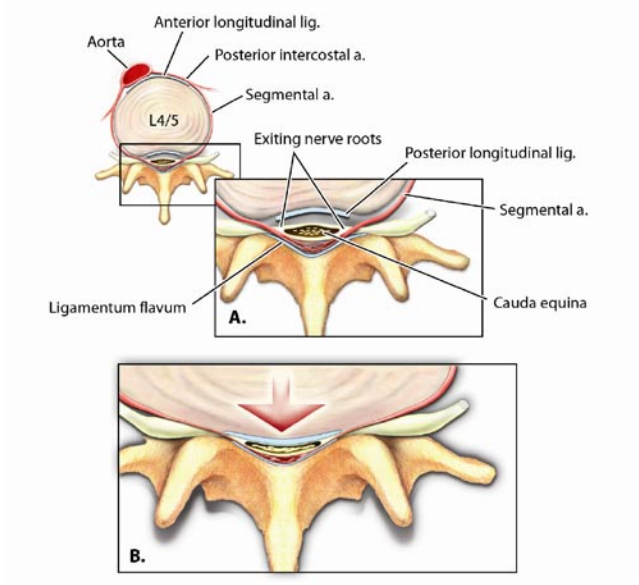

Figure 1. Cross-sectional anatomy of the cauda equina: (A) normal anatomy and (B) compression of cauda equina from disc herniation. Abbreviations: a., artery; lig., ligament. Copyright 2008, Kathleen Gebhart, CMI.

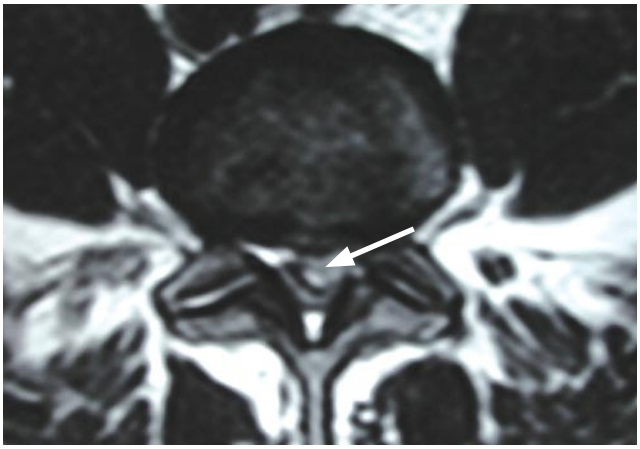

Figure 2. Axial T_2-weighted magnetic resonance imaging shows extruded disc fragment compressing nerve roots of cauda equina (white arrow).

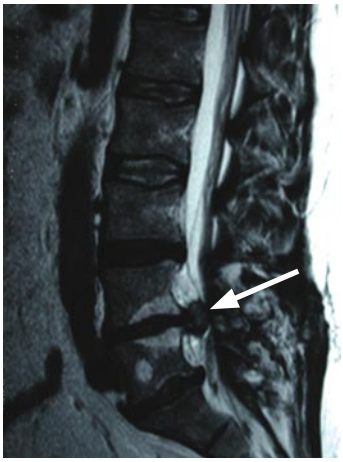

Figure 3. Sagittal T_2-weighted magnetic resonance imaging shows extruded disc fragment compressing nerve roots of cauda equina (white arrow).

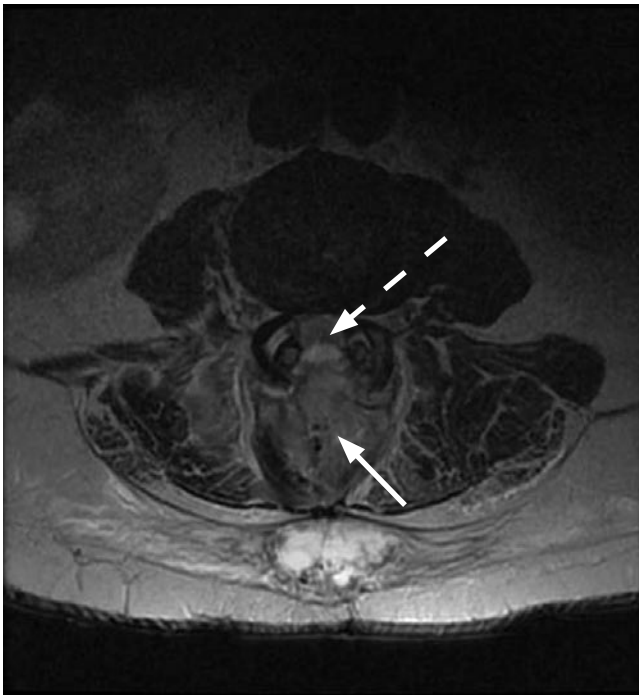

Figure 4. Axial T_2-weighted magnetic resonance imaging shows postoperative epidural hematoma. White solid arrow represents epidural hematoma after lumbar laminectomy; white dashed arrow represents thecal sac compression from hematoma.

Vascular Anatomy

The spinal cord depends on 3 longitudinal arterial feeders—the anterior spinal artery and 2 posterolateral spinal arteries.[13-16]

The anterior spinal artery extends from vertebral arteries to the conus medullaris, located over the median sulcus of the spinal cord. The anterior spinal artery supplies most of the internal substance of the cord and almost all the gray matter. However, the anastomosis between the anterior and posterior longitudinal system supplies the periphery of the white matter.[13,14]

The posterior spinal artery (dorsal spinal artery) also arises from the vertebral artery. It passes posteriorly, descending anterior to the posterior roots of the spinal nerves. The vasocorona is an anastomotic plexus formed by segmental feeders that reinforce the posterolateral spinal arteries.[13,16]

Medullary feeders course along the internal aspects of their respective roots. These feeders reinforce the longitudinal arterial system.[13-15] Ventral proximal radicular arteries branch from the anterior vasa corona and receive their blood supply from the anterior spinal artery. Distal radicular arteries are segmental ganglionic plexus branches that supply the distal part of the roots.[13,14] The filum terminale artery is a single artery that arises from the termination of the anterior spinal artery.[15] This artery travels anterior to the filum.

Etiology/Pathogenesis

CES has multiple etiologies (Figure 1). Trauma is an obvious source. Blunt or direct spinal injury through the cauda equina can cause significant injury. Sacral fractures, as described by Bonnin, can also cause CES.[3,17] Hematomas, abscesses, lymphoma, solid tumors, and other space-

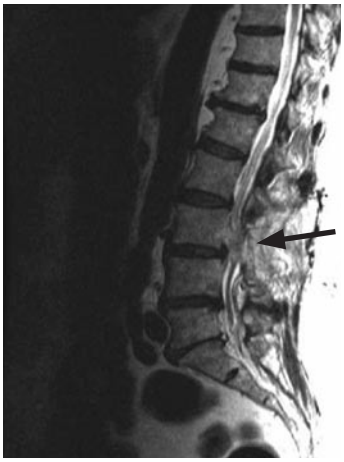

Figure 5. Sagittal T_2-weighted magnetic resonance imaging shows postoperative epidural hematoma. Black arrow represents thecal sac compression from epidural hematoma.

occupying lesions that compress nerve roots have been described as causes of CES.

Arslanoglu and Aygun[18] recently reported a case in which ankylosing spondylitis eroded the posterior elements and traction on the lumbar nerve roots and led to CES. Mohit and colleagues[19] described how an inferior vena cava thrombosis led to CES in a 16-year-old patient and how an inferior vena cava thrombectomy was required to relieve symptoms. The literature includes fewer than 20 reports of cases in which sarcoidosis caused CES; the most recent report, by Kaiboriboon and colleagues,[20] was published in 2005.

Nerve Root Compression

A well-described cause of CES is spinal stenosis with canal narrowing and ligamentum flavum infolding. Histologic examination of compressed nerve roots reveals congestion and dilation of intraradicular veins with infiltration of inflammatory cells. Magnetic resonance imaging (MRI) has been used to confirm edema of the cauda equina at the stenosed segments.[3,21,22]

Serotonin may have an advanced role in the nerve root compression cycle.[23] Normally, serotonin has a vasodilative effect on healthy nerve roots. Chronically compressed nerve roots react with vasoconstriction in the presence of serotonin. In a study of 45 dogs, Sekiguchi and colleagues[23] inserted balloons deep to the lamina and kept them inflated for 1 week; their results suggested that endothelial cell dysfunction induced by serotonin might lead to contraction of blood vessels under chronic compression.

In a 2004 follow-up study with rats, Sekiguchi and colleagues[21] found that mild cauda equina compression induced tumor necrosis factor α (TNF-α) expression and demyelination, though increased compression induced TNF-α expression and degeneration associated with macrophage invasion. They also discovered that lesions proximal to the dorsal root ganglion may not produce significant allodynia.

Autoimmune Reaction/Wallerian Degeneration

Lee and Wolfe[24] postulated that nerve root compression disrupts the nerve–blood barrier. Proteins that now enter the central spinal nerves act as antigens and cause an autoimmune reaction. This mechanism may propagate the cycle of nerve Wallerian degeneration.

Sekiguchi and colleagues[21] found similar autoimmune reactions with demyelination and degeneration with increased nerve compression. Nerve roots showed demyelination after mild cauda equina compression. Axonal degeneration occurred with increased compression of the cauda equina. TNF-α–immunoreactive cells increased in any level of cauda equina compression. With increased compression, macrophages became evident.

Delamarter and colleagues[25] analyzed evoked potentials and the pathology of nerve compression. They discovered that chronic mild compression (25%) may not show signs of neurologic dysfunction and may show only mild changes in cortical evoked potentials and that chronic moderate compression (50%) may show signs of mild motor weakness with major changes in cortical evoked potentials; however, chronic severe constriction (75%) may show signs of significant weakness and urinary incontinence and signs of complete nerve root atrophy at the level of the constriction. They found that chronic severe constriction blocked the axoplasmic flow, leading to distal motor Wallerian degeneration and proximal sensory Wallerian degeneration.

History and Physical Examination

The clinical diagnosis of CES is made with thorough history-taking and physical examination; radiologic studies are used to confirm the diagnosis and to delineate the nature and location of the lesion. The aim of the initial patient interview is to establish the nature and chronicity of the symptoms, possible etiology, and excretory organ dysfunction.

Of primary importance in the common clinical scenario of back pain and radiculopathy is delineating the difference between acute disc herniation and CES, which occurs with 2% of all lumbar disc herniations. Although radicular symptoms may be present with CES, there will also be "saddle anesthesia" (sensory changes in the groin area) and vesicular or rectal dysfunction.[26-31]

CES may present acutely or chronically (in the latter case, symptoms take a more indolent course). In both cases, the most common symptoms are severe back pain and radiculopathy (83% and 90%, respectively).[26,32,33] This picture may be confusing, as 71% of patients have a prior history of back pain or sciatica. In acute CES, however, back pain increases severely and suddenly, and there are sensory changes in dermatomal distribution plus motor weakness and possible urinary retention resulting in incontinence and need for catheterization. Saddle anesthesia should immediately raise suspicion for CES.

Thorough history-taking may reveal the etiology of acute CES. Trauma history should be suspected, as up to 62% of patients report a recent episode of trauma.[27] The most common traumatic events include falls, motor vehicle accidents, weight-lifting, and chiropractic manipulations.[27,28,30,34] Risk factors for other etiologies (eg, previous spinal surgery, anticoagulation, fevers, chills) should be identified. Incidence of CES after lumbar spinal surgery

has been reported to be 0.1% to 0.2%.[35] In the case of postoperative hematoma, time of presentation of neurologic deficits can vary. The progression usually occurs over several hours but occasionally over several days.[36]

Patients with chronic CES typically present with recurring and insidiously increasing back pain and with gradual unilateral or bilateral sensory or motor loss. CES onset is heralded by bowel and bladder dysfunction that progresses gradually over several days to weeks.[27,28,32] Type of CES presentation (acute vs chronic) has not been shown to be clinically relevant in terms of patient outcome.[28,36]

Besides being relevant to the diagnosis, time course and type of bladder dysfunction help determine the prognosis of the final functional outcome. Time since symptom onset inversely correlates with chances of return of function.

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Furthermore, though incidence of urinary retention (60%) and incidence of incontinence (55%) are similar, patients have a significantly better chance of improvement in retention than in incontinence.[32] Bowel dysfunction may also be present, but the diagnosis of CES is more often based on urinary dysfunction, as urinary excretion typically occurs more often than bowel excretion.[29] The basis of urinary dysfunction becomes difficult to ascertain in the immediate postoperative period in a patient with a Foley catheter.[37,38] Characterization of clinical progression of a postoperative epidural hematoma has been described as onset of sharp peri-incisional pain to paresthesias, radicular pain, and bilateral neurologic deficits, which can sometimes be compounded by fevers in patients with abscesses.[39,40]

Onset of erectile dysfunction is an uncommon but prognostically poor symptom. It is present on initial presentation in less than 5% of patients with CES. Still, up to 30% of patients experience some form of erectile dysfunction on long-term follow-up, despite timely treatment.[32]

A thorough neurologic examination helps establish the spinal level of the lesion and differentiate between complete and incomplete loss of function. Most patients have an objective sensory deficit in the lower extremities and weakness less than or equal to 4/5, and 76% of patients also have decreased perianal sensation.[32] Reflexes may be decreased, with more than half of patients presenting with an absent ankle jerk reflex. Increased reflexes and long tract signs may be present with a lesion above the L1–L2 disc. Rectal evaluation may reveal decreased rectal tone, which further supports the diagnosis of CES.[27,41]

Radiographic Evaluation and Imaging Modalities

Plain radiograph is usually the first study obtained for a patient with complaints related to the spine, but the value of this imaging modality in evaluating patients with CES is limited. Spine radiographs are good in searching for evidence of spine trauma, listhesis, scoliosis, and disc degeneration but are poor in visualizing intervertebral disc herniations (the most common cause of CES) and spinal cord and root compression.

Myelography historically has been important in evaluating cord compression. However, use of this invasive procedure has been limited since the advent of more sensitive and specific studies, such as MRI and computed tomography (CT) with myelography. A positive finding on a plain myelogram consists of a partial or complete block of contrast in the spinal column (ie, hourglass constriction). A complete block may initially prevent imaging of the spinal column distal to the lesion; flexion and extension images after sitting in a flexed position for 1 minute may improve flow of contrast throughout the spinal column.[42,43]

MRI is the current study of choice in evaluating patients with suspected CES (Figures 2–5). This modality can be used to evaluate for both extrinsic causes of neural compression (eg, tumors, disc herniation, hematoma, infection) and intrinsic pathology.[43,44] When an infectious or neoplastic etiology is suspected, administration of intravenous contrast may provide more detail.[45,46] The disadvantage of MRI is that it is contraindicated in the presence of pacemakers, aneurysm clips, and metal fragments in the eyes or near vital structures. In addition, claustrophobic patients may not tolerate lying in a tube for a prolonged period. Such patients may require sedation, open MRI, or a different imaging modality.

CT with myelography can be used for good visualization of the spinal column. It is an invasive procedure with inherent risks, but it is the study of choice when MRI is contraindicated. CT myelogram provides better evaluation of the osseous structures than MRI does and better evaluation of the spinal cord than plain myelography does. Statistically, CT myelogram has higher false-positive and lower false-negative rates than MRI does.[44]

Bladder studies are occasionally useful in diagnosing CES, but their effectiveness as both diagnostic and prognostic tools has not been definitively established. The spinal cord micturition center is located at S2–S4 levels (anatomically near the L1 vertebrae). Injuries leading to bladder dysfunction may result in detrusor hyperreflexia and detrusor sphincter dyssynergia in suprasacral injuries and detrusor areflexia in sacral injuries.[47] Postvoid residual bladder volumes may be increased with detrusor areflexia; these may be measured either with a Foley catheter placement or with bladder ultrasound after urination. Although there is no absolute normal value, healthy volunteers have been noted to have

less than 30 mL postvoid residual bladder volume[48]; values of more than 100 mL should raise suspicion of urinary retention. Formal urodynamic testing is invasive and unnecessary for diagnosis of CES but may be useful in following bladder function after treatment.[49]

Classification

CES has been classified into incomplete CES (CESI) and complete CES (or CES with true retention; CESR).

In CESI, patients present with motor and sensory changes, including saddle anesthesia, but have yet to develop full retention or incontinence of either bowel or bladder.[29] Instead, the visceral changes they undergo are of neurogenic origin, such as straining micturition, possibly using abdominal compression to assist in voiding, loss of urgency, and alteration of urinary sensorium.

In CESR, patients have already developed true retention. Secondary to loss of the visceral neurologic signal to the central nervous system, painless urinary retention, and eventually overflow incontinence, is experienced.[29] Similarly, either retention or incontinence of the bowel may be experienced. Normally, urinary symptoms are recognized secondary to the usually frequent voiding and elimination events.[29] This distinction becomes more difficult in the postoperative period in a patient with a Foley catheter. Early recognition of cord compression and immediate decompression has been found to lead to a halt in the progress of neurologic deficits.[38,50]

Surgical Technique

The accepted surgical technique for CES treatment is wide laminectomy and extensive decompression with foraminotomies if needed for stenosis, as CES is often secondary to a large lumbar disc herniation.[26,28,51-53] Several authors also aggressively remove disc space material after decompression.[26,53] As for compressive hematomas or abscesses, thorough evacuation becomes the mainstay of treatment.[38,50]

Cases of laminotomy/hemilaminectomy with microdiscectomy have been reported,[39] but this technique is not recommended, out of concern that undue traction might be placed on the thecal sac and nerve roots during decompression, potentially worsening neurologic injury. There are no published reports of comparison studies of the outcomes of these techniques.

A few cases of transthecal (intradural) decompression have been reported.[54,55] Authors have claimed that this technique may help reduce traction injury to nerve roots, particularly when decompressing calcified central herniations.[51] Currently, most authors consider this technique unnecessary.[26,28,53]

Timing of Surgery

The most important issue regarding CES is timing. There are many articles about surgical outcomes and timeliness of surgery, and much confusion has arisen from the studies that have been reported.

Early investigations of CES outcomes tended to show little evidence for early decompression. Jennett,[56] who in 1956 was one of the first authors in the modern literature to report on CES, treated 25 patients but provided no evidence for early decompression.

Shephard[30] in 1959 studied a series of 13 patients with CES. Two had CESI; the other 11 had CESR of 5 days' to 2 years' duration. The author concluded that CES duration is less important than presence of either visceral or sensory involvement. He advocated for early decompression but provided no clear analysis of patient outcomes.

In 1979, Tay and Chacha[31] reviewed the cases of 8 CES patients, 7 of whom presented with urinary retention. Time to surgical decompression was 1 to 14 days for 7 patients; the eighth had an indolent course over 3 months. All patients obtained immediate pain relief and partial vesicular control at 14 days. Seven patients recovered full bladder control at 5 months, though postvoid residuals were not measured. Overall motor recovery was "good," but all patients had poor sensory and sexual function recovery. There was no discrimination between patients decompressed before the currently accepted 48-hour mark and patients decompressed after that mark.

In 1981, O'Laoire and colleagues[57] studied 29 patients and found no correlation between time from onset to decompression and level of recovery. Kostuik and colleagues[28] retrospectively reviewed the cases of 31 CES patients, 1 of whom had refused surgery. Patients were divided into 2 groups: (a) those who had acute-onset CES and underwent surgery within 48 hours of onset and (b) those who had insidious onset of symptoms and underwent surgery within 5 days of hospital admission secondary to uncertainty surrounding their diagnosis because of lack of urinary retention at presentation . Fifty percent of the acute-onset patients and 10% of the insidious-onset patients had residual bladder dysfunction. Although the authors implied that patients with acute-onset CES had a worse outcome, the result can be framed differently—that the outcome is worse for CESR patients (acute-onset group) than for CESI patients (insidious-onset group). There was no correlation between time from onset to decompression and recovery, though the authors recommended that CESI not be allowed to progress to CESR. At 2 years, 27% of the patients had residual sexual dysfunction, 10% had residual weakness, and 20% had residual sensory changes.

Shapiro[26] retrospectively reviewed 14 patients with CES on the basis of the 48-hour time frame set forth by Kostuik.[28] Of the 13 patients who presented with incontinence (implied CESR), 7 underwent surgery within 48 hours of presentation and subsequently regained bladder control and returned to unassisted ambulation, and an eighth had chronic sciatic pain. Of the 7 patients who underwent surgery after 48 hours, only 2 (of 6) regained continence (1 was never incontinent), 3 (of 7) had permanent weakness requiring an assistive device for ambulation, and 2 (of 7) had chronic sciatica.[26]

In an often referenced article on decompression timing, Shapiro[33] (2000) reported retrospectively reviewing 44 patients. Twenty of these patients underwent surgery within

48 hours of symptom onset: 17 patients within 12 hours, 1 within 12 to 24 hours, 2 within 24 to 48 hours. All patients who underwent surgery within 24 hours returned to full strength by 1 year; the 2 patients who underwent surgery within 24 to 48 hours regained 4/5 strength by 2 weeks. By 6 months, 95% of the patients who were decompressed within 48 hours had normal bladder function; 100% of the men resumed sexual activity, though there were subjective reports of decreased erection strength or sensation; and 6 of the 7 women resumed sexual activity, though all reported increased difficulty having an orgasm, and 1 could not achieve orgasm. Of the 24 patients who were decompressed more than 48 hours after symptom onset, 58% had 0/5 to 2/5 weakness, 63% continued to catheterize, 71% had chronic sciatic pain, and 31% of men were unable to achieve erection at 1 year.

In 2003, Hussain and colleagues[51] reviewed 20 CES patients, 6 of whom presented within 48 hours of symptom onset and underwent surgery within 5 hours. There was no significant difference in urologic outcome or overall quality of life between patients who were decompressed within 48 hours and patients who were decompressed after 48 hours.

Ahn and colleagues[27] conducted a large meta-analysis (322 patients) of surgical outcomes. All patients who refused surgery or who had CES with an etiology other than lumbar herniation were excluded. Patients were divided into 5 groups according to time from visceral symptom onset to decompression: less than 24 hours, 24 to 48 hours, 2 to 10 days, 11 days to 1 month, and more than 1 month. No discrimination was made between CESI and CESR. Significant associations were found for certain groups. Patients with preoperative rectal dysfunction or preoperative chronic low back pain were at 11-fold increased risk for postoperative urinary incontinence. Postoperative rectal dysfunction was worse in patients with preoperative chronic low back pain. Patients with postoperative rectal dysfunction also had a worse prognosis with respect to return of sensory functions. Overall outcome was defined by resolution of pain, sensory and motor deficits, and urinary, rectal, and sexual dysfunction. No difference in outcome was found among the 3 groups that underwent decompression more than 48 hours after symptom onset. The authors concluded that there was no statistical difference between patients who were decompressed within 24 hours and patients who were decompressed within 24 to 48 hours, and ultimately these 2 groups were combined.

Kohles and colleagues[52] critically reviewed the statistics in the meta-analysis by Ahn and colleagues[27] and, citing the small sample size, low statistical power, and flawed statistical methodology of that study, concluded that its findings ultimately "understat[ed] the value of early surgical decompression." According to Kohles and colleagues, the conclusion regarding no significant difference in outcomes between decompression groups (<24 hours vs 24 to 48 hours) cannot be asserted, and most likely the risk for poor outcomes increases continuously with increasing time.

That assessment was echoed by Lawton and colleagues[50] in a retrospective review of surgically treated spinal epidural hematomas. Outcomes were inversely related both to time from symptom onset to surgery and to duration of maximum deficit. Outcomes also correlated with severity of preoperative neurologic deficits. Immediate surgical evacuation was recommended. Complete recovery was experienced by 43% of patients and functional recovery by 87%.

In another meta-analysis, Todd[58] specifically examined 1 variable (time from symptom onset to surgery) and 1 outcome parameter (resumption of socially normal bladder function). The study was structured in this fashion to avoid the confusion (generated by earlier reports) as to what exactly constitutes a "good" outcome. After the meta-analysis and then a reanalysis, Todd[58] concluded that urologic outcome varies according to time from symptom onset to decompression and that there is stronger evidence for a 24-hour rather than 48-hour window of opportunity for improved outcome.

Other outcome studies have been conducted. In 2007, McCarthy and colleagues[32] reviewed a cohort of 56 patients who presented with CES. Of the 26 patients who underwent surgery within 48 hours of presentation, 5 were decompressed within 24 hours of symptom onset. Although there was a trend toward improved postoperative urinary and bowel disturbance in cases decompressed within 24 hours, it was not statistically significant.

Criticism of this time-dependent outcome comes on several points. The first, already mentioned, is the confusion (generated in the literature) regarding how CES is defined, plus the lack of time-dependent analyses of outcomes in early studies.[30,53,56] Further, few meta-analyses have discriminated between CESI and CESR—a point made by Gleave and Macfarlane[29] in their review. The retrospective nature of previous analyses is also a potential weakness, though one that must be accepted, for a prospective randomized study of early versus delayed surgery would not be ethical given the already established trend of time-dependent outcomes. Finally, bringing a patient emergently to the operating room involves practicalities (eg, availability of qualified staff). Emergent operations expose patients to additional risk that may not be present in a more controlled situation, particularly in the setting of CESR.[29,33,59]

Three types of delay in decompression were described by Shapiro.[33] First, there may be a delay on the part of the general or primary care practitioner in diagnosing CES or in seeking specialist consultation. Second, there may be a delay in obtaining a diagnostic study, mainly an MRI. Third, the surgeon may delay decompression for a more convenient setting—for example, a nonemergent setting in which more qualified staff are available.

Conclusions

At the time this review was written, it was agreed that CESI cases, or indeterminate cases (eg, postoperative cases), should be surgically decompressed emergently, as the neurologic and urologic outcomes are clearly improved provided the patient does not progress to CESR. There is no clear consensus regarding the urgency of decompression for patients with CESR. However, most authors, including the writers of this review, advocate early decompression,

preferably within 24 or 48 hours, for all patients with CES, provided the patient's medical condition and the presence of qualified support staff allow for it. We encourage other investigators to compare both CESI and CESR patient outcomes with respect to time-dependent decompression.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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