

CONUS MEDULLARIS SYNDROME-A REVIEW

¹Kausalyah Krisna Malay, ²Saravanan, ³Revathy Duraisawamy, *⁴Dhanraj Ganapathy

ABSTRACT

The spinal cord tapers and ends at the level between the first and second lumbar vertebrae in an average adult. The most distal bulbous part of the spinal cord is called the conus medullaris, and its tapering end continues as the filum terminale. Distal to this end of the spinal cord is a collection of nerve roots, which are horsetail-like in appearance and hence called the cauda equine. These nerve roots constitute the anatomic connection between the central nervous system (CNS) and the peripheral nervous system (PNS). They are arranged anatomically according to the spinal segments from which they originated and are within the cerebrospinal fluid (CSF) in the subarachnoid space with the dural sac ending at the level of second sacral vertebra. Conus medullaris syndrome refers to a characteristic pattern of neuromuscular and urogenital symptoms resulting from the simultaneous compression of multiple lumbosacral nerve roots below the level of the conus medullaris. These symptoms include low back pain, sciatica (unilateral or, usually, bilateral), saddle sensory disturbances, bladder and bowel dysfunction, and variable lower extremity motor and sensory loss .

KEYWORDS: *cauda equine; conus medullaris; distal bulbous spinal cord tapers; low back pain; saddle sensory*

I. INTRODUCTION

During development, the vertebral columns grow more rapidly than the spinal cord. Spinal nerves exit the vertebral columns at more progressive oblique angles because of the increased distance between the spinal cord segments and the corresponding vertebrae. Lumbar and sacral nerves travel almost vertically down the spinal canal to reach their foramen to make an exit.[1]The spinal cord ends at the intervertebral disc in between the first and second lumbar vertebrae is known to be a tapered structure which is called as conus medullaris. It consists of sacral spinal cord segments. The filum terminale is the fibrous extension of the cord. It is a non-neural element that extends down to the coccyx.

¹Graduate Student, Department of Prosthodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Chennai, India.

²Assistant professor, Department of Anatomy, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Chennai, India.

³Senior Lecturer, Department of Prosthodontics, Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Chennai, India.

⁴Professor and Head Department of Prosthodontics, Saveetha Dental College and Hospitals, Saveetha Institute of Medical And Technical Sciences, Chennai – 600077 Tamil Nadu, India.

The cauda equine is a bundle of intradural nerve roots at the end of the spinal cord. These cauda equine is seen in the subarachnoid space distal to the conus medullaris. Sensory innervations provide cauda equine to the saddle area, sphincters from motor innervations, and the bladder and lower bowel from parasympathetic innervations. However, the nerve root in the cauda equina region carries sensation from the lower extremities, perineal dermatomes, and outgoing motor fibers to the lower extremity myotomes.

Primary blood supply to the conus medullaris is obtained from three major spinal artery which are the anterior median longitudinal arterial trunk and two posterolateral trunks.[2] Least prominent source of blood supply include radicular arteries the branch of aorta, lateral sacral arteries, and the fifth lumbar, ilio-lumbar, and middle sacral arteries. Even though the vascular supply of cauda equine is not in a segmental fashion, but it contributes latter. It is not like the blood supply to the peripheral nerve. The nerve roots also supplies from diffusion of surrounding CSF. Moreover, zone of relative hypovascularity will be seen in the proximal area of the nerve roots.[3]

II. PATHOLOGY

Any lesion that compresses cauda equina nerve roots leads to a syndrome called conus medullaris . Compressed nerve roots are particularly susceptible to injury, because they have a poor developing epineurium. A well-developing epineurium such as peripheral nerves have the ability to protect against compressive and tensile stresses.[4] The micro vascular systems of nerve roots have a region of relative hypovascularity in their proximal third.

Increase in vascular permeability followed by subsequent diffusion from the surrounding cerebral spinal fluid supplement the nutritional supply. This increased permeability property is associated with tendency toward edema formation of the nerve roots, which results in edema compounding initial and also might seem to be slightly injured. Many studies show that the magnitude is not the only reason but also the length and the speed of obstruction were also have an important role in damaging the cauda equina region.[5] Blood flow reduces in the intermediate nerve segment when pressure is applied along the path of the nerve in cauda equina.[6]

III. CAUSES

Any narrowing of the spinal canal that compresses the nerve roots below the level of the spinal cord causes conus medullaris syndrome.[7] There are numerous reasons for this conus medullaris syndrome have been reported, including disc herniation, traumatic injury intradural disc rupture, spinal stenosis secondary to other spinal conditions, primary tumours like ependymomas and schwannomas, metastatic tumours, arteriovenous malformation or haemorrhage, infectious conditions and iatrogenic injury.[8, 9]

Lumbar stenosis and spinal trauma including fractures are the most common causes of conus medullaris syndromes.[10] On the other hand, the herniated nucleus pulposus has caused 2-6% of cases of conus medullaris syndrome[11, 12, 13] Moreover, neoplasm such as metastases, astrocytoma, neurofibroma, and meningioma affects 20% of all spinal tumours this region. Some other rare conditions are also leads to conus

medullaris syndrome, for example, inferior vena cava thrombosis spinal arteriovenous malformations, advanced-stage of ankylosing spondylitis, neurosarcoidosis and multiple sclerosis[14]

IV. INFLAMMATORY AND INFECTIOUS CONDITIONS

Long-lasting inflammation of the spine, including Paget disease and ankylosing spondylitis, may leads to conus medullaris syndrome secondary to spinal stenosis or fracture. Infectious conditions, including epidural abscess, may lead to deformity of the nerve roots and spinal cord.[15] The general symptoms are severe back pain and a rapid progression in motor weakness. Infectious condition of conus medullaris syndrome can be pyogenic or nonpyogenic.

Generally, pyogenic abscesses are found in an immunocompromised or host with poor nourishment. *Staphylococcus aureus* causes epidural abscesses which increases the incidence of infections with methicillin-resistant *s.aureus*, *Pseudomonas* species, and *Escherichia coli* also been recorded [16]. Nonpyogenic condition causing an abscess is rare, including tuberculosis. Resurgence of tuberculosis is secondary for immunocompromised individuals with HIV infection. They requires a high index of suspicion, as the development of conus medullaris syndrome can be followed by an indolent course.[17] Conus medullaris syndromes are classified as clinical syndromes of the spinal cord.

Conus medullaris syndrome is rare condition, and it can be traumatically as well as traumatically. Although infrequently, it is a diagnosis that must be considered important as patients chief complaint is lower back pain associated with neurologic complaints, especially urinary symptoms.[18]

V. TREATMENT

So far, there is no proven medical treatment exists. The therapy is available which is directed towards treating the underlying cause of this syndrome. For penetrating trauma, steroids have not shown any significant benefit. Surgical procedure is still controversial. The timing of decompression is controversial, with immediate, early, and late surgical decompression shows vary in results.[19, 20, 21] For mechanical compression of the cauda due to disk herniation, surgical intervention may be indicated. In acute conditions it is mandatory to do surgical decompression as soon as possible to relieve the nerves from pressure and increasing the space of the spinal canal. A practice guidelines have been introduced for the management of low back pain.[22, 23] Admit patients to the appropriate services usually under neurology, neurosurgery, or orthopedic surgery with frequent neurologic checks.

Ethically, the physician should start examine the patient at the time of admission [24,25]. Patients in whom has acute cauda equina syndrome should be considered to not be treated or investigated on an outpatient basis without evaluation by a consultant and appropriate imaging.[26, 27] For deep venous thrombosis/pulmonary embolism, patients should be given with antiembolic compression stockings and subcutaneous heparin for 3 months as prophylaxis.[28] Low molecular weight heparin is also an approved prescription for prophylaxis. Ultrasound should be taken as the initial screening test in the lower extremities region. Patients with herniated disk recommended treatment is the laminectomy, followed by discectomy and

gentle retraction for decompression of the canal. Patient education, Biofeedback, and relaxation therapies may also be used.[29]

REFERENCE

1. **Mauffrey C, Randhawa K, Lewis C, Brewster M, Dabke H.** Cauda equina syndrome: an anatomically driven review. *Br J Hosp Med (Lond)*. Jun 2008
2. **Olmarker K, Rydevik B, Hansson T, Holm S.** Compression-induced changes of the nutritional supply to the porcine cauda equina. *J Spinal Disord*. Mar 1990
3. **Delamarter RB, Sherman JE, Carr JB.** 1991 Volvo Award in experimental studies. Cauda equina syndrome: neurologic recovery following immediate, early, or late decompression. *Spine (Phila Pa 1976)*. Sep 1991
4. **Olmarker K, Rydevik B, Holm S.** Edema formation in spinal nerve roots induced by experimental, graded compression. An experimental study on the pig cauda equina with special reference to differences in effects between rapid and slow onset of compression. *Spine (Phila Pa 1976)*. Jun 1989
5. **Olmarker K, Rydevik B, Holm S, Bagge U.** Effects of experimental graded compression on blood flow in spinal nerve roots. A vital microscopic study on the porcine cauda equina. *J Orthop Res*. 1989
6. **Olmarker K, Holm S, Rydevik B.** Importance of compression onset rate for the degree of impairment of impulse propagation in experimental compression injury of the porcine cauda equina. *Spine (Phila Pa 1976)*. May 1990
7. **Olmarker K, Holm S, Rosenqvist AL, Rydevik B.** Experimental nerve root compression. A model of acute, graded compression of the porcine cauda equina and an analysis of neural and vascular anatomy. *Spine (Phila Pa 1976)*. Jan 1991
8. **Metser U, Lerman H, Blank A, Lievshitz G, Bokstein F, Even-Sapir E.** Malignant involvement of the spine: assessment by 18F-FDG PET/CT. *J Nucl Med*. Feb 2004Takahashi K, Olmarker K, Holm S, Porter RW, Rydevik B. Double-level cauda equina compression: an experimental study with continuous monitoring of intraneural blood flow in the porcine cauda equina. *J Orthop Res*. Jan 1993
9. **Rydevik BL, Pedowitz RA, Hargens AR, Swenson MR, Myers RR, Garfin SR.** Effects of acute, graded compression on spinal nerve root function and structure. An experimental study of the pig cauda equina. *Spine (Phila Pa 1976)*. May 1991
10. **Rydevik B.** Neurophysiology of cauda equina compression. *Acta Orthop Scand Suppl*. 1993
11. **Pedowitz RA, Garfin SR, Massie JB, Hargens AR, Swenson MR, Myers RR, et al.** Effects of magnitude and duration of compression on spinal nerve root conduction. *Spine (Phila Pa 1976)*. Feb 1992
12. **Todd NV.** An algorithm for suspected cauda equina syndrome. *Ann R Coll Surg Engl*. May 2009;91(4):358-9; author reply 359-60
13. **Olivero WC, Wang H, Hanigan WC, Henderson JP, Tracy PT, Elwood PW, et al.** Cauda equina syndrome (CES) from lumbar disc herniations. *J Spinal Disord Tech*. May 2009
14. **Kingwell SP, Curt A, Dvorak MF.** Factors affecting neurological outcome in traumatic conus medullaris and cauda equina injuries. *Neurosurgical Focus*. 2008

15. **Fujisawa H, Igarashi S, Koyama T.** Acute cauda equina syndrome secondary to lumbar disc herniation mimicking pure conus medullaris syndrome--case report. *Neurol Med Chir (Tokyo)*. Jul 1998
16. **Raj D, Coleman N.** Cauda equina syndrome secondary to lumbar disc herniation. *Acta Orthop Belg*. Aug 2008
17. **Kothbauer K, Seiler RW.** [Tethered spinal cord syndrome in adults]. *Nervenarzt*. Apr 1997
18. **Rooney A, Statham PF, Stone J.** Cauda equina syndrome with normal MR imaging. *J Neurol*. May 2009
19. **Harrop JS, Hunt GE Jr, Vaccaro AR.** Conus medullaris and cauda equina syndrome as a result of traumatic injuries: management principles. *Neurosurg Focus*. Jun 15 2004
20. **Fisher RG.** Sacral fracture with compression of cauda equina: surgical treatment. *J Trauma*. Dec 1988
21. **Schizas C, Ballesteros C, Roy P.** Cauda equina compression after trauma: an unusual presentation of spinal epidural lipoma. *Spine (Phila Pa 1976)*. Apr 15 2003
22. **Thongtrangan I, Le H, Park J, Kim DH.** Cauda equina syndrome in patients with low lumbar fractures. *Neurosurg Focus*. Jun 15 2004
23. **Haldeman S, Rubinstein SM.** Cauda equina syndrome in patients undergoing manipulation of the lumbar spine. *Spine (Phila Pa 1976)*. Dec 1992
24. **Muthukumar T, Butt SH, Cassar-Pullicino VN, McCall IW.** Cauda equina syndrome presentation of sacral insufficiency fractures. *Skeletal Radiol*. Apr 2007
25. **Kebaish KM, Awad JN.** Spinal epidural hematoma causing acute cauda equina syndrome. *Neurosurg Focus*. Jun 15 2004
26. **Chen HJ, Liang CL, Lu K, Liliang PC, Tsai YD.** Cauda equina syndrome caused by delayed traumatic spinal subdural haematoma. *Injury*. Jul 2001
27. **Zuccarello M, Powers G, Tobler WD, Sawaya R, Hakim SZ.** Chronic posttraumatic lumbar intradural arachnoid cyst with cauda equina compression: case report. *Neurosurgery*. Apr 1987
28. **Raaf J.** Removal of protruded lumbar intervertebral discs. *J Neurosurg*. May 1970
29. **Kostuik JP, Harrington I, Alexander D, Rand W, Evans D.** Cauda equina syndrome and lumbar disc herniation. *J Bone Joint Surg Am*. Mar 1986