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Early spinal decompression after documentation in the initial CES-S and CES-R stages of Cauda Equina syndrome: saving both the patient and the clinician

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ABSTRACT

Background: Cauda equina syndrome (CES), described by Mixter Barr in 1934, is a rare and acute surgical emergency, and in our observation, the slower stages of bowel, bladder or limb dysfunction are more common than that claimed in the literature. This paper is to remind clinicians to document all the clinical changes of an evolving CES because patients could already be slipping into a progressive CES but will not reveal them unless inquired.
Methods: The IRT Programme for Medicos and Doctors (IRTP) of KRUSHI Orthopaedic Welfare Society, an NGO based in India, emphasises "preventive orthopedics", conducted this compilation study which is a mixed or ambispective study design of 650 patient data of which 450 patient data was from direct study in our KOWS research purview and 150 patients from various data bases like the Scopus, Web of Science, PubMed, JSTOR, and ScienceDirect.
Results: Incidence of early stages of CES may be missed if only bladder dysfunction is given importance while taking in the patient information by the clinician as only of 33% of the patients complain of bladder dysfunction whereas genital numbness is 47%, sexual dysfunction is of 53% of incidence when carefully enquired into.
Conclusions: The slower forms of cauda equina syndrome are usually missed by the clinicians if they trivialise the red flags of the autonomic dysfunction which are more frequent than that noticed and also the radiologist is equally responsible for not reporting enough about the redundant nerves (RND), the spinal canal diameters as smaller canals promote CES even with a smaller compression. If the clinician asks the right questions, the patients in these slower CES (S), CES(R) will never be missed. This article highlights the sigmoid curve pathophysiology and highlights the time frame and emphasis the early stages of CES(S) and CES (R) to be the best stages where the surgery is beneficial.

Keywords: Cauda equina syndrome, Documentation, Atypical presentation, International research trainee programme, Redundant nerves, Spinal canal stenosis

INTRODUCTION

Cauda Equina syndrome (CES) has devastating consequences. Hence, all doctors must document and decompress the cauda equina nerves in the early stages of CES-S and CES-R itself and not wait for the classical CES to develop, when it will become too late for a good result after decompression, which will not save the patient and leads to a disproportionately high litigation (22% litigation cases filed globally).[1] This research focuses on the red flags that should never be taken lightly.

Aim

The importance of teaching/refreshing medicos and clinicians about the benefits of early release of pressure on

the Cauda Equina nerves on time (prognosis is good) before a surgical emergency of a full-blown CES arises (prognosis is poor).

The aim is to highlight that the sacral nerves could also be compressed in isolation due to various reasons, causing CES, and to highlight the "grumbling" Cauda equina symptoms in the elderly, which have their challenges.

Objective

The objective of this publication is to create full awareness of the four stages of the CES and to save patients even before the next stage of CES was reached.

METHODS

This compilation study design was a mixed or ambispective study done by the Research Centre of KRUSHI Orthopaedic Welfare Society, an NGO based in Andhra Pradesh, India. This study was done between April 2005 – 2025 and the selection criteria was patients with soft and bony causes of lumbar spinal canal stenosis without prior intervention comprising case histories of 150 patient articles from various databases like PubMed and others as a retrograde study of 450 patients in real-time clinical presentations for the past 20 years in KOWS research centre. This mixed or ambispective study combined both retrospective and prospective data collected to analyse a cohort over time to leverage existing data so as to investigate past exposures and outcomes, and at the same time following the cohort into the future for ongoing data collection. This approach allows for the study of both past events and their ongoing impact.

Exclusion criteria

The patients with other similar presentations of low back ache with urinary tract frequency with infections, pelvic floor laxity, extreme obesity, prostrate hypertrophy, and diabetes were excluded in this research study.

The Helsinki guidelines were followed, and patient rights and privacy were protected and monitored by the Ethics Committee of the Krushi Orthopaedic Welfare Society IRT programme.

Statistical analysis included data collection, organisation, analysis, interpretation, and presentation, which were done by the IRT Programme members.

RESULTS

Autonomic dysfunction is more frequent, but sadly not enquired into enough sadly and also poorly documented, only as a passing enquiry. The clinician must politely explain the lumbar and sacral nerve innervations to the various organs correlating the CES deficits and then only the patient will be happy to reveal their private information from which they were too shy to speak out about their sexual, bladder, bowel functions and perianal pain, as they never correlated their complaints to low back ache and maybe that's why the chief author gets valuable information from his patients in this research study as he spends some time slowly explaining its significance.[2]

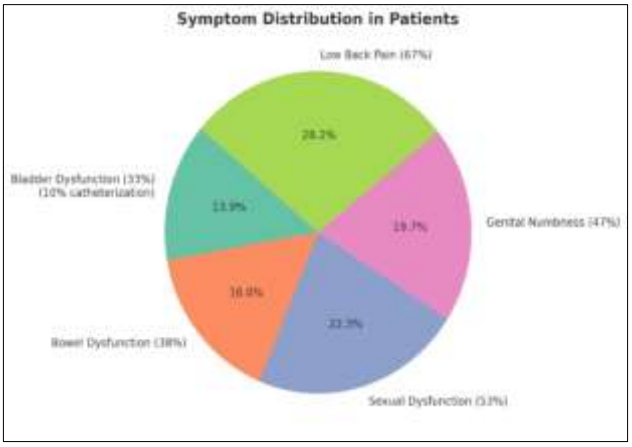

Figure 1: The composition of each complaint for better patient and doctor understanding.
Image credit: Krushi OWS research wing

From Figure 1, the following observations were made: bladder dysfunction (33%), 10% needed urinary catheterization, bowel dysfunction (38%), sexual dysfunction (53%), genital numbness (47%), and low back pain (67%).

Urinary and faecal incontinence with catheter use, sexual dysfunction, and genital numbness were more commonly seen in patients with CES (R).

Anatomical significance of the Cauda Equina versus Conus Medullaris

The Cauda equina nerves act like peripheral nerves comprising L1 to S5, coccygeal nerves containing the axons of the sensory and motor nerves supplying the legs, bladder, bowel and the perineum and the anus. So, anal pain must never be ignored.

The spinal cord is given protection from all around by the vertebral cage with its soft tissue protective reinforcements like the adipose tissue, dura mater, arachnoid mater, pia mater, CSF, and ligamentum flavum.

CES results from compression and disruption of the function of CE nerves. It can involve the conus medullaris or distal to it and most often occurs when damage occurs to the L3-L4 nerve roots.[3]

CES and CMS are neurosurgical emergencies as they can present with similar symptoms early in their course. CES is commonly due to an intervertebral disc.

DISCUSSION

Preventive orthopaedics

No patient must go into the CES (complete) stage just because of an overlook of the CES (S) and CES (R) stages, as there are four kinds of 'patient -doctor' scenarios.

The patient is ignorant of the link between lumbago and genital, bowel, and bladder dysfunction.

The patient is non-trusting and also afraid of surgery, usually goes through the CES (S), CES (R), CES (I), and CES (C) stages, wasting much time and finally develops CES either acutely but once CES (complete) occurs- no surgery can give good results, and this is a law suit opportunity if documentation is missed about each visit by the clinician.

Elderly with grumbling Cauda Equina symptoms

Clinician is casual and inattentive. The patient is good and eager for the proper and timely treatment, even surgical, but the clinician is not asking the right questions nor documenting them, and the examination is just a casual run-through of questions without proper clinical examination. This also is compounded by the way the hospital management pressurizes the doctors to complete the file within 4-6 minutes so that more patients can be seen in each hour they are paid.

The medical student often wonders how are the sacral nerves being compressed in a lumbar region, cause bowel, bladder, sexual, and perineal dysfunctions?

The sacral nerves are in the central groups, and the lumbar nerves are at the sides to exit into their respective intervertebral foramina at each level, explaining why the central massive disc herniations produce more incidence of CES.

Can the disc herniation disappear or reduce with time?

As per the literature the herniated disc reduces in size with time due to an absorptive process that involves neo-vascularisation, infiltration of the herniated disc by inflammatory cells like macrophages, granulocytes, and lymphocytes, and sequestrated discs show greater resorption.[3,4]

This is the time where conservative treatment can be given to the patient and is the important point that as long as there are no signs and symptoms of early stages of CES, i.e. CES (suspect) and CES (risk), conservative treatment can be chosen as there is evidence that there can be a spontaneous resolution of a disc herniation but other causes of CES should be kept in mind.

Other causes of pinching, leading to a slow and continuous pressure on the cauda equina nerves, which do not regress like the bony spinal canal stenosis, intraspinal conditions, and diseases, whereas a disc herniation can retract in some patients to a great extent over time.

Atypical CES may be seen due to the following factors.

Overlapped Conus Medullaris syndrome

CES with CMS

A case report was published by Mirza et al, in which CES was reported but accompanied by ankle clonus, which complicated the clinical picture.[5,6]

In this case report, the patient presented with lower motor neuron signs in the lower limbs but also exhibited clonus—a sign of upper motor neuron involvement. This finding led to a reconsideration of the diagnosis of CES and a revisit of the differential diagnosis to exclude other possibilities.

After ruling out UMN lesions and inflammatory and demyelinating lesions, an emergency Laminectomy and discectomy were performed at the massive disc herniation level at L5-S1 level. The patient recovered, but the clonus remained and slowly disappeared over several months. It was then inferred that the massive L5-S1 disc was the cause.

Intradural disc herniation

It is illustrated in Figure 2.[7]

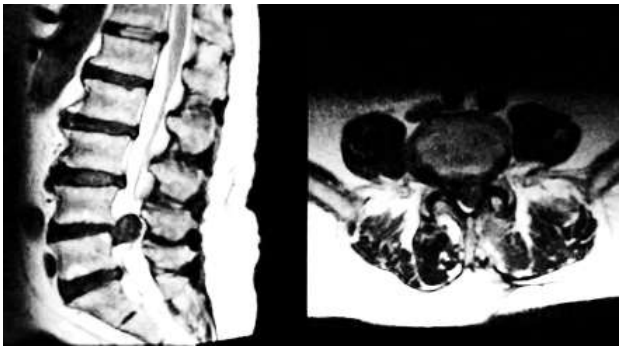

Figure 2: Intradural disc both on sagittal and axial sections.[8]

Hematoma/abscess

Here, the student must have a clear idea about how to infer after seeing an MRI scan sagittal image and learn to appreciate each layer of the spinal cord – epidural space, subdural space, subarachnoid space, and pia mater.[6]

Tension CSF leak

This type of CSF leak can occur after a direct or indirect injury or following a surgical procedure.[9]

Tumours

Diastematomyelia and other congenital abnormalities causing CES.[10-12]

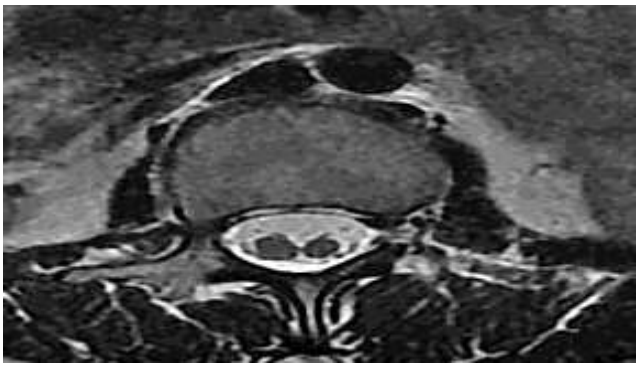

Figure 3: Cord syndrome or diastematomyelia causing cramming of the Cauda Equina nerves and increased risk for CES.
Image credit: research files of KRUSHI Orthopedic Welfare Society

MRI scan negative CES

The causes can be due to various reasons, such as root compression not visible on MRI and other non-compressive etiologies causing neurological dysfunction.[13]

Retracted intervertebral disc prosthesis is a reminder that the diagnosis of CES is rare in literature, but not so rare as seen by the red flags we see in our daily practice.

Figure 4: Retracted intervertebral prosthesis playing the exact role of an extruded disc on the anterior thecal wall.

Arterio venous malformations can become a compressive entity inside the spinal canal.[14]

Peri neural cysts or Tarlov cysts cause back pain depending on their location and compression effects on the cauda equina nerves. If the bone can get scalloped and thinned down to this extent…imagine what this pressure can do to the Cauda Equina nerves.[15]

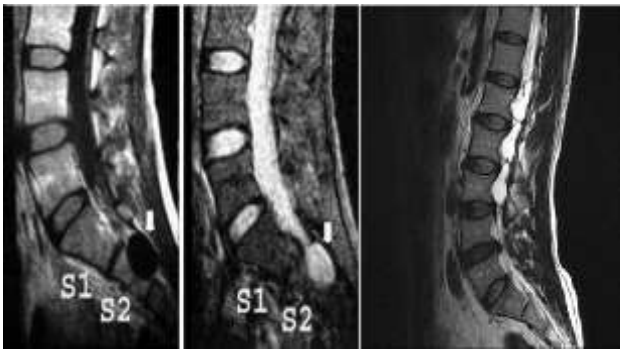

Figure 5 (a and b): Show the peri neural cysts or Tarlov cysts.
Image Credit: Research Files of Krushi Orthopedic Welfare Society, India

Large disco osteophyte complexes/ossification of the ligamentum flavum (yellow ligament) can be a constant source of compression on the spinal cord.[16]

Spinal nerve anomalies like duplication, abnormal mergers, and thickening.

Thrombosed aorta, and vena cava can also be included.[17]

A small disc herniation can initiate CES if the spinal canal diameter is small, caused by bony stenosis or soft tissue hypertrophy, facet hypertrophy, or vertebral instability.

Grumbling CES

The elderly patients develop degenerative verterbal desease and the spinal canal becomes steonotic and the symptoms have to be carefully assessed and dealt accordingly as this leads to a slow CES.[18]

Classifications

There are several classifications, but in this study, the classification used is that described by Lavy et al, where four stages of deterioration of CES were described - CES [S]- suspected: patients who do not have CES symptoms but may go on to develop CES; CES [I]- incomplete: patients who present with urinary difficulties of a neurogenic origin like loss of desire to void, poor stream, needing to strain to empty their bladder, and loss of urinary sensation. These patients could develop CESR and are a surgical emergency; CES [R] -retention: when there is a scenario of painless urinary retention and overflow incontinence; the bladder is no longer under executive control. An urgent surgical opinion and surgical decompression are necessary, and CES [C]- complete: patients who have objective symptoms like loss of cauda equina function, absent perineal sensation, a weak anal sphincter and paralysed bladder and bowel.[19]

All patients had been surgically decompressed by open discectomy.

Lumbar spinal canal size might play a part in the caudal compression in lumbar herniated disc patients.

The relation between MRI and clinical CES features has never been studied.

Continuous Neurological deterioration is more important than the duration of compression in deciding the prognosis.

Pathophysiology of Cauda Equina nerve compression

It is important to note that the normal CE nerves are damaged with pressure and altered to pathological RNR-CE nerves and finally to a damaged CE nerve.

We agree with the work by Fraser's group, who stated that although lumbago with leg signs are often seen, for a diagnosis of CES, there should be a dysfunction of one or more sacral nerves S2 and below, which present as bowel and bladder dysfunction, reduced sensation in the saddle area, sexual dysfunction- reduced or absent sensations, erectile dysfunction, and reduced orgasms.[20]

As chronic pressure rises, various presentations of urinary difficulties of neurogenic origin may appear as follows: altered urinary sensation, loss of desire to void, and poor stream with difficult voiding. CESR occurs when the bladder is no longer under executive control, and there is painless retention of urine with overflow.

It is the responsibility of every clinician to document the deficit, however minor it may seem, because, at the next meeting, there could be an observable deficit – a time for immediate spinal surgery.

An MRI scan is needed for confirmation of the nerve root compression but it is important to remember that there can be a patient landing in CES after a normal MRI scan.

Poor documentation of the stage of CES may result in the loss of opportunity to recover, as Pronin et al have pointed out that compressive pressure is a crucial aspect of CES pathophysiology.[21]

The unnoticed compressive pressure causes an increasing degree of ischaemia, which explains the pathophysiology of the four stages of CES and early decompression removes the mechanical and perhaps chemical factors that cause progressive neurological damage.

Notable exceptional patients present Also, not all CES patients experience low back pain and changes in sensory and motor function in the lower limbs.

Prognosis can be foretold by using the modelling series of electrophysiological studies. Modelling suggested a sigmoidal response, with particularly deterioration when the mean arterial blood pressure is exceeded and sustained for approximately 1 hour which is a proof of electrophysiological dysfunction in acute cauda equina compression seen as a sigmoid curve.

After decompression, the outcome depends on the degree of compression. An irreversible lesion results when the pressure exceeds the systolic pressure rather than the duration. Prognosis was most strongly linked to residual pre-decompression function.

As per modelling studies, it was revealed that the following factors contribute to poor prognosis in CES as follows.

Electrophysiological dysfunction is a non-linear sigmoidal curve.

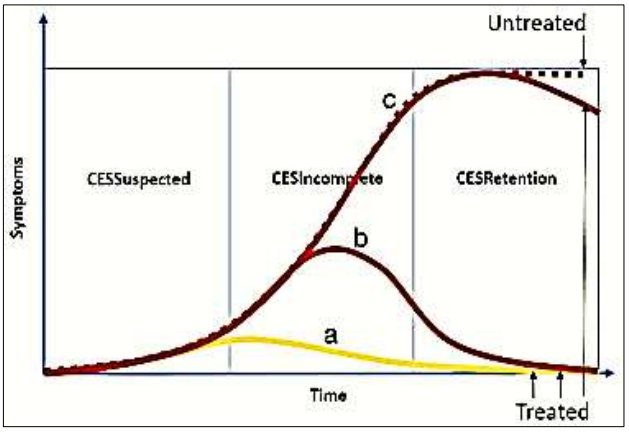

Figure 6: S-shaped nonlinear sigmoidal curve in which the neurological deterioration is well depicted to be slow and gradually increasing with increasing pressure, and after a certain threshold, this deterioration suddenly shoots up as the patient enters into the CES incomplete stage and if at least at this time an emergency spinal decompression is done a favourable result can be hoped for but once the stage of CES Complete is established there is irreversible damage to the innervated organs and the limbs, bowel bladder and the sexual organs will never recover. This speaks volumes about the importance of timing in early intervention.

A sudden increase in pressure beyond the mean arterial pressure is the critical point that sets in permanent damage.

If this high-pressure state exists beyond 1 hour, the prognosis is the poorest as the neurological damage is permanent, and spinal decompression at this stage is futile. It may lead to adverse medicolegal consequences, as there is an incidence of 22% MLC being filed against the surgeon in CES patient care.

Modelling decompression studies revealed the following: with each minute delay to decompression, recovery of function is reduced by 0.21%, each additional mm Hg of compression was forecasted to reduce function by 0.53% of normal performance, however, the electrophysiological

dysfunction is unlikely to progress at lower compressive pressures, even for longer durations, smaller diameters of the spinal canal due to various etiologies can precipitate a CES due to even a small disc herniation, noticeable is an hourglass type of cord compression by the disc herniation from the front and by the hypertrophied ligamentum flavum from behind causing a scenario of severe throttling effect on the spinal nerves.

CES occurs when there is a stenosed spinal canal due to bony causes, soft tissue caused by severe facet hypertrophy, a spondylolisthesis, an extradural, intradural, ischemic tumour, hematoma, Tarlov cyst or perineural cyst.

Chatha et al mentioned cut-off values for the the average sagittal diameters above which CES can develop as follows: at L1, the spinal canal diameter should not be less than 14.1 mm A-P, at the L1-L2 disc space, A-P spinal canal diameter should be less than 15.6 mm, at L2 should not be less than 13.2 mm A-P spinal canal diameter, at the L2-L3 disc space, the A-P spinal canal diameter should not be less than 15.1 mm, at L3, the spinal canal diameter should not be less than 12.6 mm A-P, at the L3-L4 disc space, the A-P spinal canal diameter should not be less than 13.8 mm, at L4, should not be less than 12.4 mm A-P spinal canal diameter, at the L4-L5 disc space A-P, the spinal canal diameter should not be less than 12.9 mm, at L5, it should not be less than 12.4 mm A-P spinal canal diameter, and at the L5-S1 disc space, the A-P spinal canal diameter should not be less than 11.6 mm.

The spinal canal A-P diameter decreases from L1 to L5 as it transits from lumbar type to sacral type.

Risk factors to be watched for

It is illustrated in Figure 8.

The above findings are also supported by Todd et al.

Bilateral sciatica, progressive dysfunctional bowel, bladder and perianal functions.

CES can rarely be seen in pregnancy (especially in twin pregnancy).[22,23]

Metastatic spinal cord compression can also cause CES, scan-negative CES.

Hoeritzauer and colleagues introduced the concept of 'scan negative CES' where the patient has CES-type symptoms without compression on the MRI scan.

In slow CES the cauda equina nerves become thickened over time.[24]

Thickness of the cauda equina nerves: 2 to 3 mm.

Thickened nerves are not normal nerves because they become redundant nerve roots (RNRS), elongated, enlarged, tortuous nerve roots, which become more susceptible to compressive degeneration and are more likely to cause CES (as these (RNRs) are even more compressed in spinal stenosis, supported by the findings of the MRI, which reveals thickening, enhancement of the nerve roots indicating inflammation or degeneration.

If the radiologist reports the presence of RNRs, it conveys that these degenerated RNRs cause an increased risk of CES.

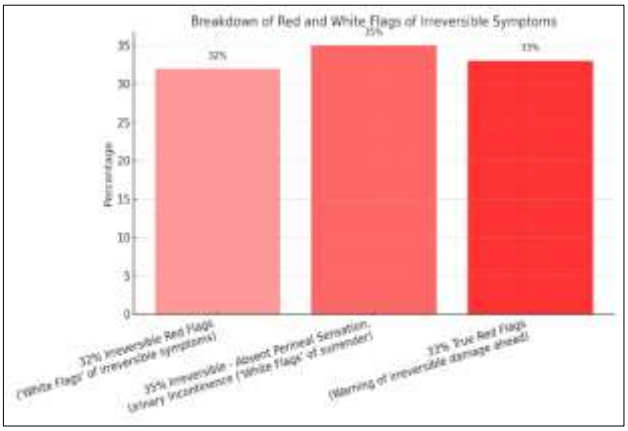

Figure 7: The warning signs – the true red flags [33%], where there is time for salvage the spinal function and the seemingly red flags which in reality are white flags of surrender, where even after surgery the outcome is bleek.[8]

Various aetiologies which cause thickening of the cauda equina nerves: inflammatory and infective causes like neuro sarcoidosis, arachnoiditis, neoplastic causes: primary tumours, secondary tumours, neurolymphomatosis, demyelinating causes, congenital: hereditary sensory motor neuropathy [HSMN]-Charcot-Marie-tooth 1A, acquired: Guillain-Barre syndrome (GBS), chronic inflammatory demyelinating poly radiculo neuropathy (CIDP), and pitfalls or dangers of missing these scenarios: filum terminale lipoma, thrombosis, and diastematomyelia.

Although usually not reported, the redundant nerves are seen in the MRI scan by a keen radiologist and reported.

The main problems of neglect arise due to a delayed diagnosis and treatment, specifically the time of surgery.[25]

The teacher should emphasise on the students to check for bulbo cabernous reflex which is 100% sensitive and specific compared to perianal sensations [60% sensitivity and 59% specificity], bulbo cavernous reflex [100% sensitivity and specificity], rectal tone [80% sensitivity and 86% specificity], post-void residual bladder [80% sensitivity and 59% specificity], and last is the perianal sensation [60% sensitivity and 59% specificity], which we

routinely and casually inquire about in our history taking, which can become a grave oversight.

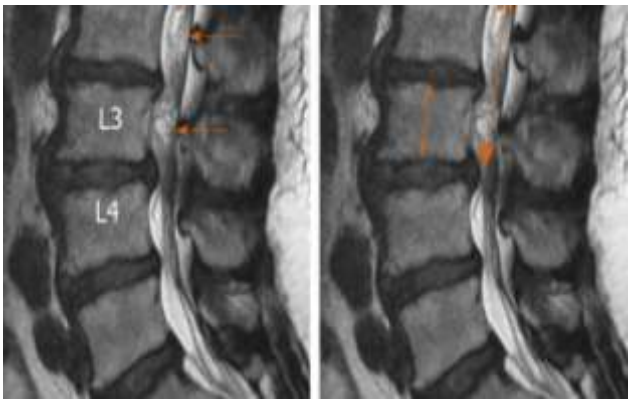

Figure 8: The sagittal section of the lumbar spine where, above and below a stenosed spine, the redundant nerves are visible and noticeable. A good radiology report alerts the clinician, allowing for early surgical intervention.

Image credits: Krushi Orthopedic Welfare Society, VTZ, A.P, India

Demographic data

Our study identifies 650 patients, Indians, mean age 43 years, patients selected after excluding similar CES like diseases and this study found an incidence of CES [S] to be 13% and the incidence of CES [I] to be 8%, incidence of CES [R] to be 1% and that of CES [C] to be of 0.5%. These various stages were found more in females between age groups of 30-60 with increasing occurrence of some degree of autonomic disturbance, earlier in congenital spinal canal stenosis and late in degenerative spinal canal stenosis.

No relation was found to the socioeconomic status.

Chief author's comment

Hence, the clinician must act quickly to avoid missing the early stages of CES.[26]

How many clinicians are doing the clinical tests? Physical examination has taken a back seat, and both the clinician and the patient are paying a high price. How many are testing the Bulbo cavernous reflex, rectal tone, and perianal sensation in every low back patient who does not show symptoms of bladder dysfunction?

When the sensitivity and specificity of peri anal sensation are so low…. why are the other more sensitive clinical tests not being emphasised? Are we not misguiding them?

The responsibility lies with the clinician to recognise the exact cause of progressive CES and its various rare aetiologies.

Clinicians from other fields may also advise patients to wait and watch at times. Therefore, this kind of hindrance also determines the patient's fate.

The acute lumbago may vanish as the compression decreases, but the questions remain.

Did the severe disc compression decrease to the point of near normalcy? Has the spinal canal become restored to its original at the end of the alternative therapy, exercises, yoga, loss of weight or with special procedures like Radiofrequency ablation, which only takes away the pain component in the lumbago … and does nothing to the compressing disc fragment?

Are we allowing the compression to prevail? Now we have to ask this 'how many patients with acute disc after RFA land in CES? To date, no studies have been conducted on this point.

The spinal canal may not always return to its exact original dimensions; it often improves with time and appropriate treatment, allowing for reduced pressure on the spinal nerves and alleviating associated symptoms.

Discussion of a patient with 13 years follow up done in Krushi Orthopaedic welfare society IRTP research cell

Patient with on off-low back pain, claudication and a recent increase in anal sphincter dysfunction.

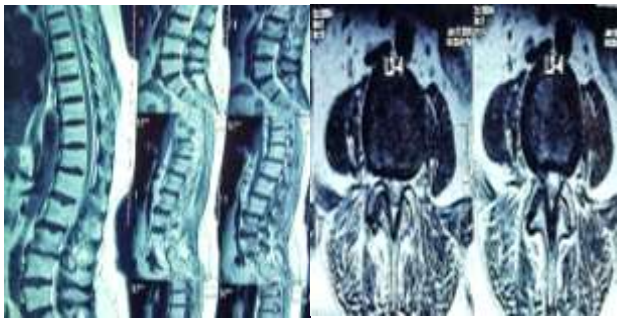

Figure 9: Severe bony spinal canal stenosis at L3-L4 with ligamentum flavum hypertrophy and disc bulge causing CES(S) stage anal sphincter weakness.

The hemangiomas at D11 and D3 did not progress but caused on-and-off pain. The lumbar claudication increased, and the cauda equina nerves were getting compressed due to increasing soft tissue spinal canal stenosis over the fixed bony stenosis, as can be seen in the reports and pictures of sequential MRI scan images. Patient currently is developing anal sphincter weakness and cannot hold her bowel for more than a few minutes in the mornings, a neurosurgeon who examined her advised physiotherapy and medication for neuropathic pain as she had no neurological deficit (the patient was not asked for bladder or bowel dysfunctions). An orthopaedic surgeon opined that she needs spinal canal decompression to release the pressure on the cauda equina nerves as there is

anal sphincter weakness and very severe claudication at the spinal canal in the lumbar region especially at L3-L4.

Here in this scenario the orthopedic surgeon wants to practice 'preventive orthopedics' while the neurosurgeon wants to wait further and watch while the patient is already in CES(R).

The patient is currently refusing surgery, but she has already progressed to the CES (I) stage.

Treatment

In the chief authors experience surgery is the mainstay choice – minimal open decompression is preferred especially when multiple levels lumbar discs or stenosis are involved which is a contraindication for endoscopic decompression. The reason being the better decompression that could be achieved in his vast experience with best results. However, there is probably a first publication 'single incision tubular decompression to treat multi-level lumbar spinal stenosis: a retrospective review' in 2022 for a brief mention on newer trends but the chief author prefers the open minimal procedures as of now.[27,28]

In the chief authors practical experience in the treatment of a recent incomplete CES patient [one case], external pudendal nerve and genital nerve stimulation practically helped the patient.[27] She regained her urethral and anal sphincter strength after being referred to a physiotherapist after a detailing by Dr. Kiran K. Nandivada V. S. and in a matter of 10 days the patient became catheter and diaper free. More study is needed to recommend this to all.

Limitations

Patients were lost to follow up, were dissuaded by different levels of clinical visits to other spinal and non-spinal surgeons who advised patients as per their own experience and learning curve. On the other side, patients were lost to follow-up as the journey of these patients can be for several days, months, and years.

CONCLUSION

Diagnosis and management is a team-based approach. The radiologist plays a vital role in preventing CE compression from progressing to a complete CES stage by giving elaborate reports to alert the clinician about the CE nerves thickening on magnetic resonance imaging. It is the responsibility of every clinician to document the deficit, however minor it may seem, because, at the next meeting, there could be an observable deficit – a time for immediate spinal surgery. The patients have chronic low back pain in nearly 70% of all cases of CES with or without sciatic pain, and bilateral symptoms usually appeared in the final stages. In the other 30% of cases, those with acute forms of CES, the diagnosis is usually delayed due to unidentified symptoms. Several studies and meta-analysis reports indicate a better prognosis if surgery is performed within the first 48 hours from the onset of symptoms. Once the vesical function is affected with or without neurological involvement, the surgical results are poor, and then early surgery does not have a good prognostic role. Open spinal decompression is the best option compared to endoscopic decompression because the decompression can never be adequate from an endoscopic decompression due to space limitations varying learning curves, and it also is a contraindication in multiple disc herniation.

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