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A STUDY OF THE PROGNOSTIC FACTORS IN CERVICAL SPONDYLOTIC MYELOPATHY

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF THE PROGNOSTIC FACTORS IN CERVICAL SPONDYLOTIC MYELOPATHY"** is the bonafide original work of **Dr. M.GANAPATHI VEL KANNAN** in partial fulfillment of the requirements for Branch – II, M.Ch Neurosurgery, examination of THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY to be held in August 2010. The period of post-graduate study and training was from July 2007 – August 2010.

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DECLARATION

I solemnly declare that this dissertation "**A STUDY OF THE PROGNOSTIC FACTORS IN CERVICAL SPONDYLOTIC MYELOPATHY"** was prepared by me in the Institute of Neurology, Madras Medical College and Government General Hospital, Chennai under the guidance and supervision of Professor of Neurosurgery, Institute of Neurology, Madras Medical College and Government General Hospital, Chennai between 2007 and 2010.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the University requirements for the award of degree of M.Ch. Neurosurgery.

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CONTENTS

INTRODUCTION

Cervical spondylosis is a common degenerative disorder of the cervical spine which affects almost every person over 40 years, earlier or later. Pre existing cervical canal narrowing either congenital or acquired makes the patient vulnerable to neurological deficit with onset of cervical spondylosis. Cervical spondylotic myelopathy (CSM) is thought to be caused by cervical canal narrowing due to disc protrusion, ossification of posterior longitudinal ligaments (OPLL) or degenerative spondylosis.

Typically, patients with myelopathy have symptoms and signs for several years before seeking medical attention. Although the progression is usually slow, the course often involves a progressive decline if the disease is left untreated. A small percentage of patients exhibits a more rapid onset of progression of symptoms and signs.

Once patients have presented with the signs and symptoms of cervical spondylotic myelopathy, most have some degree of permanent disability, little changes of symptoms resolution is possible with conservative treatment, therefore many different surgical methods have been developed to expand the cervical spinal canal anteriorly or posteriorly. Although the surgical outcome is directly associated with preoperative severity, there is little information about the role of factors

which determine the outcome. Several studies has been done to determine the prognostic factors in outcome.

Many factors have been studied like, patients age, duration of symptoms, pathological changes to the spinal cord, cervical axial canal area, anteroposterior diameter, intramedullary high signal intensity on T_2 weighted Magnetic Resonence images(T_2 MRI) and their effect in prognosis.

The various factors affecting the prognosis in CSM have been studied by various authors. But there is no comprehensive study comparing the impact of various factors on the outcome.

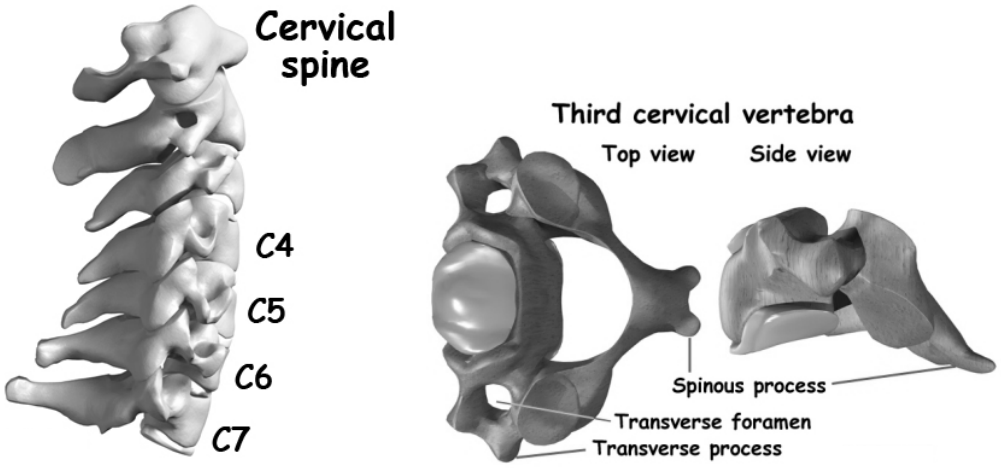
In this study, an attempt has been made to study the impact of various individual factors affecting the diagnosis in CSM. A comprehensive prognostic scale incorporating the prognostic factors has been evolved and this scale also has been evaluated.

AIM OF THE STUDY

- To assess the role and effect of the following factors in the outcome of the patients treated for CSM surgically.
    - Patient's age
    - Duration of symptoms
    - Neurological disability (Nurick's Grade)
    - Effective canal diameter
    - Number of levels of compression
    - Hyperintense signal changes in T_2 MRI
- To evaluate a new prognostic scoring system to determine the outcome of the CSM patients preoperatively.

REVIEW OF LITERATURE

RELEVANT ANATOMY



The cervical spine is uniquely adapted to allow for a wide range of motion including flexion, extension, rotation and lateral bending. It consists of seven vertebrae stacked on top of each other, spinal ligaments, and the cervical segments of the spinal cord, which run within the spinal canal (vertebral foramina).

The C_1 (atlas) and C_2 (axis) vertebrae are anatomically different from the other vertebrae. They are shaped to allow flexion, extension and lateral rotation at the occipital-C_1 and C_1-C_2 joints respectively. Normally, the cervical spine has a lordotic curvature, i.e., it is concave posteriorly.

Vertebrae are made up of anterior vertebral bodies, which bear 90% of the load placed on the spine, and posterior vertebral arches. The

arch is formed by bony structure called pedicles and lamina. There are five joints between all adjoining cervical vertebrae except C_1 – C_2: an anterior curvilinear disc separating adjacent vertebral bodies. The C_1 – C_2 articulation lacks an intervertebral disc. The transverse process of cervical vertebrae are modified to form lateral masses in cervical vertebrae. Synovial joints known as facetal joints connect adjacent cervical vertebrae posterolaterally.

Spinal ligaments include the anterior and posterior longitudinal ligaments, which are continuous bands that run along the vertebral bodies, and the ligamentum flavum, a thick band that attaches between the lamina of each vertebra.

The spinal canal houses the spinal cord and is surrounded anteriorly by the vertebral bodies, intervertebral discs, and the posterior longitudinal ligaments, laterally and posteriorly by the bony vertebral arch and posteriorly by the ligmanetum flavum.

The largest area of the cervical spinal canal is C_1, from C_2 distally, the cervical spinal canal funnels down, markedly decreasing the diameter for the cord. At the C_1 level, the spinal cord occupies just one half of the canal. It occupies three quarters of the canal at the C_5- C_7 levels, which helps to explain why cervical spondylotic myelopathy predominantly occurs in the lower cervical spine. The normal cervical spinal canal from

C_3 to C_7 is 17mm to 18mm in its sagittal diameters. Diameters of fewer than 12 mm have been found to be critical in the development of cervical myelopathy. Spinal cord is thickest in cervical region. The canal size does not increase in size after 10 years.

The cervical spinal cord receives its blood supply from the anterior spinal artery, paired posterior spinal arteries and radicular arteries. Anaestamoses are usually insufficient, and occlusion of one radicular artery places the cord at risk for infarction. Within the ventral median fissure, the anterior spinal artery divides into short or proximal branches that supply the anterior funicular and the central gray matter. The anterior spinal artery also is responsible for providing perfusion via long perforating branches that terminate in the lateral funiculus. The lateral corticospinal tracts are supplied primarily by the long perforating branches with the medial portion of these tracts receiving additional supply from posterior spinal arteries and short perforating branches. The descending fibers of the cortical spinal tracts are arranged somatotopically. The fibers entering the upper extremities are situated more medially and the fibers responsible for innervating the lower extremity are located more laterally.

Historical note and Nomenclature

Cervical spondylosis is a common degenerative condition of the cervical spine. It is a combination of degenerative changes in the intervertebral disc, the facet joints and the ligamentum flavum.

Cervical spondylotic myelopathy in the most serious consequences of this degenerative process, especially when associated with a congenitally narrow cervical vertebral canal (Kadoya et al 1985)[1]. As Benzel (200)[2] has shown spondylosis is a natural process of agony is seen in 10% of individuals by the age of 25 years and in 95% by the age of 65 years and is often preceded by mild segmental instability (Benzel 2001; Shedid and Benzel 2007)[3].

The combination of these degenerative changes causing narrowing of the cervical spinal canal (spondylosis) and spinal cord injury (myelopathy) give rise to the term “Cervical Spondylotic Myelopathy” (CSM).

Although the pathological existence of intervertebral disc herniation was realized as early as 1929 by Schmorl and Andrea, its clinical correlation with well described syndromes of compressive myelopathy was not thoroughly reviewed until 1956 by Clark and Robinson.

Direct surgical attack of the anterior compressive disc spur complex was not developed until the late 1950s with the work of Smith and Robinson (1958) and Cloward (1963).

Epidemiology

Chronic cervical degenerative changes of the cervical spine are the most common cause of progressive spinal cord and nerve root deterioration (Crandall and Batzdorf 1966)[4].

Ninety percent of men older than 50 years and of women older than 60 years display radiographic evidence of degenerative changes of the cervical spine (Batzdorf 1991)[5].

Increased recognition and treatment of CSM over the 10 years has led to doubling in admission and seven fold increase in its surgical treatment in stanford university (Lad et al 2009)[6].

Etiology

Although the cause of cervical spondylotic myelopathy has not been definitively pinpointed, 4 main theories for the development of this disease are available including,

1. Vascular hypothesis
2. Compression hypothesis
3. Hybrid of the first 2 (Mouw and Hitchon 1996)[7].

4. Dentate ligament theory (Levine 1997)[8].

The cervical spinal cord receives its blood supply from the anterior spinal artery, paired posterior spinal arteries and radicular arteries. Anaestomosis are usually insufficient and occlusion of one radicular artery places the cord at risk for infarction.

The lateral cortical spinal tracts are supplied primarily by the long perforating branches of the anterior spinal artery with the medial portion of these tracts receiving additional supply from posterior spinal arteries and short perforating branches.

After examining cross sections of the cadaveric spinal segments, Breig and colleagues (1966)[9] noted that the anteroposterior compression and compensatory lateral widening of the spinal cord compromise the longer perforating vessels .

Therefore ischemia secondary to compression may affect the lateral columns more than the anterior columns, explaining why the lower extremities are affected earlier and more severely than the upper extremities (Mouw and Hitchon 1996)[7].

In the last hypothesis, Levine(1977)[8] showed that in mechanical model of cervical spinal stenosis, tension transmitted from stretched dentate ligaments to the cord predicted pattern of injury typically seen clinically .

Pathogenesis and pathophysiology

A functional spinal segment consists of the vertebral body with its intervertebral disc both superiorly and inferiorly. A component of this functional spinal segment is the three joint complex consisting of the intervertebral disc, the superior facet and the inferior facet (Farfan 1980)[10].

As the human disc ages, it loses elasticity, disc space height and the ability to distribute forces. These changes alter the force vectors of the posterior elements. Annular bulge increases mobility, and setting of the adjacent vertebral bodies result in buckling of the ligamenting flavum.

Osteophytic spur formation occurs as an attempts to offset this increased dynamic mobility. Osteophytic formation provides stabilization between both adjacent vertebral bodies and increases the weight bearing surface of the vertebral body endplates (Brain et al 1952)[11].

The posteriorly and posterolaterally evolved spurs with facetal hypertrophy, plays a dynamic role in narrowing the overall surface area of the cervical spinal canal.

Some patients suffer from dynamic myelopathy. This syndrome consists of patients having few symptoms when maintained in the normal or flexed position, but marked exacerbation when the neck is extended,

caused by buckling of the ligamentum flavum and posterior longitudinal ligament combined with osteophytic narrowing in hyperextension.

Clinical manifestations

Patients with cervical spondylosis usually present with neck pain with or without radiculopathy or myelopathy. Even painless progression of sensorimotor deficit is seen in patients with more centrally located compression, as the roots are relatively spared.

Myelopathy develops in approximately 5% to 10% of patients with clinically symptomatic spondylosis (Young 1991)[12] but cervical spondylosis is the most common cause of myelopathy in middle aged and elderly patients (Crandall and Batzdorf 1966 ; Montgomery and Brower 1992)[13]. It rarely seen before the age of 40 and often no injury is elicited or the patient reports only a series of minor or repetitive stress.

Because CSM may involve compression of the descending lateral corticospinal, ascending spinothalamic, posterior column as well as compression of the lower motor neurons in the central gray of the spinal cord, patients present with a variety of symptoms.

Emphasis should be placed on obtaining a history of numbness and clumsiness in the hands, decreased fine motor movements, and subtle gait disorders. Suspicion of the clinical entity remains the single greatest element in early identification of CSM.

The clinical features, duration and natural history of this condition vary considerably because of their relationship to the inconstant combination of mechanical, dynamic forces and period of vascular ischemia (Mc Cormick 1992)[14]. Consequently various pattern of myelopathy have been described.

Crandall and Batzdorf (1996)[4] classified patients into 5 groups based on their dominant syndromes.

(i) The transverse syndrome involves the corticospinal, spinothalamic and dorsal column tracts and produces severe spasticity, frequent sphincter involvement and Lhermitte sign.

(ii) The motor system syndrome involves the anterior horn cells and corticospinal tracts and produces marked spasticity but no sensory disturbances.

(iii) The central cord syndrome produces severe motor and sensory disturbances with a greater expression of weakness in the upper extremities and spasticity in the lower extremities.

(iv) The Brown–Sequard syndrome produces the typical contralateral sensory deficits and ipsilateral motor deficits.

(v) The brachialgia and cord syndrome involves the lower motor neurons of the upper extremities and produce radicular pain.

An additional presentation of cervical spondylotic myelopathy is posttraumatic central cord syndrome. In these relative minor trauma, can precipitate a neurologically devasting injury. The pathophysiology is thought to involve vascular injury to the relatively poorly perfused medial region of the cord from buckling of the hypertrophic ligament both anteriorly and posteriorly.

The physical findings associated with cervical spondylotic myelopathy vary depending on the exact level of compression, the degree of compression modified by aggravating factors and the span of segments compressed in the cervical spinal cord. Symptoms may be characterized by lower motor neuron involvement at the level of clinical lesion and upper neuron involvement at the levels below the site of compression.

Clark (1988)[15] proposed that sensory findings in myelopathy usually include the loss of pain and temperature, proprioception, and vibration below the level of the lesion, with relative sparing of touch.

The most common presentation is spastic weakness of the hands and forearms before involvement of proximal upper extremity muscles and hand numbness with paresthesias which may be painful. In the lower extremity, the proximal musculature is affected early, making it difficult for the patient to get up out of a chair, get out of car or climb stairs.

Wasting of hand musculature is a late findings and is often symmetric because of the central nature of osteophytic compression in the canal.

Reflex are generally hyperreflexic below the level of compression and hyporeflexic at the level of the anatomic lesion. Pathologic reflexes such as the presence of the Babinski reflex in the lower extremities and Hoffmann reflex in the upper extremities, characterize upper motor neuron involvement in cervical spine. Furthermore, clonus may be present in the lower extremities. Lhermitte sign may be present when the patient flexes and extends the neck, producing a feeling of electrical shock down the spine and is classically attributed to dysfunction of the posterior columns.

Lunsford and colleagues(1980)[16] reported that although a significant number of patients manifest hyperreflexia (87%) only about 50% have the Babinski reflex and about 20% have the Hoffmann reflex.

It should be remembered that at least 10% to 20% of patients with cervical spondylosis have some degree of symptomatic lumbar stenosis as well, blunting the increased tone and hyperreflexia of the lower extremities produced by the upper spinal compression and adding radicular leg pain (Shedid and Benzel 2007)[3]. Treatment should be first be directed towards the cervical component, which often improves or

alleviates the lumbar component. Bladder and bowel symptoms tend to occur infrequently and late in cervical spondylotic myelopathy. Bladder dysfunction was demonstrated in 15% and bowel dysfunction in 18% of the patients (Hukuda et al 1985)[17] Epstein and colleagues (1987)[18] found that 20% of patients exhibited bladder dysfunction with various degrees of urinary retention. The most common urinary symptoms in early spondylotic myelopathy are urinary urgency and frequency, particularly in women. Impotence in males may occur with more chronic and severe cord compression.

Myelopathy hand, introduced by Ono and colleagues in (1987)[19], is a loss of power of adduction and extension of the two ulnar fingers and inability to rapidly grasp and release these fingers. Burning hand syndrome from chronic vascular injury to the medial sensory tracts tends to be bilateral and symmetric, provoked by light touch that produces sustained burning. This is generally an indication of late permanent hyperpathic sensory derangement that may respond little or not at all to surgical decompression.

Gait abnormalities are noticeable in patients with cervical spondylotic myelopathy. Gorter(1976)[20] noted that cervical myelopathy usually presents initially as subtle gait disturbance with gradual deterioration . He stated that spasticity and paretic dysfunction occur first

and are followed by numbness and loss of fine motor movements in the upper extremities. A myelopathic gait with a resistant jerking motion may appear.

Prevention

The only known prevention for cervical spondylotic myelopathy is maintaining nutritional health, avoiding cigarette smoking, which accelerates degenerative changes in the intervertebral disc and attention to proper posture, including ergonomics in the workplace.

Beyond this, early recognition of the condition with surgical decompression can prevent a spinal degenerative process from resulting in a permanent neurological syndrome.

DIFFERENTIAL DIAGNOSIS

Cervical spondylotic myelopathy usually affects patients over 50 years of age, although the disorders is the most common cause of cervical cord dysfunction in the elderly, radiographic confirmation is warranted to confirm that an observed myelopathy is caused by cervical degeneration and not by another pathological process.

Cervical spine instability with canal compromise due to chronic subluxation is not uncommonly seen in the elderly patient and should be easily distinguished radiographically and by MRI.

Similarly, $C_{1–2}$ instability from rheumatoid pannus and chronic cervicomedullary compression may have a similar presentation.

Other conditions whose presentation may mimic cervical spondylotic myelopathy include disseminated sclerosis, multiple sclerosis, AIDS myelopathy, lupus myelopathy, beta lipoproteinemia, B_{12} deficiency, tumours, syringomyelia, chiari malformation, primary lateral sclerosis and vertebrobasilar ischemia.

Although cervical spondylotic myelopathy can frequently present as an isolated, painless, spastic lower paraparesis, if the imaging does not support a cervical compressive cause, the diagnostic workup should aim higher. Chronic subdural hematoma and sagittal meningioma may produce this syndrome as well.

An important diagnosis to consider in the differential is amyotrophic lateral sclerosis, in which patients present with a combination of upper and lower motor neurons disease as well. However, the key differentiating factor is the region of lower motor neuron involvement, the patients with cervical spondylotic myelopathy usually have cervical nerve root related lower motor neuron symptoms and signs; whereas patients with amyotrophic lateral sclerosis have other lower motor neuron affliction as well.

To complicate matters, one study found nearly 48% of amyotrophic lateral sclerosis patients have some degree of cervical spondylosis, with 8% undergoing surgery for progressive symptoms (Yamada et al 2003)[21].

Electromyographic findings, such as thoracic paraspinal muscle denervation preferentially in amyotrophic lateral sclerosis versus cervical spondylotic myelopathy, have been described as possible aids in differentiating diagnosis (Kuncl et al 1988)[22]. Tongue atrophy and EMG (Heffez et al 2004)[23] evidence of tongue muscular denervation favours the diagnosis of amyotrophic lateral sclerosis.

Diagnostic work up

Most middle aged and elderly individuals suffering from spinal cord afflictions have some degree of degenerative changes of the cervical spine on imaging studies; therefore a careful history and meticulous examination are essential for the accurate correlation of abnormalities observed with imaging and the patient clinical picture.

Plain radiographs including anterior, posterior, lateral and oblique views should be obtained for patients with cervical spondylotic myelopathy. Flexion and extension radiographs are important for further evaluation of a patient with suspected cervical instability.

Plain radiographs provide important information with respect to mass lesions, infection, trauma and congenital abnormalities. The size of

the spinal canal can be assessed and anatomic landmarks can be identified that may be helpful for intraoperative localization.

However plain radiographs do not provide an adequate assessment of soft tissue anatomy, the overall spinal column dimensions or the spinal cord architecture.

In mid 1980s, prior to the advent of MRI, the gold standard for imaging was CT myelogram. It provides better definition of a herniated disc or spondylotic ridge. It provides an accurate assessment of the effect of the spondylotic ridge on the spinal cord itself. It also provides evaluation of the cross sectional area of the cord and subarachnoid space, which may be of prognostic value.

In a clinical review, Badami and colleagues (1985)[24] found that patient with cord to subarachnoid ratio of greater than 50% had good functional recovery after surgical decompression. The disadvantage of CT is that it exposes the patient to a formidable amount of radiation and requires a lumbar or cervical spinal tap, with a potential for the potential common complication (10% incidence) of postmyelographic headache and much rarer complication of nerve or spinal cord injury.

MRI allows the examination of the cervical spine for disc and spondylotic disease without exposing the patient to ionizing radiation or invasive myelography. It provides excellent detail of the spinal cord,

nerve roots, sub arachnoid space and soft tissue abnormalities like soft disc herniation and is preferred in assessing the presence of an intramedullary process and intradural or extradural neoplastic processes.

MRI may also provide prognostic value in patients undergoing operative decompression. In a study of more than 600 patients, 56% of patients whose MRI demonstrated increased signal intensity within the spinal cord on T_2 weighted images, indicating spinal cord damage, had no improvement following decompression compared with 15% of patients with no abnormal signal (Takahasi et al 1989)[25].

One option to better define the bony anatomy for measuring canal size, evaluate the quality of previous fusion areas, and determine the size of pedicles and location of vertebral artery foramina for surgical planning is a thin section cervical CT. This study avoids the risks and patient discomfort of a myelogram and coupled with contrast MRI, will usually suffice for preoperative evaluation.

Management

Ideally, cervical spondylotic myelopathy is a surgically treated entity but certain patient presents an unacceptable risk, frequently pulmonary or cardiac, for general anaesthesia and surgical intervention. Attention is then directed at medical management of chronic arthritic and neuropathic pain, spasticity and associated bladder dysfunction as

indicated (Mazanek and Reddy 2007)[26]. These techniques are frequently effective in management of the postoperative patient with residual symptoms.

Advances in spinal imaging and accumulation of clinical experience have provided some clues as to indications and timing of surgery for cervical myelopathy. When surgery is properly carried out long term results are expected to be good and stable. Surgery to relieve the compression may reduce the pain and disability though it is associated with a small but definite risk. The short term effects of surgery, in terms of pain, weakness or sensory loss, have been proven to be superior.

Analysis of the literature regarding CSM does not demonstrate conclusively the superiority of either the anterior or the posterior approach (Carol and Ducker 1988)[27]; however, no true comparative prospective trial has ever been established.

Instead, successful surgery depends on proper patient selection and selection of the approach that provides optimal decompression of the spinal cord. As a general rate, the anterior approach is appropriate for patient's whose pathological compression occur anterior to the spinal cord at three or fewer vertebral segments. For most patients with cervical disc disease or spondylosis, the compression lesion lies anterior to the spinal cord. For this reason, it is felt that the anterior approach provides

more through and complete decompression. Also, for patients whose preoperative lateral cervical spine reveals a kyphotic deformity, the anterior approach is mandated for cervical decompression because a posterior approach could exacerbate the kyphosis, worsening the myelopathy.

The two most common anterior approaches for disectomy are (1) the Cloward technique and (2) the Smith – Robinson Technique.

The Smith - Robinson technique replaces the removed intevertebral disc and osteophytic bar with a tricortical horseshoe – shaped graft. Initially this was provided by harvesting a tricortical wedge from the patient's iliac crest. Over the past two decades, this technique has been replaced by bone from other sources, including allograft iliac crest and machined corticocancellous allograft spacers that can achieve comparable rates as autografts, particularly with availability of fusion "enhancers" such as demineralised allografts bone matrix.

The introduction of anterior cervical plating systems has also improved poster operative alignment and fusion by supporting the fusion construct with a more even distribution of forces on the grafts and the patients own bone.

In the Cloward technique, the superior and inferior endplates as well as the interverbral discs are removed in a circular fashion and the

removed segments are replaced with a round dowel graft. In order to achieve adequate decompression, the posterior osteophytic bar must be resected regardless of technique performed.

Bone grafting is an adjuvant in the treatment of cervical spondylotic myelopathy via the anterior approach. Though previous studies (Grisoli et al 1989)[28] have shown that no difference exists in single level fusion versus no fusion, others (Schemidek and smith 1988)[29] have advocated that bone grafting alleviates immediate post operative pain and helps prevent further kyphosis that may render the spinal cord more compressed, worsening the myelopathy. Regardless of the technique used, patients should have some type of bone grafting procedure performed to eliminate kyphosis at the level of fusion.

Preferred options for interbody fusion materials include iliac crest or fibular strut autograft, cadaver allograft, and titanium or PEEK (polyether ether ketone) cage packed with demineralised bone matrix (DBM) or recombinant bone morphogenic protein (BMD).

PEEK cages have the advantages of modulus of elasticity similar to bone, allow minimal subsidence during the month of on going fusion and thus maintained the restored foraminal and disc space height and lordosis achieved at surgery (Kulkarni et al 2007)[30].

Use of BMD cause postoperative soft tissue swelling, dysphagia and even fatal airway obstruction, this complication is likely dose dependent and further studies are needed to determine safety of BMD (Vaidhya et al 2007)[31];(Tumialan et al 2008)[32].

The advantage of maintaining or restoring motion after anterior decompression of the cord by the placement of prosthetic cervical disc has not been established in the generally older spondylotic population; the real or theoretical risk of accelerated adjacent segment disc deterioration following fusion is more a concern for the younger, more active patient with soft disc rupture (Seo and Choi 2008)[33].

Multiple anterior discectomies with resection of associated osteophytes in spondylotic myelopathy may not allow adequate visualization and resection of the compressive complex; significant spurring may be left against the cord behind the vertebral bodies above and below one or more disc space. For this reason, anterior medial corpectomy with strut grafting may be recommended for patient's whose compression spans multiple herniated disc segments. This approach allows complete removal of the anterior cord impingement, creates a stable spine and may avoid the pseudoathrosis that can occur with multiple level grafts in the elderly, osteoporotic patient.

Fessler and colleagues (1998)[34] performed multilevel corpectony and fusion with or without instrumentation in 93 patients. Symptomatic improvements was seen in 92% of patients, and there was complication rate of 18%.

Whether performed as one or more anterior discectomies or as a corpectomy, the specific risks of the anterior approach include spinal cord injury as well as failure to adequately decompress the spinal cord, nerve root injury and dural tear with spinal fluid leak, injury to soft tissue dissected (esophagus, carotid sheath structure), injury to the recurrent laryngeal nerve and vocal cord paralysis, sympathetic chain injury (Horner syndrome), hematoma formation with compromise of airway or spinal cord, infection and failure to achieve fusion.

More studies are coming out advocating the use of PEEK cages in place of allograft bone, demonstrating comparable rates of fusion in elderly patient while avoiding reliance on bone bank materials.

Posterior technique are performed in patients whose pathologic compression encompasses more than 4 vertebral body segments, and for those patients in which the posterior canal compromise by thickened ligamentum flavum, overgrown facet joints or congenitally short pedicles is as great as or greater than the anterior compression elements.

The two techniques used for posterior decompression include cervical laminectomy with or without lateral mass plating or laminoplasty. Though both techniques allow for adequate posterior decompression, laminoplasty was developed to help maintain posterior stability and prevent postoperative laminectomy kyphosis.

The incidence of post laminectomy kyphosis done without fusion has been reported at 21% (Steinmetz et al 2003)[35] but may be less in those patients who present with adequate cervical lordosis before surgery.

Lateral mass plating has virtually eliminated the need for cervical laminoplasty in the adult spine, as it achieves a high rate of fusion without risk of recurrent canal compromise (Horgan et al 1999)[36].

Gok et al 2009[37] reported 81% improvement in myelopathy over 17 months of postoperative recovery in 54 consecutive patients undergoing laminectomy and instrumented fusion.

Specific risks of the posterior approach for spinal cord decompression include spinal cord injury or failure to adequately decompress the spinal cord, dural tear with spinal fluid leak, instability and late kyphosis, infection, hemorrhage and hematoma formation and combined with posterolateral fusion, nerve root and vertebral artery injury and failure to achieve fusion.

Literature on the prognostic factors in cervical spondylotic myelopathy

Cervical spondylotic myelopathy is an intermittently progressive disease process without significant change of reversal when no treatment is administered. Conservative therapy rarely solves the myelopathy, most have some degree of permanent disability with little chance of resolution of symptoms; therefore patients with moderate or severe myelopathy are candidates for surgery. Many different surgical methods have therefore been developed to expand cervical canal anteriorly and posteriorly.

The most important factors for successful outcomes in patient treated surgically are related to

(i) Age of the patient

(ii) Duration of symptoms

(iii) Neurological disability (Nurick's grade)

(iv) Effective canal diameter

(v) Number of levels of compression

(vi) Hyperintense signal changes in T_2 MRI

(i) Role of age in prognosis

Cervical spondylotic myelopathy develops approximately in 5 to 10% of patients with clinically symptomatic spondylosis (Young et al

1991)[12] But cervical spondylosis is the most common causes of myelopathy in middle age and in elderly patients (Crandall and Batzdorf et al 1966)[5]. It rarely seen before the age group of 40 years.

Fujiwara et al(1989)[56], conducted a prospective study and concluded age, canal stenosis are important predictors of prognosis .Kun et al(2005)[48], in Korean study analysed 13 prognostic factors and said age play an role in outcome. Jae Sung et al (2010)[51] in his prospective study also concluded age is an one of the factor in outcome, and patients less than 40 years have better prognostic value.

Langston (2009)[57], in a prospective analysis found that age, preoperative sensory evoked potential, duration of symptoms and pre operative Nurick's grade are the important factors deciding outcome. Fouyas et al (2002)[58], in 10 years of prognostic outcome study, concluded age forms an important factor.

(ii) The Role of Duration of symptoms in outcome

Prolonged compression of the spinal cord can result in irreversible histological and physiological changes such as intraneural fibrosis, demyelination and loss of neuron within the spinal cord. The results of operative treatment is generally are better in patients who undergo decompression early than later.

Lees and Turner (1963)[38] concluded clinical exacerbation added to neurological deficits in patients with more than 10 years of myelopathy. Symon and Lavender (1967)[39] showed 67% of patients displayed a linear relentless progression of neurologic deterioration rather than stabilization. Suri et al (2003)[40] in a prospective study of 146 patients with cervical spondylotic myelopathy noted that patients with less than or one year duration of symptoms showed significant greater motor recovery following operation than did those with a longer duration of symptoms ($P<0.005$)

Tanaka et al (1999)[41] in a study of forty seven patients who were more than sixty five years old, found that the preoperative duration of symptoms strongly influenced recovery of function following operative treatment, the authors recommended that decompression surgery should be attempted even in patients who are more than eighty years old, provided the duration of symptoms is less than three years. Lee et al (1997)[42] in a prospective study has shown that patients with duration of symptoms less than a year shows better prognosis than patients with duration of more than one year. Yony Jinkun et al (2007)[43] in a prospective study of 26 patients showed shorter duration of symptoms showed better results.

(iii) Role of effective canal diameter in prognosis

The importance of size and shape of the spinal canal in connection with the occurrence of symptoms of cord compression and recovery pattern has long been recognized.

Lindgreen (1937)[44] who first pointed out the importance of sagittal diameter in the cervical region. Burrows (1963)[45] measured the sagittal diameter of the 300 normal adults and compared with the spinal canal of cervical spondylotic patients and concluded that the sagittal diameter of the spinal canal is of definitive diagnostic significance in cervical spondylotic myelopathy.

Sodeyama et al(1999)[46] recommended posterior decompression of the cervical spinal cord in patients with myelopathy who had multiple levels of impingement of the spinal cord with spinal canal diameter of < 11 mm. Handa et al(2002)[47] has proved that canal stenosis is a main factor determining the prognosis. Kan et al (2005)[48] has analysed 13 prognostic factor and found canal diameter has a positive correlation with the prognosis of the patient.

Jae Sung et al (2010)[51] in a prospective study showed that sagittal canal diameter and number of segments involved is a main prognostic factor in cervical spondylotic myelopathy. Post laminoplasty patients

with post operative canal diameter above 12 mm showed good recovery. [Kohno et al (1997)][49]

The diameter seem to be, on average, 3 mm smaller in patients with cervical spondylosis and even smaller in patients with congenital cervical stenosis.The dimension of the cervical canal corresponds to the distance from the spondylotic process to the dorsal aspect of cervical spinal canal.

White and Panjabi (1988)[52] reported that the patients with diameters less than 14.8mm were at greater risk of developing cervical spondylotic myelopathy. Fergusson and Caplan (1985)[53] and Pattern (1977)[54] reported the distance must be less than 13mm, where as Asgari (1996)[55] and Fager (1973)[56] wrote that the average spinal canal diameter in patients with myelopathy was 14mm with normal diameter between C_3 and C_7 being approximately 17 to 19mm with slight variation between sexes.

(iv) Role of extent of disease involvement in prognosis of cervical spondylotic myelopathy

Myelopathy from the primary pathophysiology mechanism of cervical spondylosis may be referable to one or more cervical segments. The disease process is typically contiguous, but may proceed rostrally and/or caudally.

Crandall and Batzdorf (1966)[4] observed that the involvement of two intervertebral levels was the most common and always included the C_{5-6} interspace. The most affected levels by both disc herniation and chronic spondylosis are C_{6-7}, followed by C_{5-6}. As stated earlier, osteophytes formation is accelerated by motion and is therefore more common at C_{5-6} and C_{6-7}, where most of the cervical flexion and extension occur.

Fujiwara et al (1989)[50], in a prospective study, showed that number of level of involvement contribute an important factor for prognosis. Jae Sung Abu et al (2010)[51] has also noted number of segments involvement is an important prognostic factor. One or two segment involvement has best prognosis compared to three or more segments involvement.

(v) Role of hyperintense signal changes in T_2 MRI

Magnetic resonance imaging may show focal areas of signal change within the cervical spinal cord at or adjacent to sites of maximal compression. Low signal abnormalities on T_1 weighted images and high signal abnormalities on T_2 weighted images have both been associated with greater clinical disability or decreased neurological recovery following decompressive surgery.

These changes, generally referred to as myelomalacia, may represent intraspinal oedema, neuronal death, proliferation of neurological cells, and/or demyelination. Earlier operative intervention may be indicated for patients with these changes in an attempt to halt or reverse the changes within the substance of the spinal cord.

Takahashi et al (1989)[59], described the MR imaging findings of intra medullary high signal intensity on T_2 weighted MR images in cervical spondylotic myelopathy. Matsuda et al, (1991)[60] on analysis of 29 compressive myelopathy discussed the role of increased T_2 signal in prognosis. Since then, a number of authors, Mehalic et al (1990)[60], Harada et al (1992)[61], Hukuda et al (1996)[62] have reported that intra medullary T_2 hyperintense signal change is a predictor of poor recovery after surgical decompression.

Morio et al (1994)[63], Wada et al (1995)[64], Okais et al (1997)[65] on the other hand, have reported no clear correlation between the surgical outcome and intramedullary hyperintense T_2 changes.

Chi Jen Chen et al, (2001)[66], subdivided intramedullary hyperintense signal intensity in T_2 images into two types. Type I had a predominantly faint and fuzzy border and Type II had a predominantly intense and well defined border. On prospective analysis of 64 patients, patients with well defined border of intramedullary hyperintense signal on T_2 weighted images had a worse prognosis.

MATERIALS AND METHODS

The study was conducted between September 2007 to March 2010 in the Institute of Neurology, Government General Hospital, Chennai. Our study population consists of people from all over Tamilnadu and Southern Andhra Pradesh.

A total number of 85 patients were included in this study. All these patients were selected from those treated in the Neurosurgery department of the Government General Hospital, Chennai.

Patients with CSM, having definitive clinical and MRI features were included in the study. Other causes of spastic quadriparesis were carefully excluded. The patients who were not fit for surgery due to anaesthetic problems were excluded from the study. The follow up duration is from minimum of 2 months to maximum of 2 ½ years. Patients who lack regular follow up are excluded from the study.

The detailed history, duration of symptoms, mode of progression of weakness, highest level of cord compression, severity of involvement were studied and charted out.

Nurick's Grading was used to evaluate the severity of cervical myelopathy preoperatively and post operatively.

NURICK'S DISABILITY SCORE

Grade	Signs and Symptoms
0	Signs or symptoms of root involvement but no evidence of spinal cord disease
1	Signs of spinal cord disease but no difficulty walking
2	Slight difficult in walking that prevented full time employment
3	Difficult in walking that prevented full-time employment or the ability to do all housework, but that was not so severe as to require someone else's help to walk.
4	Able to walk only with someone else's help or with the aide of a frame
5	Chair bound or bed ridden

Patients age, duration of symptoms, preoperative neurological disability (Nurick's grade), effective canal diameter, number of levels of compression, intramedullary hyperintense signal changes in T_2 MRI, were evaluated.

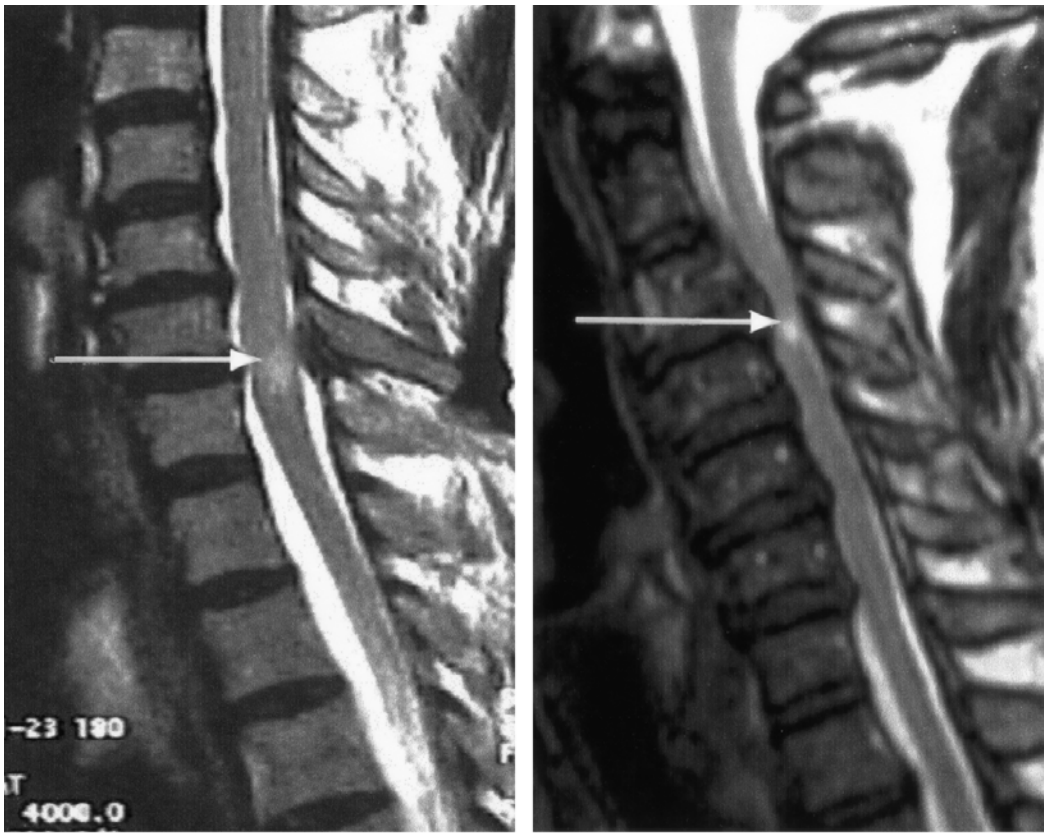
A new prognostic scoring system has been devised in the Institute of Neurology (The MIN prognostic scale for CSM), incorporating the major prognostic factors and has been evaluated.(vide Table below)

MIN PROGNOSTIC SCALE FOR CERVICAL SPONDYLOTIC MYELOPATHY

PROGNOSTIC FACTORS	SUBDIVISIONS	SCORE
AGE	< 40	3
	40 – 60	2
	> 60	1
Duration of symptoms	< 1 Year	3
	1 – 2 Years	2
	> 2 Years	1
Neurological Disability (Nurick's Grade)	0 – 2	3
	3	2
	4 to 5	1
Effective canal diameter	> 11 cm	3
	9 – 11	2
	< 9 cm	1
Number of levels of compression	1 level	3
	2 levels	2
	3 or more levels	1
Intramedullary signal changes in MRI	No change	3
	T_2 signal ill defined	2
	T_2 signal well defined	1

Total score - 18

Hyperintense Signal Change in T_2 MRI



a. b.

a. **T_2 Signal ill defined**

b. **T_2 signal well defined**

Surgical methods

The anterior approach is selected for patients whose pathologic compression occurs anterior to the spinal cord at three or fewer vertebral segments. Also, for patients whose preoperative lateral cervical spine reveals a kyphotic deformity, the anterior approach is selected for cervical decompression because the posterior approach could exacerbate the kyphosis and worsening the myelopathy.

Titanium box cage/bone graft was used for interbody fusion and to maintain the restored foraminal and disc space height and lordosis achieved at surgery. Anterior cervical disectomy with cage /bone graft interbody fusion was preferred for one or two level vertebral segment involvement and anterior medial corpectomy with cage fixation for three levels vertebral segment compression.

Posterior approach was used in patients with pathological compression encompassed more than three vertebral body segments and for those patients in whom the posterior canal compromised by thickened ligamentum flavum, overgrowth of facet joints. The technique used for posterior decompression was cervical laminectomy from C_3 to C_7 with or without lateral mass plating.

Radiological assessment

X ray cervical spine lateral view was taken in all patients. Standard lateral radiograph were taken in neutral position of the cervical spine with a tube distance of 140cm. The current in X- ray machine is kept constantly at 55 Kilovolts and 100mA.

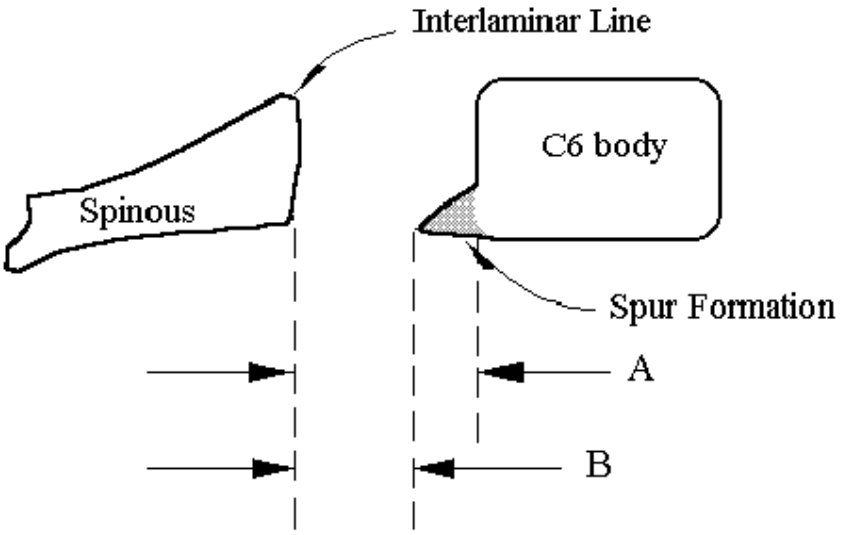
The distance between the posterior prominence of the posterior osteophytes to nearest point on spino laminar line is measured at each level of cervical vertebra and maximum level of compression is noted. The above effective canal diameter is again confirmed with sagittal MRI cervical spine.

MRI cervical spine, T_1 weighted, T_2 weighted, axial, sagittal cuts were done in all patients under study, the diagnosis is confirmed, the effective canal diameter at various cervical level, number of level of compression are studied. The exact details of the spinal cord, nerve roots, subarachnoid space and the soft tissue abnormalities (soft disc herniation, OPLL, thickened ligamentum flavum were assessed).

Increased signal intensity within the spinal cord on T_2 weighted images were studied. T_2 signal change, either ill-defined or well defined were noted. The patients were followed at a period of 1 months, 6 months

and 1 year duration and the outcome scale in compared with Nurick's grade.

Measurement of Effective Canal Diameter



A – Mid Sagittal Canal Diameter, B – Effective Canal Diameter

Statistical analysis

Parametric statistical analysis was performed using ANOVA test. All analysis were performed using 17.5 Version. Significance level was set at $p < 0.05$.

OUTCOME ANALYSIS

Outcome was categorized into three groups as :

1) Improvement (+1), 2) Stationary (0), 3) Deterioration (-1) depending on the preoperative and postoperative Nurick's grading.

The outcome were recorded as per the latest postoperative follow up.

RESULTS

I. Overall clinical results

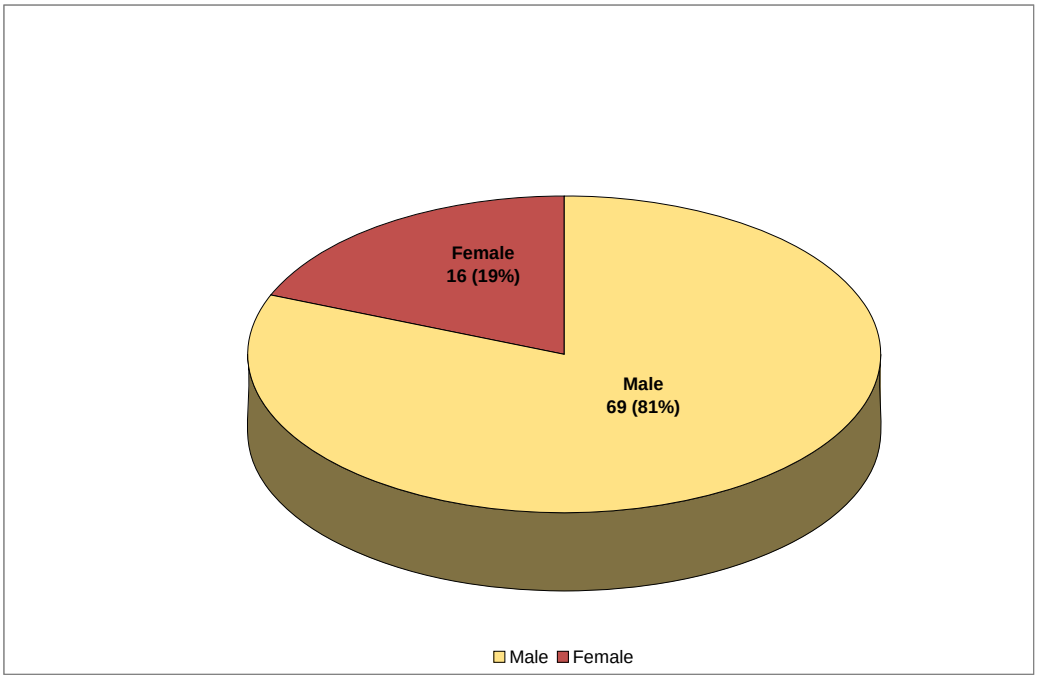
(i) Distribution of gender

Total number of patients under study : 85

Total number of males : 69

Total number of females : 16

Distribution of gender



(i) Age distribution

Of 85 patients, the age distribution of the patients is shown as below.

Age group	Number of Patients
< 40	23
40 – 60	45
> 60	17

The average age of the 85 patients was 49 ± 12 years.

(ii) Distribution of duration of symptoms in patients

Duration of symptoms	Number of patients
< 1 year	24
1 – 2 years	43
> 2 years	8

The range of duration of symptoms varies from 16.8 ± 7.2 months.

(iii) Distribution of preoperative neurological disability, evaluated by Nurick's grade

Preoperative Nurick's grade	Number of patients
0 – 2	34
3	38
4 – 5	13

The average preoperative Nurick's grade (Neurological disability) is 2.

(iv) Distribution of effective canal diameter

Effective Canal diameter	Number of patients
> 11 cm	19
9 – 11 cm	49
< 9 cm	17

The average effective canal diameter was 10.0 ± 1.3cm.

(v) Distribution of Number of levels of compression in patients

Number of level	Number of patients
1 level	20
2 levels	24
3 or more levels	41

The average lesional length was 2.4 ± 1.

(vi) Distribution of intramedullary hyperintense signal change in T_2 MRI

Intramedullary hyperintense signal change	Number of patients
No change	45
T_2 signal ill defined	16
T_2 signal well defined	24

(vii) Distribution of preoperative MIN Prognostic score

MIN Prognostic Score	Number of patients
6	2
7	6
8	5
9	7
10	6
11	8
12	4
13	6
14	8
15	6
16	4
17	10
18	3

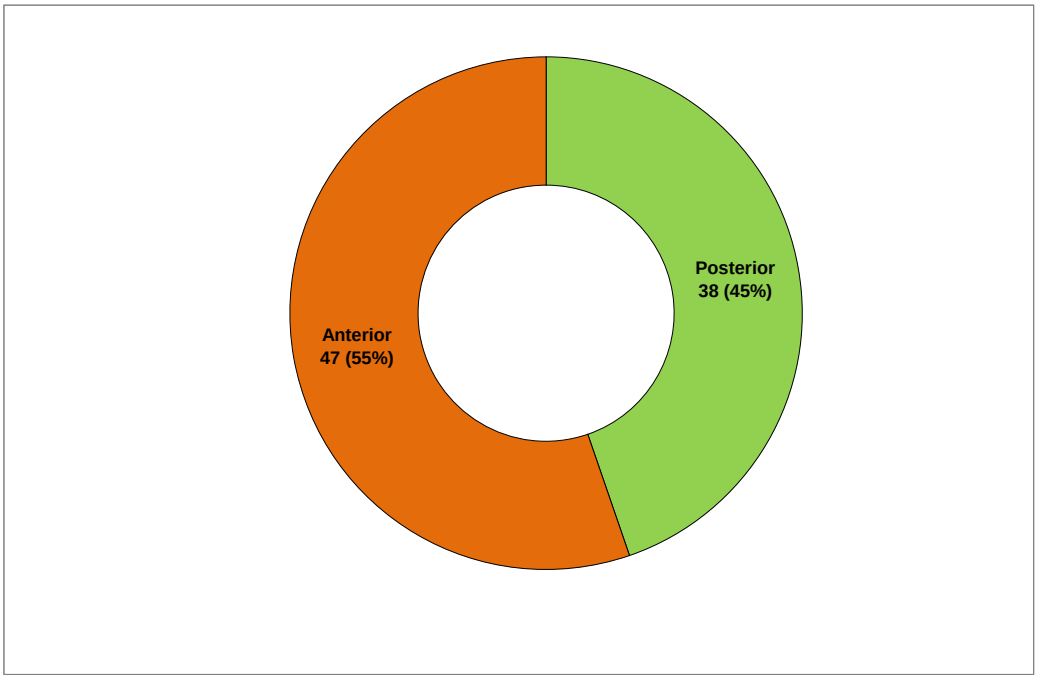
(viii) Distribution of Outcome

Outcome	Number of Patients
Improved (+1)	34
Stationary (0)	28
Deteriorated (-1)	23

(ix) Distribution of Surgical approaches

Surgical approach	Number of patients
Anterior	47
Posterior	38

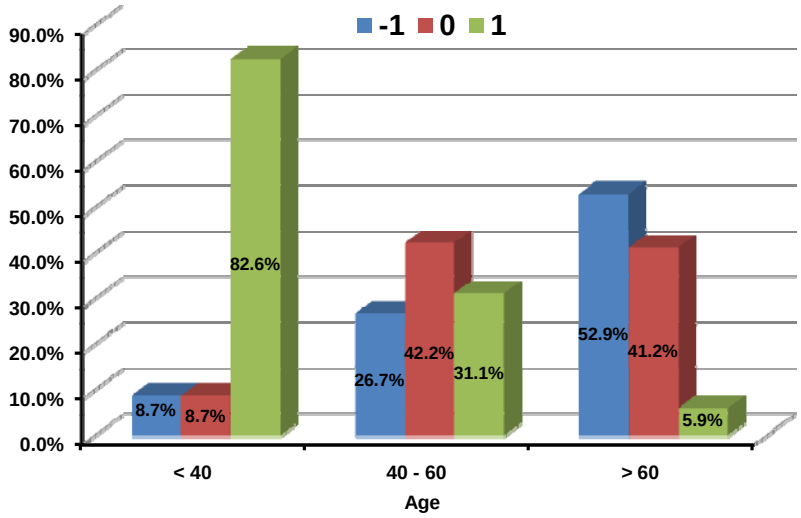
Distribution of surgical approach



CORRELATION OF VARIOUS FACTORS AND OUTCOME

AGE VS. OUTCOME

Age	-1	0	1	Total
< 40	2	2	19	**23**
40 - 60	12	19	14	**45**
> 60	9	7	1	**17**

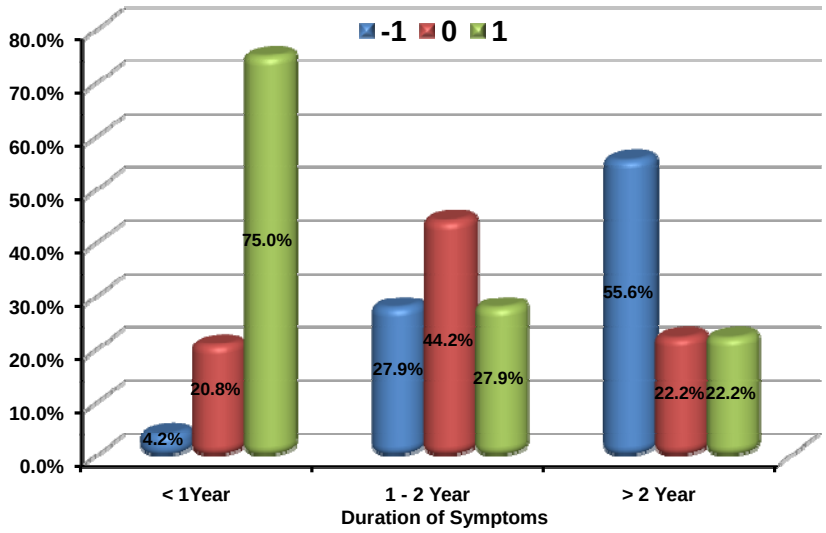


The average age was 49 ± 12 years. There was statistically significant difference in outcome between age groups.

(P-value – 0.000 < 0.05) (ANOVA test)

DURATION VS. OUTCOME

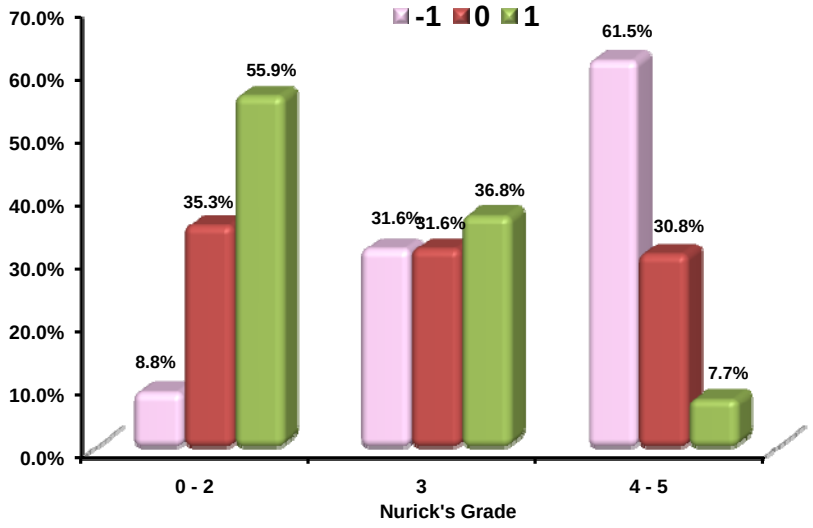
Duration	-1	0	1	Total
< 1Year	1	5	18	**24**
1 - 2 Year	12	19	12	**43**
> 2 Year	10	4	4	**18**



The range of duration of symptoms varies from 16.8 ± 7.2 Months. There was statistically significant difference in outcome between duration of symptoms (P-value – 0.000 < 0.05) (ANOVA test).

NURICK'S GRADE VS. OUTCOME

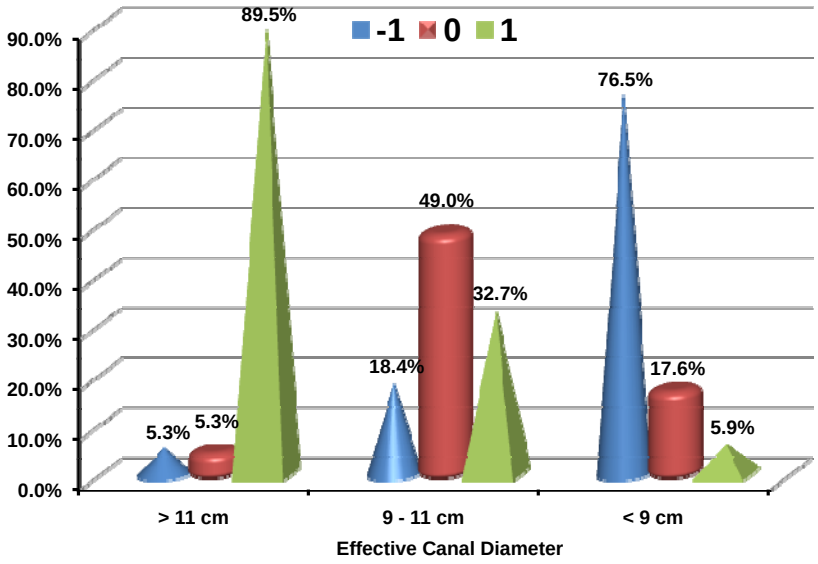
Nurick's Grade	-1	0	1	Total
0 - 2	3	12	19	**34**
3	12	12	14	**38**
4 - 5	8	4	1	**13**



The average Nurick's grade was 2.2 ± 0.7. There was statistically significant difference between Nurick's grade and outcome (P-value – 0.000 < 0.05) (ANOVA test)

EFFECTIVE CANAL DIAMETER VS. OUTCOME

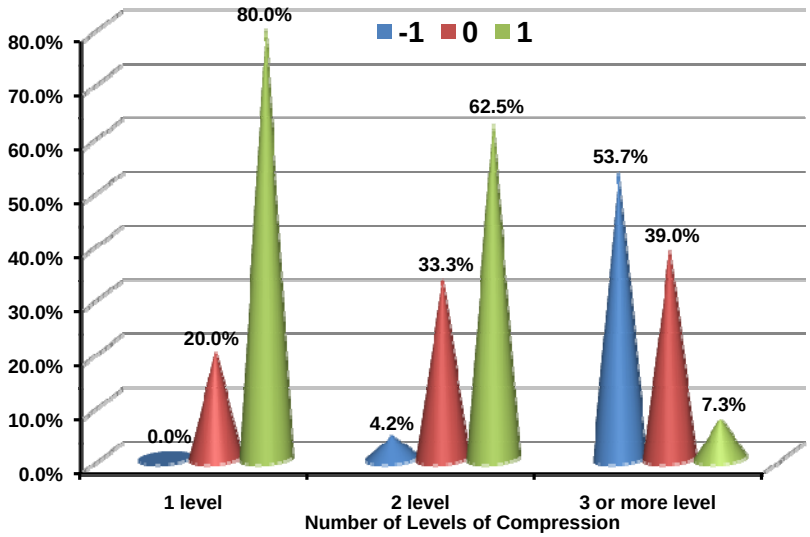
Canal Diameter	-1	0	1	Total
> 11 cm	1	1	17	**19**
9 - 11 cm	9	24	16	**49**
< 9 cm	13	3	1	**17**



The average effective canal diameter was 10.0 ± 1.3 cm. There was statistically significant difference between effective canal diameter and outcome (P-value – 0.000 < 0.05) (ANOVA test)

NUMBER OF LEVELS OF COMPRESSION VS. OUTCOME

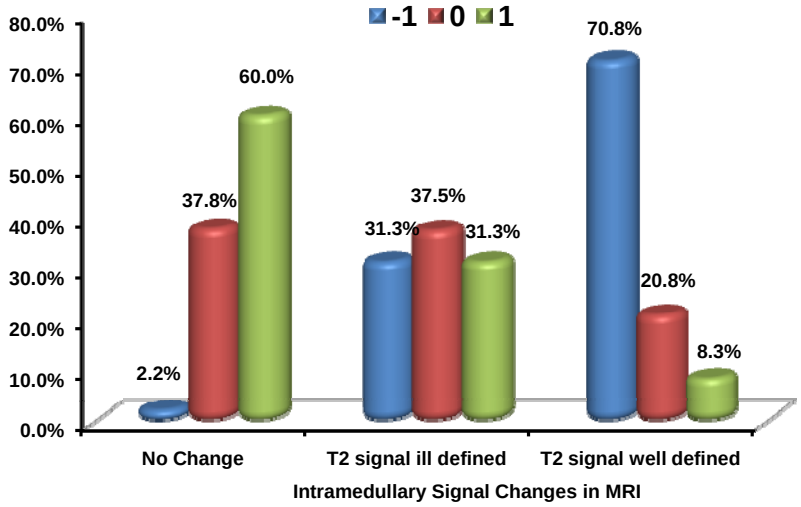
No. of levels	-1	0	1	Total
1 level	0	4	16	**20**
2 levels	1	8	15	**24**
3 or more levels	22	16	3	**41**



There was statistically significant difference in the outcome between the number of levels of compression (P-value – 0.000 < 0.05) (ANOVA test).

INTRAMEDULLARY SIGNAL CHANGES IN MRI VS. OUTCOME

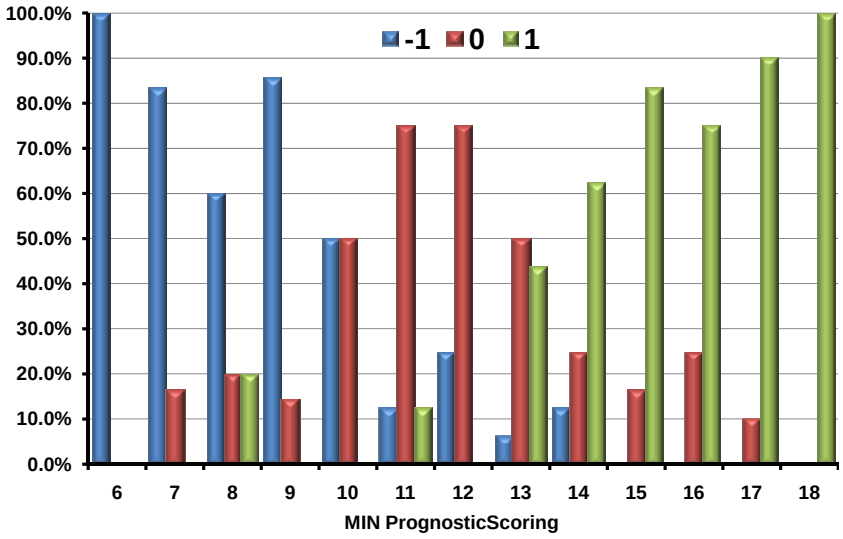
MRI Change	-1	0	1	Total
No Change	1	17	27	**45**
T_2 hyperintense signal ill defined	5	6	5	**16**
T_2 hyperintense signal well defined	17	5	2	**24**



There was statistically significant difference in outcome between group of patient with and without intramedullary Signal Changes in MRI and also the type of T_2 signal change (P-value – 0.000 < 0.05) (ANOVA test).

MIN PROGNOSTIC SCORING FOR CERVICAL SPONDYLOTIC MYELOPATHY AND OUTCOME

Prognostic Factors Total	Outcome			Total
	-1	0	1	
6	2	0	0	**2**
7	5	1	0	**6**
8	3	1	1	**5**
9	6	1	0	**7**
10	3	3	0	**6**
11	1	6	1	**8**
12	1	3	0	**4**
13	1	8	7	**16**
14	1	2	5	**8**
15	0	1	5	**6**
16	0	1	3	**4**
17	0	1	9	**10**
18	0	0	3	**3**
Total	**22**	**27**	**36**	**85**



There was stastically significant difference between MIN prognostic scores and outcome(P value-0.000<0.05)(ANOVA test)

DISCUSSION

The management of CSM is a challenge because of multifactorial pathogenesis and unpredictable response to treatment and variety of surgical approaches and philosophy in management. Though the factors like age, duration of symptoms, neurological status, radiological findings etc., are known to affect the outcome, there is no large comprehensive study in the available literature incorporating all the factors. In this study, a series of 85 cases of CSM treated surgically have been evaluated and the contribution of the individual factors to the outcome have been analysed.

An attempt have been made to formulate a comprehensive prognostic scale incorporating all these factors and evaluate the same.

1. AGE

CSM is a disease of middle age and elderly patients. It's rarely seen before the age of forty years. (Crandall and Bradzdorf 1966)[4]. In Naderi (1988)[69] study of 27 patients with CSM showed better neurological improvement in patients younger than 60years. Langston (2009)[57] in his study confirm age of the patient as one of the important prognostic factor. Jae sung (2010)[51] in his prospective study also

concluded, patients less than 40 years have better outcome. Fujiwara (1989)[50] and Kun (2005)[48] also confirms age as one of the prognostic factor in outcome. Fouyas (2002)[58] in a 10 years of prognostic study found age is the important prognostic factor in outcome.

In the present study of 85 patients, 23 patients were under age group of 40 years, 45 patients were in the age group of 40-60 years, and 17 patients in the age group above 60 years.19 patients out of 23 patients (82.6%) under 40 years showed improvement, out of 45 patients in age group 40-60 years, 19 had static outcome,14 improved and12 worsened, out of 17 patients above 60 years only one showed improvement 7 remained static and 9 patients worsened. This is accordance with other major studies, which conclude that age group below 40 years have better outcome than those above 60 years.

2. DURATION OF SYMPTOMS

Prolonged compression of the spinal cord will result in irreversible histological and physiological changes and loss of neuron within the spinal cord. The operative treatment is generally better in patients who undergo earlier decompression.

Suri (2003)[40] noted significant motor recovery in patients with duration of symptoms less than one year. Tanaka (1999)[41] recommended

decompression surgery even in patients more than 80 years old, provided duration of symptom is less than three years, the inability to walk has been present for less than three months, and the patient is physically able to undergo an operation. In another study, improvement in gait following laminoplasty was reported in 92% (eleven) of twelve patients in whom symptoms had been present less than eighteen months, as opposed to 77% (ten) of thirteen patients with a longer duration of symptoms before the operation (JBJS 2006).

In contrast, some authors have reported no correlation between the duration of preoperative symptoms and the clinical outcome following an operation. Arnasson et al (1987)[70] in a study of thirty-eight patients with cervical myelopathy who underwent surgery, the clinical outcome was not influenced by duration of preoperative symptoms.

In the present study of 85 patients, 24 patients had duration of symptoms less than one year, 43 patients had duration of symptoms one to two years and 18 patients had durations of symptoms more than two years. 18 out of 24 patients(75%) presenting within one year of duration showed improvement, 19 out of 43 patients having duration of symptoms one to two years had static outcome 12 improved and 12 worsened. 10 patients out of 18 (56%) presenting after two years worsened, 4 remained static and only 4 showed improvement. The present study has shown

good correlation between duration of symptoms and outcome, with duration of symptoms less than one year indicating good prognosis and duration of symptoms more than two years indicating poor outcome.

3. PREOPERATIVE NEUROLOGICAL STATUS (NURICK'S GRADE)

Langston(2009)[57] in his study showed preoperative Nurick's grade along with age, duration and preoperative sensory evoked potential is important prognostic factor, patients with Nurick's grade of two or less showed better outcome.

In this study of 85 patients, 34 patients presented with Nurick's grade 0-2, 38 patients with Nurick's grade 3 and 13 patients with Nurick's grade 4-5.19 of 34 (56%) patients presenting with Nurick's grade 0-2 showed improvement.8 out of 13 (62%) patients with Nurick's grade 4-5 worsened.

4. EFFECTIVE CANAL DIAMETER

The normal midsagittal canal diameter from C_3 to C_7 is 17 to 18mm. The patients with an osseous canal measuring <13 mm are considered developmentally stenotic. Static or dynamic translation between vertebral bodies may further decrease the available canal area and precipitate the development of myelopathy. Handa (2002)[47] in his

study showed that canal stenosis is a main prognostic factor in CSM. Kun (2005)[48] in an analysis of 13 factors in prognosis of the CSM preoperatively concluded effective canal diameter is an important prognostic factor. Kohno (1977)[48] showed better recovery in post laminoplasty patients when postoperative canal diameter is above 12mm. White and Panjabi (1988)[52], Fergusson (1985)[53], Asgari (1996)[55], Fager (1973)[56], Jae-sung (2010)[51] also noted effective canal diameter is one of the important prognostic factor and better prognosis is seen when the effective canal diameter is above 11mm.

In this study of 85 patients, 19 patients had effective canal diameter of above 11cm, 49 patients with effective canal diameter of 9-11 cm and 17 patients with effective canal diameter of less than 9 cm. 17 out of 19 patients (89.5%) with effective canal diameter more than 11cm showed improvement; 24 out of 49 patients with canal diameter 9 to 11 cm were stationary and 16 showed improvement. 13 out of 17 patients (76%) with effective canal diameter less than 9cm worsened. This is in agreement with other studies, confirming the role of effective canal diameter on the outcome in the CSM.

5. NUMER OF LEVELS OF COMPRESSION

The most common levels affected by CSM is C_{5-6} and C_{6-7} where most of the cervical flexion and extension occur and as stated earlier

osteophyte formation is accelerated by motion. The disease proceed typically contiguous and may proceed rostrally and or caudally.

Crandall and Batzdorf (1966)[4] showed that commonly in CSM two levels of involvement is seen. Fujiwara (1989)[50], Jae Sung Abu (2010)[51] in their study showed one or two levels of cervical cord compression showed better outcome than three or more levels of compression. But Fessler (1998)[34] in his study showed that extent of disease was not a negative predictor of clinical outcome.

In the present study of 85 patients, 20 patients had one level of compression, 24 patients had two levels of compression, 41 patients presented with three or more levels of compression. 16 out of 20 patients (80%) with a single level of compression improved. 15 out of 24 patients (62.5%) with two levels of compression improved and 8 out of 24 remained static. In the group of 41 patients with three or more levels of compressions, 22 patients (53.7%) worsened and 16 patients remained static. The present study have confirmed the finding that patients with single or two levels of compression have better outcome.

6. INTRAMEDULLARY SIGNAL CHANGE IN T_2 MRI

Magnetic resonance imaging with low signal abnormalities on T_1 weighted images and high signal abnormalities on T_2 weighted images

have been associated with greater clinical disability. Chi-Jen Chen (2001)[66] in a study of 64 patients, divided intramedullary high signal intensity on T_2 images into type 1 having a faint, fuzzy border and type 2 having a well defined border. In the analysis, type 2 lesion had a poor prognosis.

In the present study of 85 patients, 45 patients presented with no intramedullary signal change in T_2 weighted MRI, while 16 patients had ill defined hyperintense signal change in T_2 MRI and 24 patients had well defined hyperintense signal change in T_2 MRI. 27 of 45 (60%) patients with no intramedullary signal change in T_2 MRI improved and 17 remained stationary;wheras 17 of 24 (70%) with well defined hyperintense signal in T_2 MRI worsened. This again, emphasis the role of intramedullary hyperintense signal change in T_2 MRI in the outcome.

7. MIN PROGNOSTIC SCALE FOR CSM IN PREDICTING OUTCOME.

MIN prognostic scale for CSM has been devised incorporating all these prognostic factors

In this study 16 out of 20 patients (80%) with MIN prognostic score of 9 and less worsened; 20 out of 34 patients (59%) with the score between 10 and 13 were static; 25 of 31 patients (81%) with the score 14 and above improved. The MIN prognostic score has good correlation

with outcome. This is a retrospective study; a prospective study using the MIN prognostic scale for CSM is being undertaken to validate this scale further. This is a first instance, in the available literature, of a comprehensive prognostic scoring system in CSM.

CONCLUSION

A series of 85 cases of CSM have been analysed and the factors which contribute to outcome have been studied and the following conclusions have been drawn:

1. Major factors affecting outcome in CSM are age, duration of symptoms, neurological disability (Preoperative Nurick's grade), effective canal diameter, number of levels of compression, intramedullary hyperintense signal change in T_2 MRI,.

2. Patients with age less than 40 years, duration of symptoms less than one year, preoperative Nurick's grade 0-2, effective canal diameter more than 11cm, single level of compression, absence of hyperintense signal change in T_2 MRI are favorable factors in CSM.

3.Patients with age more than 60 years, duration of symptoms more than two years, Nurick's grade 4-5, effective canal diameter less than 9 cm, preoperative three or more levels of compression, presence of well defined hyperintense signal change in T_2 MRI are adverse prognostic factors in CSM.

4. The new prognostic scale (MIN prognostic scale for CSM) is a simple, comprehensive prognostic scoring system suitable for routine application and has shown good correlation with outcome. Majority of patients who have score of 9 and less worsen, majority of patients with score between 10 and 13 remain static and patients who have score 14 and above improve.

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PATIENT CONSENT FORM

STUDY TITLE:

Study centre : Department of Neurosurgery, MMC, Chennai – 600003.

Patient's name :

Patient's age :

Identification No:

Patient may check ( ) these boxes

I confirm that I have understood the purpose of this study. I have the opportunity to ask the questions and all my questions and doubts were answered to the best of my satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at anytime without my legal right being affected. ☐

I understand that sponsor of the clinical study. Other's working on the sponsor's behalf, the ethic's committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation n to it, even if I withdraw from the study. I agree to this access, however, I understand that my identity would not be revealed. In any information released to the third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully to cooperate with the study team, and to immediately inform inform the study staff if I suffer from any deterioration in my health or my well being or any expected or unusual symptoms. ☐

I hereby give consent to participate in this study. ☐

Signature/ Thumb impression of the patient:

Place:

Patient's name and address:

Signature of the investigator:

Name of the investigator:

Name : Age / Sex: IP No.: MIN No:

Address : Occupation :

D.O.A. : D.O.S.: D.O.D:

Main Complaints Duration :

1.

2.

3.

I. Weakness

1. Duration
2. Rate of Progression
3. Trauma
4. Spastic / Flail
5. Mode of Progression
6. Structure involved

II. Sensory

1. Pain
    a. Nature
    b. Site
    c. Radiation
    d. Aggravating / Relieving factor
2. Type of Sensory loss
3. Level of Sensory loss
4. Involvement of posterior column

III. Bowel and Bladder involvement

1. Duration
2. Type of bladder involvement
3. Constipation

IV. Any Cranial Nr involvement

V. Etiological factor

VI. Relevant Past History

VII. Personal History

VIII. General Examination

1. Neurocutaneous marker
2. Scoliosis / Kyposis
3. Neck / Ht Ratio

IX. Spinomotor examination

	UL		LL	
	RT.	Lt	Rt.	Lt
Bulk				
Tone				
Power 1. Shoulder 2. Elbow 3. Wrist 4. HIP 5. Knee 6. Ankle				

X. Sensory system Level

Touch
Pain
Temperature
Position
Vibration

XI. Reflex

DTR
Superficial reflex

XII. Respiratory function

1. Breath holding time
2. Single breath count
3. Chest expansion

XIII. Other Presentation

1. Central Canal Syndrome
2. Brown Sequard Syndrome

XIV. Clinically structured involved

XV. Clinically Highest level of cord compression

XVI. Cervical lordosis

XVII. X – ray

1. Canal diameter
2. Degenerative changes

XVIII. CT Cervical spine

XIX. MRI Cervical spine

1. Level of compression
2. No. of cervical segment involved
3. Myelomalacic changes
4. Ligamentum flavum hypertrophy
5. Anterior / Posterior cord compression.

XX Surgery

1. Approach
2. Time interval between onset of symptom and surgery
3. Immediate Post OP

4. Follow Up

XXI. Conservative Management

1. Type
2. Duration
3. Follow Up

XII. 1. Nurick's grading

MIN PROGNOSTIC SCALE FOR CERVICAL SPONDYLOTIC MYELOPATHY

PROGNOSTIC FACTORS	SUBDIVISIONS	SCORE
AGE	< 40	3
	40 – 60	2
	> 60	1
Duration of symptoms	< 1 Year	3
	1 – 2 Years	2
	> 2 Years	1
Neurological Disability (Nurick's Grade)	0 – 2	3
	3	2
	4 to 5	1
Effective canal diameter	> 11 cm	3
	9 – 11	2
	< 9 cm	1
Number of levels of compression	1 level	3
	2 levels	2
	3 or more levels	1
Intramedullary signal changes in MRI	No change	3
	T_2 signal ill defined	2
	T_2 signal well defined	1

Total score :

MASTER CHART

Cervical Spondylotic Compressive Myelopathy										Prognostic Factors							
Index	Age	Sex	Duration of Symptoms (Months)	No. of Segment involved	Anterior / Posterior	Mid Sagittal Canal diameter mm	MRI T2 Change	Pre op Nurick's	Post Op Nurick's	Age	Duration	No. of Level	Canal Diameter	MRI Change	Nuricks Grade	MIN Total	Outcome
1	60	M	14	2	Anterior	11.5	0	3	2	2	2	2	3	3	2	14	1
2	37	M	10	3	Posterior	10.5	0	3	4	3	3	1	2	3	2	14	-1
3	64	M	20	4	Posterior	8	2	4	5	1	2	1	1	1	1	7	-1
4	50	F	18	3	Posterior	9	0	3	3	2	2	1	2	3	2	12	0
5	38	F	10	1	Anterior	11.5	0	2	1	3	3	2	3	3	3	17	1
6	54	M	19	3	Posterior	8	2	3	4	2	2	1	1	1	2	9	-1
7	44	M	15	2	Anterior	10.5	0	3	3	2	2	2	2	3	2	13	0
8	63	M	25	4	Posterior	8	2	4	5	1	1	1	1	1	1	6	-1
9	61	M	25	3	Posterior	8	2	3	4	1	1	1	1	1	2	7	-1
10	60	M	18	3	Posterior	10.5	1	2	3	2	2	1	2	2	3	12	-1
11	37	M	18	4	Posterior	9.5	1	4	4	3	2	1	2	2	1	11	0
12	38	M	8	1	Anterior	11.5	0	2	1	3	3	3	3	3	3	18	1
13	45	F	20	1	Anterior	11	0	2	1	2	2	3	2	3	3	15	0
14	55	M	15	1	Anterior	10.5	0	3	3	2	2	3	2	3	2	14	0
15	47	M	30	4	Posterior	9.5	2	3	4	2	1	1	2	1	2	9	-1
16	65	F	11	1	Anterior	10.5	0	2	2	1	3	2	2	3	3	14	0
17	50	M	13	4	Posterior	7.5	2	3	4	2	2	1	1	1	2	9	-1
18	45	M	11	2	Anterior	9.5	0	4	4	2	3	2	2	3	1	13	0
19	44	M	14	4	Posterior	9.5	0	2	2	2	2	1	2	3	3	13	0
20	61	M	18	3	Anterior	10.5	2	2	3	1	2	1	2	1	3	10	-1
21	49	F	30	4	Posterior	8.5	2	4	5	2	1	1	1	1	1	7	-1
22	35	M	17	3	Posterior	11	1	3	4	3	2	1	3	2	2	13	-1
23	34	F	18	2	Anterior	9	0	3	2	3	2	2	2	3	2	13	1
24	36	M	11	1	Anterior	9.5	0	2	1	3	3	3	2	3	3	17	1
25	55	M	30	4	Posterior	7.5	2	4	4	2	1	1	1	1	1	7	0
26	28	M	11	2	Anterior	9	0	2	1	3	3	2	2	3	3	16	1
27	61	M	18	3	Anterior	8.5	0	2	2	1	2	1	1	3	3	11	0
28	29	M	11	3	Posterior	10.5	0	2	1	3	3	1	2	3	3	15	1
29	21	M	8	1	Anterior	11.5	0	2	1	3	3	3	3	3	3	18	1
30	16	M	10	3	Posterior	10.5	0	3	2	3	3	1	2	3	2	14	1
31	53	M	36	4	Posterior	10	1	3	3	2	1	1	2	2	2	10	0

MASTER CHART

32	43	F	11	2	Anterior	12.5	0	2	1	2	3	2	3	3	3	15	1
33	55	M	18	4	Posterior	9.5	2	4	4	2	2	1	2	1	2	10	0
34	65	M	15	4	Posterior	8	2	3	4	1	2	1	1	1	2	8	-1
35	55	M	18	4	Posterior	10.5	1	2	2	2	2	1	2	2	3	10	0
36	55	M	18	4	Posterior	8	2	4	5	2	2	1	1	1	1	8	-1
37	50	F	20	4	Posterior	7.5	2	4	4	2	2	1	1	1	1	8	0
38	68	M	14	3	Anterior	9.5	0	2	2	1	2	1	2	3	3	12	0
39	50	M	9	1	Anterior	9.5	0	2	1	2	1	3	2	3	3	14	1
40	65	F	16	3	Anterior	10.5	0	3	3	1	2	1	2	3	2	11	0
41	62	F	16	2	Anterior	11	0	2	2	1	2	2	2	3	3	13	0
42	48	M	18	2	Anterior	10.5	0	3	2	2	2	2	2	3	2	13	1
43	62	M	25	4	Posterior	8	2	4	5	1	1	1	1	1	1	6	-1
44	35	M	15	2	Anterior	11.5	1	2	1	3	2	2	3	2	3	15	1
45	35	M	9	1	Anterior	10.5	0	1	0	3	3	3	2	3	3	17	1
46	52	M	9	1	Anterior	13	0	2	1	2	3	3	3	3	3	17	1
47	37	M	15	3	Posterior	9.5	1	3	3	3	2	1	2	2	2	12	0
48	49	F	22	3	Posterior	10.5	1	3	3	2	2	1	2	2	2	11	0
49	45	M	9	1	Anterior	10.5	0	2	2	2	3	3	2	3	3	16	0
50	40	M	10	1	Anterior	11.5	0	2	2	2	3	3	3	3	3	17	0
51	60	M	25	3	Posterior	9.5	1	4	5	2	1	1	2	2	1	9	-1
52	70	M	25	3	Posterior	8.5	1	3	4	1	1	1	1	2	2	8	-1
53	73	M	20	3	Posterior	9.5	0	3	3	1	2	1	2	3	2	11	0
54	57	M	10	1	Anterior	12.5	0	2	0	2	3	3	3	3	3	17	2
55	44	M	8	1	Anterior	12.5	0	2	1	2	3	3	3	3	3	17	1
56	45	M	20	4	Posterior	9	2	3	4	2	2	1	2	1	2	10	-1
57	50	F	11	1	Anterior	13	0	2	1	2	3	3	3	3	3	17	1
58	40	M	18	2	Anterior	10.5	0	3	3	2	2	2	2	3	2	13	0
59	37	F	8	1	Anterior	11.5	0	2	1	3	3	3	3	3	3	18	1
60	34	M	15	2	Anterior	10.5	1	3	2	3	2	2	2	2	2	13	1
61	67	M	14	3	Posterior	9.5	2	3	4	1	2	1	2	1	2	9	-1
62	53	M	20	4	Posterior	8.5	2	3	4	2	2	1	1	1	2	9	-1
63	38	M	13	2	Anterior	9.5	1	3	2	3	2	2	2	2	2	13	1
64	60	M	15	2	Anterior	10.5	0	2	1	2	2	2	2	3	3	14	1
65	54	M	36	4	Posterior	9.5	2	3	3	2	1	1	2	1	2	9	0
66	44	M	42	4	Posterior	8.5	2	4	5	2	1	1	1	1	1	7	-1

MASTER CHART

67	39	M	13	2	Anterior	9.5	1	3	2	3	2	2	2	2	2	13	1
68	64	F	14	1	Anterior	11.5	0	3	1	1	2	3	3	3	2	14	2
69	41	M	9	1	Anterior	12.5	0	1	0	2	3	3	3	3	3	17	1
70	56	M	25	3	Posterior	11	1	3	3	2	1	1	2	3	2	11	0
71	72	M	18	2	Anterior	10.5	0	2	2	1	2	2	2	3	3	13	0
72	65	F	25	2	Anterior	9.5	2	2	3	1	1	2	2	1	3	10	-1
73	33	M	11	1	Anterior	11.5	0	3	1	3	3	3	3	3	2	17	2
74	41	M	19	2	Anterior	9.5	1	2	2	2	2	2	2	2	3	13	0
75	40	M	11	1	Anterior	10.5	0	3	2	2	3	3	2	3	2	15	1
76	54	M	18	2	Anterior	9	2	3	2	2	2	2	2	1	2	11	1
77	55	F	11	2	Posterior	10.5	2	2	2	2	3	2	2	1	3	13	0
78	57	M	36	4	Posterior	8.5	2	4	5	2	1	1	1	1	1	7	-1
79	60	M	18	3	Posterior	9.5	1	3	4	2	2	1	2	2	2	11	-1
80	48	M	11	2	Anterior	11.5	0	3	1	2	3	2	3	3	2	15	2
81	55	M	25	2	Anterior	8.5	2	4	3	2	1	2	1	1	1	8	1
82	26	M	18	3	Posterior	10.5	0	3	2	3	2	1	2	3	2	13	1
83	35	M	9	1	Anterior	12	0	2	1	3	1	3	3	3	3	16	1
84	34	M	15	2	Anterior	9.5	1	3	2	3	2	2	2	2	2	13	1
85	35	M	8	1	Anterior	11.5	0	2	1	3	1	3	3	3	3	16	1