Uncovering Cauda Equina Syndrome: An In-depth Analysis from a Cutting-Edge Medical Perspective

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Abstract: Cauda equina syndrome is a rare condition caused by compression of the cauda equina nerve roots. Although its definition remains somewhat inconsistent, typical clinical manifestations include varying degrees of sensory loss, motor weakness, and bowel and bladder dysfunction. There is currently no definitive diagnostic method; clinicians primarily rely on thorough medical history collection and physical examination, complemented by advanced imaging techniques such as MRI and CT, to determine the location and extent of nerve root compression. Suspected cases necessitate urgent spinal surgical intervention and decompression. Even with emergency surgery, the prognosis remains less than ideal. Given the complexity of this syndrome and its potential legal ramifications, clinicians must acquire a deep understanding of its clinical characteristics, diagnostic approaches, and treatment strategies. Through effective communication, standardized medical practices, comprehensive medical record-keeping, and efficient care, patient long-term outcomes can be optimized and the risk of litigation due to medical malpractice minimized.

Keywords: Cauda equina syndrome; clinical presentation; risk factors; prognosis; review

1. Introduction

Cauda Equina Syndrome (CES) is a highly disabling spinal disorder caused by compression of lumbosacral spinal canal nerve disease, which greatly affects the quality of life of patients [1, 2]. Recent studies have investigated that the annual incidence of CES is 2.7/100,000 [3], while it can be up to 1 in 10,000 in individual countries [4]. Anatomically, the cauda equina is a peripheral nerve root that branches from the terminal sheath capsule of the spinal cord and usually begins at the level of lumbar 1 vertebrae [5]. Because of the lack of Schwann cell coverage of the distal nerve roots and the inadequate vascular distribution in the proximal third of the lumbar 1 vertebrae, this makes the cauda equina highly susceptible to compression and tensile stress [6].CES is a relatively difficult clinical problem for surgeons because of the complexity of the etiology and clinical presentation, and relatively poor prognostic outcomes.

2. Definition

In 1934, Mixter and Barr [7] first provided a clear definition of Cauda Equina Syndrome (CES), describing it as a clinical manifestation in which patients exhibit symptoms such as lower limb numbness, pain, loss of motor function, and bladder/rectal sphincter dysfunction in the context of disc herniation. Since then, there has been considerable debate regarding the clinical definition of CES. A recent review of 212 articles concerning CES definitions and clinical manifestations found up to 17 different definitions [8]. Among the various clinical definitions of CES, bowel dysfunction and bladder dysfunction are typically considered primary diagnostic criteria, with the latter being deemed essential for a definitive diagnosis by most physicians. However, it is notable that, despite sexual dysfunction being a common issue among CES patients, it is rarely mentioned in these definitions [9, 10], even though many patients express a desire for more information regarding sexual function prognosis [11]. Furthermore, CES can be categorized into acute and chronic forms based on the onset and duration of the disease. Acute CES is generally caused by a sudden, massive protrusion of the nucleus pulposus due to external forces, resulting in rapid onset and severe symptoms, with the vast majority of patients

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opting for emergency treatment. In contrast, chronic CES refers to persistent damage to the cauda equina within the spinal canal, manifesting as lower limb and pelvic pain, as well as symptoms related to the gastrointestinal, urinary, and chronic nervous systems [12].

The definition of CES is complex and interwoven, with overlapping criteria from various sources, leading to significant uncertainty in identification. In a recent study on CES definitions, despite providing evaluators with all available literature on CES definitions, the internal consistency for identifying CES categories among nine evaluators was only 0.34 [13], indicating a low level of agreement. This result suggests that in the real world, variations in diagnostic outcomes may arise due to differing information sources referenced by clinicians [13]. Consequently, clinicians should not rely solely on categorical definitions when making treatment decisions for CES patients, but should instead undertake individualized assessment and management to ensure the best possible treatment outcomes for patients.

3. Cause of disease

There are various etiologies of CES, and lumbar disc herniation is the most common etiology, accounting for 45% of all etiologies [14]. In addition to this, other etiologies such as trauma, infection, tumor, immune, hematogenous, idiopathic and medical etiologies should not be ignored. It is of concern that in patients with pre-existing lumbar stenosis, the lumbosacral spinal canal space has been reduced at baseline, and this condition predisposes them to CES, even if the degree of mechanical compression is relatively mild [5]. However, as the medical history of these patients is usually long, nerve production tolerance leads to the possibility that patients may ignore mild symptoms, thereby delaying medical attention and often providing unsatisfactory treatment outcomes [15].

4. Clinical manifestation

The clinical manifestations of CES are quite complex, with the most common symptoms including diminution of sensation in the lower extremities, motor dysfunction, abolition of lower limb reflexes, hypesthesia in the perineal region, urinary disturbance, fecal disturbance, and sexual dysfunction [16]. Among them, the characteristic symptoms such as disorders of bladder, bowel, sexual function, and hypesthesia in the perineal region are crucial for distinguishing CES from other spinal conditions with overlapping symptoms [17].

Patients with bladder dysfunction may experience some or all of the following symptoms: diminished sensation of bladder fullness, reduced urge to urinate, inability to interrupt midstream urination, or progressively worsening urinary weakness, thinning of the urine stream, increased residual urine volume, and severe urinary retention [18]. Additionally, in patients with loss of bladder tone, urination may sometimes be facilitated by contracting abdominal muscles or applying pressure to the lower abdomen [19]. Patients with impaired bowel function often present with reduced sensation around the anus and weakened anal sphincter contractions, and may even experience perianal burning or pain [18]. Constipation is often the initial symptom of bowel dysfunction, followed by potential issues such as fecal incontinence and gas incontinence [20, 21]. Sexual function is frequently overlooked in the diagnosis of CES, primarily because both clinicians and patients are reluctant to discuss this symptom [14, 22]. Some CES patients may exhibit sexual dysfunction while maintaining normal urinary function, which might be another reason for the neglect of sexual issues [23]. McCarthy et al. [24] found that male erectile dysfunction may go unnoticed preoperatively, especially during emergency surgery for acute CES. The study also noted that female sexual dysfunction might manifest as reduced sensation, difficulty with intercourse, or an inability to achieve orgasm, which may not be readily apparent during sexual activity [24].

Just as with the definitions of acute and chronic conditions, the clinical manifestations of CES also present as either acute or insidious. Acute manifestations are typically characterized by the sudden onset of severe lower back pain, sciatica, urinary retention, and perineal numbness, often caused by lumbar disc herniation, and are more commonly observed in younger individuals [25]. In contrast, insidious manifestations usually present with recurrent episodes of less severe back pain, predominantly affecting the elderly, and symptoms may persist for weeks or even years [26]. Additionally, insidious manifestations may gradually evolve to include sciatica, sensory and motor deficits, as well as bowel and bladder dysfunction. These symptoms are more prevalent in cases of degenerative lumbar stenosis compared to acute presentations and are associated with a poorer

prognosis [27].

5. Diagnosis and assessment

CES often presents with overlapping or similar clinical manifestations as conus medullaris syndrome or other lumbosacral disorders [28]. For such patients, clinicians should conduct a thorough history and physical examination, as well as additional diagnostic tests. Initially, clinicians should inquire about changes in perineal sensation during activities such as sitting, defecation, or personal hygiene (e.g., wiping with toilet paper) [29]. Additionally, further examination should include light touch or pinprick tests to assess any reduction in sensation. Moreover, it is crucial to determine whether changes in perineal sensation are unilateral, localized, or diffuse, as this impacts the patient's prognostic evaluation [30]. Although digital rectal examination can detect decreased anal tone, it is generally not recommended for diagnosing CES due to its lower sensitivity and potential for patient discomfort [31].

Magnetic Resonance Imaging (MRI) is the preferred imaging modality for diagnosing CES due to its exceptional capability in visualizing soft tissues and the cauda equina [32, 33]. However, the sensitivity of MRI is less than optimal, definitively diagnosing CES in only approximately 20% of suspected cases [34]. Bladder motor function can be directly assessed through invasive urodynamic studies [35], although the results of such tests sometimes do not correspond well with the patient's actual urinary symptoms [36]. Additionally, indirect assessment can be performed through palpation of an distended bladder or by measuring post-void residual bladder volume via ultrasound [37]. Among these methods, a larger post-void residual (PVR) volume indicates higher sensitivity [38], but a PVR \leq 200 does not exclude the possibility of CES [39]. Therefore, a combination of various diagnostic approaches is necessary to ensure the accuracy of CES diagnosis.

In addition to imaging studies and specialized physical examinations, the core outcome set assessment for CES places significant emphasis on questionnaire scales. Hazelwood et al. [41], in their postoperative follow-up of CES patients, skillfully employed a range of scale tools, including the Urological Symptom Score (USP), the Neurogenic Bowel Dysfunction Scale (NBD), and the Arizona Sexual Experience Scale (ASEX), thereby providing a comprehensive and precise evaluation of the patients' bladder, bowel, and sexual functions. Furthermore, aspects of bodily pain and function were assessed using the Visual Analog Scale (VAS) and the Oswestry Disability Index (ODI) [42]; for physical health, the SF-36 Health Survey (SF-36) and the EuroQol-5 Dimension (EQ-5D) [1, 24]; and for psychological assessment, the M-Z Depression Scale and the Mental Component Summary (MCS) [24, 43].

6. Treatment

When confronted with a suspected case of CES, a timely consultation with a spinal surgeon is crucial for accurately diagnosing the etiology of CES. If CES is determined to be caused by nerve compression, urgent surgical intervention must be undertaken without delay [44]. Historically, open laminectomy and discectomy were the preferred surgical procedures for decompression in CES [45]. However, with the continuous advancements in medical technology, minimally invasive techniques such as fenestrated discectomy and percutaneous endoscopic discectomy have significantly reduced operative time and blood loss, while effectively minimizing paravertebral tissue damage and postoperative pain syndromes, thereby providing patients with earlier opportunities for mobility and recovery [46-50]. Notably, recent developments in spinal cord stimulation, through electrical stimulation of the spinal dorsal column or posterior nerve roots, have shown remarkable improvements in motor deficits, sensory impairments, and urinary incontinence in CES patients, offering new hope for comprehensive recovery [51].

The medical community has not yet formed a uniform guideline on the precise timing of surgical decompression for CES. Since 2000, when Ahn et al. [52] first proposed the "golden rule" of decompression within 48 hours of presentation, this idea has triggered extensive discussion and controversy. Given the urgency of the onset of CES and its poor prognosis, it is ethically difficult to design randomized controlled trials in patients to investigate the optimal timing of decompression surgery. To address this dilemma, Delamarter et al [53] skillfully designed a dog-CES experimental model to explore the relationship between the timing of surgery and the degree of neural recovery. They found that all dogs recovered the same motor and bladder function after 6 weeks, whether decompression was performed immediately or at intervals of 1, 6, 24 hours or even 1 week. However, it

is noteworthy that the early decompression group showed a significant advantage in the speed of recovery, being able to return to normal faster [53]. This finding provides strong support for early decompression and suggests that the onset of CES is not a path of no return and that recovery of urinary function is still possible even after delayed decompression. Although longer delays lead to slower recovery, they do not affect the final degree of recovery. Considering the positive correlation between the time to decompression and the time to full recovery, early surgery is still recommended when conditions permit [42].

7. Postoperative recovery and influencing factors

The long and complex process of repairing cauda equina injury [1, 54] predicts that long-term follow-up after CES is often unsatisfactory. In a 13-year postoperative follow-up study, residual rates of symptoms in CES patients were reported: urinary dysfunction 38%, defectaion dysfunction 43% and sexual dysfunction 54% [11]. It is worth noting that the true prevalence of sexual dysfunction may also be higher than the currently reported prevalence. Patients may be reluctant to voluntarily report sexual symptoms due to the discomfort they may feel when asked questions related to sexual functioning [29, 55]. In addition, the age of the patient may also bias the true picture of sexual function [52].

The investigation into the prognostic risk factors of CES has been a focal point in the medical field. Kennedy et al. [30] confirmed that preoperative bladder dysfunction is a significant risk factor for poor postoperative recovery in CES, noting a pronounced correlation between the severity of preoperative bladder impairment and overall residual functional deficits. Butenschoen et al. [56] further analyzed 50 cases of acute CES with high body mass index (BMI) and discovered that patients with elevated BMI exhibited more severe preoperative CES symptoms; however, there was no difference in postoperative recovery outcomes. Additionally, by further subdividing the perineal numbness area, Kennedy et al. [30] identified that complete perineal numbness is a risk factor for poor postoperative prognosis in CES. Recently, Wang et al. [57] achieved a significant breakthrough in the prognosis research of CES, identifying six variables—stress urinary incontinence, overactive bladder, low flow, difficult evacuation, fecal incontinence, and perineal numbness—and developed the first predictive model for postoperative recovery in CES patients. Their Nomogram model elucidated that patients with a total score exceeding 148.02 are more likely to experience poor postoperative recovery in CES, aiding in the early identification of patients who may face long-term recovery challenges and allowing for more precise and effective treatment strategies [57].

8. Conclusion

The diagnosis of cauda equina syndrome is often made after irreversible damage to the cauda equina, and the prognosis is often unsatisfactory. Physicians need to be familiar with the signs and symptoms of CES and the potential impact on prognosis to avoid delays in clinical practice. Early surgical decompression can minimize further damage to the compressed nerve and shorten the patient's recovery time. If evidence of nerve compression exists on imaging, surgical decompression remains mandatory regardless of how late it is. In addition, both preoperatively and postoperatively, physicians should give adequate attention to the patient's sexual function, as recovery of sexual function is closely related to the patient's overall quality of life and psychological well-being.

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