A STUDY OF THE CLINICAL SPECTRUM AND FUNCTIONAL OUTCOME OF PATIENTS WITH NONTRAUMATIC MYELOPATHY

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF THE CLINICAL SPECTRUM AND FUNCTIONAL OUTCOME OF PATIENTS WITH NONTRAUMATIC MYELOPATHY"** is a bonafide record of work done by **Dr.D.ANUSHA** in the Institute of Neurology, Rajiv Gandhi Government General Hospital & **MADRAS MEDICAL COLLEGE, CHENNAI** in partial fulfillment of theTamilnadu Dr. MGR Medical University rules and regulations for the award of **D.M. (NEUROLOGY)** degree under my direct guidance and supervision during the academic year **2011-2014**.

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DECLARATION

I solemnly declare that this dissertation titled **"A STUDY OF THE CLINICAL SPECTRUM AND FUNCTIONAL OUTCOME OF PATIENTS WITH NONTRAUMATIC MYELOPATHY"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof. Dr. K. BHANU, Dip. NB., D.M.,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfillment of the university requirements for the award of the degree of D.M. Neurology.

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CONTENTS

ABBREVIATIONS

ATM	-	Acute Transverse Myelitis
AVM	-	Arterio Venous Malformation
BI	-	Barthel Index
CM	-	Compressive Myelopathy
CT	-	Computerised Tomography
CVJ	-	Cranio Vertebral Junction
CSF	-	Cerebro Spinal Fluid
CXR	-	Chest X- Ray
ECG	-	ElectroCardioGram
ELISA	-	Enzyme Linked ImmunoSorbent Assay
FIM	-	Functional Independent Mobiity
GCS	-	Glasgow Coma Scale
HSP	-	Heriditary Spastic Paraparesis
HIV	-	Human Immunodeficiency Virus
HTLV	-	Human T- cell Lymphotrophic Virus
IML	-	Interomediolateral
IMM	-	Interomediomedial
IVDP	-	InterVertebral Disc Prolapse
LETM	-	Longitudinally Extending Transverse Myelitis

LMN	-	Lower Motor Neuron
MANOVA	-	Multivariate Analysis of Variance
MRI	-	Magnetic Resonance Imaging
mRS	-	modified Rankin Scale
MS	-	Multiple Sclerosis
NCM	-	Non Compressive Myelopathy
NMO	-	Neuro Myelitis Optica
OPLL	-	Ossified Posterior Longitudinal Ligament
SACD	-	SubAcute Combined Degeneration
SOL	-	Space Occupying Lesion
TB	-	Tuberculosis
UMN	-	Upper Motor Neuron
VDRL	-	Venereal Disease Research Laboratory

Dedicated to my parents

Whose selfless sacrifice and constant motivation have provided me the impetus for all that I have achieved in my life

INTRODUCTION

The spinal cord is a vital and delicate structure of the central nervous system that is cushioned within the CSF, surrounded by the meningeal coverings, strong ligaments and encased within the protection of interlocking vertebral bones. Diseases of the spinal cord are termed as myelopathies, which can be secondary to trauma or may be due to non-traumatic causes. Nontraumatic myelopathies are of two types: compressive myelopathies and non-compressive myelopathies.

Myelopathies commonly present with motor and sensory deficits along with sphincter disturbances. The clinical presentation and causes of compressive myelopathies characteristically differ from those of noncompressive myelopathies, although rare presentations in either category can mimic each other[1] and pose a diagnostic dilemma to the astute clinician. The common causes of spinal cord compression are Pott's spine,fractures, infective abscess, arteriovenous malformations, spondylotic changes, spinal instability, tumours, multiple myeloma and metastases. The non-compressive myelopathies have wide and diverse etiologies like infective, inflammatory, demyelinating, vascular, hereditary causes or can be secondary to toxic exposure, metabolic disorders or nutritional deficiencies.

The management strategies between compressive and non-compressive myelopathies differ dramatically, as compressive lesions[2] usually require urgent neurosurgical intervention and decompression of the spinal cord, whereas non compressive myelopathies[3] are usually amenable to medical treatment itself.

Myelopathies usually present with devastating neurological consequences like para-/quadriparesis, neurogenic bladder, decubitus ulcers, spasticity, etc which can impair the quality of life and independence of the affected individual. The sequelae of spinal cord disorders are myriad, with few diseases like subacute combined degeneration showing dramatic response to treatment, producing only a mild impact on the patient's daily life, whereas some cases of acute transverse myelitis or cord compression can hamper the vital functions of mobility, sensation, bladder and bowel control, making the patient completely dependent on their caregivers. Little information regarding the functional outcome of nontraumatic myelopathies as a whole is available in the current literature, although the outcome of few specific myelopathies like cervical spondylotic myelopathy and acute transverse myelitis has been described.

REVIEW OF LITERATURE

Gross anatomy of spinal cord:

The spinal cord is a vital component of the central nervous system which is essential not only for the control of voluntary movements of the limbs and truncal musculature but also functions to receive the sensory information from these regions and relays them to the brain. It also controls the functioning of the viscera and the blood vessels of the thorax, abdomen and pelvis.

The spinal cord is a continuous nervous structure composed of gray and white matter and descends as continuation of the brain. The human spinal cord is composed of 8 cervical, 12 thoracic segments, 5 lumbar segments, 5 sacral segments and one coccygeal segment, thus having a total of 31 segments. These segments are described based on the pattern of spinal nerve origin from them. Usually one pair of spinal nerves emerges from each segment.

There are normally two levels of spinal cord enlargements: cervical (brachial) enlargement which extends from C5 to T1 and the lumbosacral enlargement that extends from L2 to S2. The caudal end of the spinal cord tapers down to form the conus medullaris.

The spinal cord is surrounded by cerebrospinal fluid and enclosed within the spinal meninges. The spinal meninges are the pia mater, arachnoid mater, and dura mater. External to the dura matter is the epidural space, which is filled with fat and lymphatic tissue, arterial vasculature and a venous plexus. The pia mater is tethered to the arachnoid and dura on each side of the spinal cord, between the ventral and dorsal spinal roots of each vertebral level, by a serrated tooth-like fibrous extension of the pia called the denticulate ligament.

The spinal cord receives its blood supply from a single anterior spinal artery and two posterior spinal arteries. The anterior spinal artery originates from the vertebral artery whereas the posterior spinal arteries originate either from the vertebral artery or its posterior inferior cerebellar branch. The veins of the spinal cord form a surface plexus that drains superiorly into the cerebellar veins and cranial venous sinuses and through the intervertebral veins and external venous plexuses into the azygos system.

The typical cross-section of the spinal cord shows a centrally located gray matter with peripherally oriented white matter tracts. A central canal which is a remnant of the embryological ventricular system **runs throughout the length of**

spinal cord and communicates with the fourth ventricle at the cranial end and with the terminal ventricle in the conus medullaris caudally.

Internal architecture of spinal cord:

Gray matter of the spinal cord:

The spinal cord gray matter is a ‘H’-shaped complex structure composed of neuronal cell bodies, dendrites, axons, and neuroglial cells. The gray matter is grossly divided into dorsal and ventral horns with an intermediate zone between them. The Rexed lamina, determined on the basis of cytoarchitecture seen microscopically, divides the spinal gray matter into 10 regions. The first nine laminae are arranged from dorsal to ventral whereas the tenth lamina is the group of cells surrounding the central canal. Laminae 1-4 comprise the main cutaneous receptive regions; lamina 5 receives its afferents from the viscera, skin and muscles whereas lamina 6 mainly receives proprioceptive and some cutaneous afferents. Lamina 7 neurons are responsible for the regulation of posture and movement. Lamina 8 contains cells which are the propriospinal interneurons. Lamina 9 contains clusters of large alpha motor neurons that supply the extrafusal fibers of the striated muscles involved in the movements of the axial skeleton and the limbs, along with gamma motor

neurons innervating the intrafusal fibers present in muscle spindles. Lamina 10 is the area surrounding the central canal.

Lateral spinal nucleus

This is composed of a group of cells that lie ventral to the dorsolateral tip of the dorsal horn and is considered to project the received sensory information to themidbrain, thalamus, and hypothalamus.

Lateral cervical nucleus

It is a sensory nucleus lying lateral to the lateral spinal nucleus in upper cervical levels which projects to the cerebellum, midbrain, and thalamus.

Onuf's nucleus

This is a distinct group of motor neurons in the caudal lumbosacral spinal cord seen in the ventrolateral portion of the ventral horn of the spinal cord. It supplies the perineal muscles and the anal and urethral sphincters.

White matter of spinal cord

The white matter of the spinal cord surrounds the gray matter all around except at the region where the dorsal horn touches the margin of the spinal cord. Although the white matter consists mostly of

longitudinally running axons, it also contains glial cells. A large group of axons which are located in a given area is called a funiculus. Small bundles of axons that share common features within a funiculus are called as fasciculus. A group of nerve fibers having the same origin, course, termination and function is called a tract. The horns of gray matter divide the white matter into three columns or funiculi: dorsal, lateral and ventral.

The dorsal column of white matter is primarily made up of the central processes of dorsal root ganglion cells. It is these large myelinated axons that form the main pathway conveying skin sensation as well as position sense (proprioception) from the limbs and trunk to the brain.

The lateral and anterior columns contain the various ascending and descending fiber groups. The ascending tracts include the spinothalamic, the dorsal and ventral spinocerebellar, spino-olivary, spinotectal, spinoreticular, spinocervical, and spinovestibular tracts while the descending tracts include the corticospinal, vestibulospinal, reticulospinal and tectospinal tracts. There are propriospinal fibres also, that often lies very close to the gray matter and connects one spinal cord segment with another. The largest propriospinal pathways connect the brachial and

lumbosacral enlargements which help in the coordination of limb movements.

The sympathetic fibers are dispersed within the spinal cord and ultimately project to the interomediomedial (IMM) and interomediolateral (IML) neurons which lie within lamina VII extending from the T1 to L3 cord levels of the thoracolumbar cord.

Thus, it is this complex architecture of the spinal cord and its tracts that results in the myriad of neurological manifestations in spinal cord diseases.

Spinalcord syndromes:

The various manifestations in spinal cord diseases depend on the type of involvement which can be:

1. Anterior cord syndrome
    i. Preservation of proprioception
    ii. Preservation of vibratory perception
    iii. Diminished or loss of pain and temperature sensation below lesion
    iv. Complete or incomplete motor loss
2. Posterior cord syndrome
    i. Impaired vibration sense

ii. Abnormal position sense
iii. Loss of deep pressure perception
iv. Diminished tactile localization
v. Sensory gait ataxia
vi. Tactile and postural hallucinations
vii. Spared pain and temperature perception

- *Posterolateral cord syndrome*

i. Distal extremity paresthesia
ii. Sensory ataxia
iii. Hyperreflexia
iv. Muscular spasticity
v. Bilateral toe extensor signs (Babinski's)
vi. Impaired proprioception and vibratory sense
vii. Sparing of pain and temperature sensibility

3. Central cord syndrome

i. Bandlike thermoanesthesia or thermodysesthesia
ii. Bandlike analgesia or hypoalgesia
iii. Preservation of light touch
iv. Dissociated sensory loss

4. Anterior horn syndrome

- *Polio and post-polio syndrome*
    i. Diffuse weakness (LMN)
    ii. Muscular atrophy
    iii. Reduced muscle tone
    iv. Muscle fasciculations
    v. Hyporeflexia or areflexia
- *Combined anterior horn cell and pyramidal tract disease (motor neuron disease)*
    i. UMN: Spastic paresis, Extensor plantar responses, Hyperreflexia
    ii. LMN: Muscular atrophy, Flaccid paresis, Fasciculations

5. Vascular syndromes
    - *Anterior spinal artery syndrome*
        i. Radicular and ascending leg pain
        ii. Sensory level for pain and temperature
        iii. Sudden progressive paraplegia
        iv. Flaccidity and areflexia (acute)
        v. Spasticity and hyperreflexia with Babinski's sign (late)
        vi. Sparing of touch, vibration, and temperature

vii. Urinary and fecal incontinence (uncommon)

- *Posterior spinal artery syndrome*

i. Suspended global anesthesia

ii. Regional tendon and cutaneous reflex loss

iii. Dorsal column sensory level

iv. Sparing of anterior cord functions

- *Radiculo-medullary syndrome*
- *Central cord vascular syndrome*

6. Hemisection syndrome (Brown Sequard syndrome)

i. Ipsilateral loss of vibratory perception

ii. Segmental lower motor neuron signs at the level of the lesion

iii. Ipsilateral loss of proprioception (position sense) (below level of lesion)

iv. Contralateral loss of pain and temperature sensibility (one or two segments below level of lesions)

v. Ipsilateral motor loss with spastic paresis

vi. Inability to walk

vii. Loss of normal bowel and bladder function

7. Complete spinal cord transection (transverse myelopathy)

It results in complete interruption of all ascending and descending tracts at the level of the lesion and leads to the loss of motor, sensory, autonomic, and reflex functions below that level.

8. Conus medullaris syndrome
    i. Loss of bladder control
    ii. Loss of perianal muscle control
    iii. Absent bulbocavernosis reflex
    iv. Absent anal wink reflex
    v. Flaccid paresis of lower extremity
9. Cervical medullary syndrome
    i. Respiratory insufficiency or arrest
    ii. Arterial hypotension
    iii. Varying degrees of tetraparesis
    iv. Facial sensory loss
    v. Greater arm than leg weakness
10. Multifocal cord syndrome

Compressive versus noncompressive myelopathy:

The common clinical presentation in a compressive myelopathy is:

- Pain which can manifest as root pain (radicular), vertebral pain, or funicular pain (central)
- Asymmetric motor or sensory deficits
- Ellsberg phenomenon (in case of cervical level lesions)

There are three clinical stages of spinal cord compression:

- radicular pain and segmental motor and sensory disruption
- incomplete transection
- complete cord transection.

Compressive myelopathies are further divided into:

1. Intramedullary compression

   Usually manifest with funicular pain, late UMN involvement, prominent and diffuse LMN signs, trophic changes, descending progression of paraesthesias, and early sphincter involvement.

   The common causes are: intramedullary neoplasms[4], hematomyelia and syringomyelia.

2. Extramedullary compression

   Usually present with radicular and vertebral pain, early UMN features, rarely LMN signs in segmental

distribution, ascending sensory signs, and late bladder involvement.

They are further classified as:

a. *Intradural*

The common causes are spondylosis [5], spondylolisthesis, facet joint arthropathies, ligamentum flavum hypertrophy, congenital spinal canal stenosis[6], degenerative osteophytosis, intervertebral disc prolapse or bulge, nerve sheath tumors and meningiomas.

b. *Extradural*

Usually associated with prominent spinal tenderness.

The common causes are: primary bone neoplasms, metastatic bone deposits and infections like Pott's spine, epidural abscess, etc.

Non compressive myelopathies are due to:

1. Vascular [7]
2. Infectious[8]
    a. Tropical spastic paraparesis (HTLV1 associated)
    b. HIV related vacuolar myelopathy
    c. Herpes related myelopathy

d. Syphilitic myelitis

e. Viral myelitis including poliomyelitis, etc.

3. Toxic

a. Lathyrism

b. Konzo [9]

c. Nitrous oxide toxicity

4. Metabolic and nutritional

a. Vitamin B12 deficiency[10]

b. Folate deficiency

c. Copper deficiency

5. Secondary to inflammatory disorders

a. Systemic lupus erythematosus, Behçet's disease, Sjogren's syndrome, and sarcoidosis[11]

6. Para-/post infectious

7. Paraneoplastic[12]

8. Primary demyelinating disorders

9. Radiation induced and

10. Idiopathic causes

The spectrum of nontraumatic myelopathies encompasses the above mentioned etiologies of myelopathies and can present as an acute onset illness, subacute course or as a chronic and progressive disease.

In a prospective Indian study, Chaurasia et al[13] described the etiological spectrum of 204 non-traumatic myelopathy patients presenting to a tertiary care hospital and noted that 61.7% of the cases had compressive etiology. Tuberculosis of spine was the most common cause of compressive myelopathy, followed by cervical spondylosis, whereas acute transverse myelitis and SACD were the common etiologies in the non-compressive group.

Another Indian study on the MRI based diagnostic profile of compressive myelopathies described by Yadav et al[14] showed that spinal tuberculosis was the commonest cause (24.6%) followed by spinal metastases (17.4%) and ossified posterior longitudinal ligament (7.8%).

Prabhakar et al [15] had described the clinical and radiological profile of non-compressive myelopathy in fifty seven patients in which acute transverse myelitis (ATM) was noted to be the commonest cause. It was followed in frequency, by subacute combined degeneration secondary to vitamin B12 deficiency and primary progressive multiple sclerosis, in descending order.

Das et al[16] described a study on the profile of noncompressive myelopathy in eastern India where the presentation was acute in 48.78% patients, subacute in 8.53%, chronic in 32.92%. History of relapse and remission were seen in 9.75% patients. Etiological diagnosis could be established in 71.95% of the cases whereas no aetiological factors could be found in the remaining patients.

Alvarenga et al[17] described the clinical course of 70 patients with noncompressive acute transverse myelitis, and noted that 59% of the cases were idiopathic, and those cases were also noted to have a favourable prognosis during long term neurological evaluation.

Looti et al[18] described 147 cases of nontraumatic myelopathies, determined on the basis of myelographic findings only, in a hospital based study in Cameroon and noted that majority of the cases were of compressive etiology, with metastases and infections like tuberculosis being the leading causes of compression.

Owolabi et al [19] studied the profile and outcome of 98 Nigerian patients with non-traumatic paraplegia and noted that lower limb weakness was the commonest symptom and present in 100% of the cases. 55 % had sensory deficits and another 55% manifested with sphincteric disturbances. 50% of cases had radicular pain and paresthesia was present

in 38.4%.The commonest etiological factors were tuberculosis, transverse myelitis and metastatic spinal disease.

Interesting, in a study by Modi et al[20], describing hundred consecutive myelopathy cases in Africa, nearly half of the study population were found to be HIV positive, indicating the high prevalence of retroviral disease in that region as well as its contribution to the spectrum of spinal cord diseases occurring in that area.

Moore et al [21] has described the causes of nontraumatic paraparesis and tetraparesis in a prospective study on 585 patients and noted that cervical spondylotic myelopathy was the most common cause, followed by extrinsic neoplastic or developmental tumour and multiple sclerosis respectively.

In a hospital based survey on nontraumatic paraparesis, described by Watson[22], the commonest etiology was disseminated sclerosis, followed by tumors and vascular lesions. These cases of nontraumatic paraplegia contributed to 30% of the new admissions to the spinal injuries unit.

de Seze et al[23] described the etiological and outcome profiles in 76 patients with acute transverse myelitis and found that 43% were due to multiple sclerosis, 16.5% due to systemic diseases, 14% due to spinal

cord infarcts, 6% due to parainfectious myelopathy, and 4% due to delayed radiation myelopathy. 16.5% patients had myelopathy for etiology could not be determined. Clinical outcome determined at 1 year showed good response in 88% of multiple sclerosis cases and poor outcome in 91% of spinal cord infarcts and 77% of systemic diseases.

Cordonnier et al[24] prospectively studied 55 patients with acute partial transverse myelitis and found that sensory symptoms, oligoclonal bands and brain MRI were factors that were predictive of the future conversion to clinically definite multiple sclerosis.

In a retrospective study on 53 patients presenting with first episode of acute transverse myelitis described by Gajofatto et al [25], he noted that 79% of patients were found to convert to multiple sclerosis on followup. The predictors of conversion in his study were the absence of a sensory level, absent bladder disturbances, neuroimaging abnormalities in the brain on MRI, involvement of spinal cord fewer than 3 vertebral segments, and abnormal somatosensory evoked potentials.

A retrospective case-series of patients of acute transverse myelitis presenting to a university hospital in Pakisthan over a period of 14 years was reported by Kahloon et al[26] where he noted that 60 % of the cases were idiopathic, 30% were parainfectious with maximum cases

demonstrating a thoracic sensory level. More than 90% of cases presented with paraparesis and bladder dysfunction, and one fourth of the cases were found to be quadriparetic.

Although vacuolar myelopathy is the most frequent cause of HIV associated paraparesis in individuals from developed countries, the spectrum of myelopathy associated with HIV differs widely in developing countries. Bhigjee et al [27] prospectively studied the spectrum of myelopathies in HIV seropositive South African patients and noted that only one of the thirty three patients had vacuolar myelopathy while the 36% of the patients had co-infection with HTLV-I, 18% had tuberculosis, 9% had zoster myelitis, and 6% had herpes simplex. This study showed that infective etiologies co-existing in HIV seropositive individuals was the most common cause of myelopathy in developing countries of Africa.

A considerable number of patients with clinical picture of compressive myelopathy have a normal MRI which confounds the treating doctor. One of the surgically treatable causes of compressive myelopathy is spinal arteriovenous malformations which may have no neuroimaging abnormalities except for subtle flow voids or increased T2 signal changes.

Strom et al [28] reviewed 78 patients with unexplained myelopathy who had undergone spinal angiography in his institution and found spinal AVM as the cause of myelopathy in 22 patients (28.2%). Thus, spinal angiography is an important investigation that needs to be performed in patients with unexplained myelopathy to rule out MRI negative AVMs.

Another interesting nontraumatic myelopathy frequently being reported in recent times is the surfer's myelopathy which is a form of nontraumatic spinal cord injury, exact pathophysiology of which is unclear but is probably secondary to ischemic insult to the cord due to dynamic compression, vasospasm,or thrombotic infarction of the spinal cord vasculature.

Chang et al [29] has described the clinical characteristics of a large case series of surfer's myelopathy which is typically associated with young age, inexperience in surfing, hyperextension of lumbar spine, absence of trauma, and is clinically characterized by progressive paresthesias and weakness following a prodrome of backache.

Tropical spastic paraparesis is a myeloneuropathy of noncompressive etiology being reported in the recent literature and widely associated with HTLV1 infection. One Indian study in Kerala by Oomman et al [30], however interesting noted that only one patient out of

the twenty five patients of tropical spastic paraparesis was HTLV1 positive by serology. The authors had postulated that their case series could be representative of the previously described entity "seronegative spinal spastic paraparesis".

In a prospective study by Mckinley et al[31] describing the epidemiology and functional outcome in nontraumatic myelopathy patients with quadriplegic and incomplete nontraumatic myelopathies had shorter in hospital length of stayas well as lower motor functional independent mobility (FIM) scoresat discharge and less change in FIM scores at follow-up.

Cobo Calvo et al [32] studied the outcome of eighty seven patients with acute transverse myelitis and found that 13% of his patients with definite and possible idiopathic ATM converted to multiple sclerosis. He also noted that patients with bladder dysfunction at admission or longitudinally extending spinal cord lesions on MRI had a poor functional recovery on followup.

Christensen et al[33] studied the clinical features and long term outcome in acute transverse myelopathy and noted that thoracic myelopathy was the most common location. He also noted that while one third of the patients had a good outcome, another one- third had poor

outcome with paraplegia, incontinence and severe sensory deficits, with the rest remaining static in their neurological status.

Non traumatic spinal cord lesions are also associated with frequent medical complications during in- hospital rehabilitative care [34], with both disability and medical complications negatively affecting each other.

A retrospective study on the histological diagnosis of 110 spinal cord lesions described in Pakisthan [35] showed tuberculosis as the leading cause of spinal cord pathologies followed by schwanommas.

Debette et al[36] followed up 170 consecutive patients presenting with a first episode of acute and subacute noncompressive myelopathy which were defined by a symptom onset of less than 3 weeks and duration of more than 48 hours. The outcome beyond 2 years of initial diagnosis was assessed in which the death rate was noted to be8.8 %. The functional outcome was unfavourable in those patients who had initially severe symptoms, centrally located lesions on MRI, and when the etiology of disease was neuromyelitis optica or systemic diseases. It was noted that one-third of the patient's initial diagnosis differed from the final diagnosis at the end of 2 years of follow- up. More than 50% of the patients who had undetermined etiology were found to have multiple sclerosis on followup.

Cervical compressive myelopathy is the often encountered compressive myelopathy which is amenable to surgical management. The various techniques employed for management of cervical myelopathy [37] are the posterior cervical techniques like laminectomy or laminoplasty and anterior surgical techniques like anterior cervical discectomy or anterior cervical corpectemy.

Postoperative functional outcome in microsurgically treated intramedullary spinal cord compressive lesions were described by Ebner et al[38] who noted that extended intramedullary lesions and poor pre-operative neurological status were determinants of outcome in those cases.

Schiff et al[39] described the treatment outcome of forty cases of intramedullary spinal cord metastases, in which 35 patients had undergone radiotherapy and 5 underwent surgery. Only eleven patients had survived beyond 6 months. The median survival was noted to be 4 months in patients receiving radiotherapy and 2 months for patients not receiving radiotherapy.

Putten et al[40] studied the factors affecting functional outcome after in-patient rehabilitation in patients with nontraumatic myelopathies and found that patients with lower disability scores at admisison, early onset

of rehabilitation and longer length of stay were associated with better functional outcomes.

It is interesting to note that the etiologic spectrum of nontraumatic myelopathies varies among different populations and is epidemiologically different in the various regions of the world. Data from the developing countries show that maximum cases are related to degenerative age related changes as well as malignancies in the compressive myelopathies and demyelinating diseases as the common causes in noncompressive myelopathies. However, literature from the developing countries as well as those from India shows that infections, especially tuberculosis of the spine contribute to the major bulk of compressive myelopathies, but demyelinating diseases still continue to be the commonest etiology in noncompressive myelopathies.

AIM OF THE STUDY

1. To evaluate the clinical manifestations and aetiological profile of patients with nontraumatic myelopathy
2. To study the functional status of patients with nontraumatic myelopathy at presentation and after 6 months

MATERIALS AND METHODS

Study subjects:

The study was conducted on patients attending the Neurology services at the Institute of Neurology, Madras Medical College & Rajiv Government General Hospital, Chennaifrom November 2012 to July 2013. The approval from the Institutional Ethical Committee and informed consent of the patients participating in the study were obtained.

Inclusion criteria:

Hundred patients with non-traumatic myelopathy attending the Neurology services of Rajiv Gandhi Government General Hospital, Chennai were included in the study.

Exclusion criteria:

Patients with obvious trauma associated with the myelopathy or radiological evidence of traumatic etiology were excluded from the study as the management and outcome in these patients is affected by several in-hospital factors.

Methodology:

Each patient included in the study was methodically evaluated by obtaining a detailed history and performing complete neurological examination at presentation. This was recorded on a predesigned and structured proforma along with the routine investigations done as per standard protocol of the unit.

Blood investigations which were done in all patients included complete hemogram, blood urea, serum creatinine, plasma glucose, liver function tests, CXR, ECG, HIV and VDRL.Nerve conduction studies, CSF analysis for cell count, cytology, protein, sugar as well as oligoclonal bands (in relevant cases), serum vitamin B12 levels (in relevant cases)and rheumatological investigations were done in all cases manifesting as noncompressive myelopathy.

Magnetic Resonance Imaging (MRI) of the spine with or without contrast and brain and whole spine screening was done using 1.5 Tesla MRI on all patients with focus primarily on the site of spinal cord lesion localized clinically. The sequences used were: T1 and T2 sagittal, and T2 axial and post gadolinium T1 contrast sections. CT of the spine was done in cases of suspected OPLL.

The enrolled patients were clinically and radiologically classified as compressive or non-compressive myelopathy. Details regarding the treatment given, including medical and surgical management done during the hospital stay was also noted.

Functional status of the patient using the Barthel index[41] and modified Rankin scale[42] was assessed at admission and at 6 months and recorded on the proforma with time-tolerance of a limit of +/-1 month on the day of assessment.

The Barthel index (BI) is a 10- point scoring system used to assess the degree of independence of the individual with regards to activities of daily living. The items are divided into 10 items that focus on patient's ability at self-care (feeding, grooming, bathing, dressing, bowel and bladder care, and toilet use) and mobility (ambulation, transfers, and stair climbing). The patient's performance is judged either by direct observation of his performance or by asking the patient or caregiver's regarding his degree of functional independence. The total score ranges from 0 to 100, with higher scores indicating greater functional independence. The lowest score is 0 which represents a totally dependent and bedridden state.

The modified Rankin scale (mRS) is another scale primarily used to assess the outcome of stroke patients. The mRSscale measures gross independence of the individual instead of assessing the performance of specific tasks.It incorporates assessment of both the mental and physical adaptations of the individuals to their neurological deficits. The scale consists of scores ranging from 0 to 6, with 0 corresponding to no symptoms, 5 corresponding to severe disability and 6 to dead status.

Statistical considerations:

The entire data gathered on the patients with nontraumatic myelopathy was tabulated and analysed using statistical software SPSS version 17.0. Chi square testanalysis was used for comparison between the two groups of compressive and noncompressive myelopathy. Multivariate Analysis of variance model (MANOVA) using generalized linear models approach was used to assess the significance of association of clinical and radiological factors in patients with nontraumatic myelopathy with the initial functional status at presentation and 6- month functional outcome (determined using Barthel index and mRS). All ‘p’-value less than 0.05 were considered statistically significant.

RESULTS

Hundred patients with nontraumatic myelopathy were included in the study. Of the hundred cases, forty eight patients (48%) were found to have non compressive myelopathy and fifty two (52%) patients had compressive myelopathy.

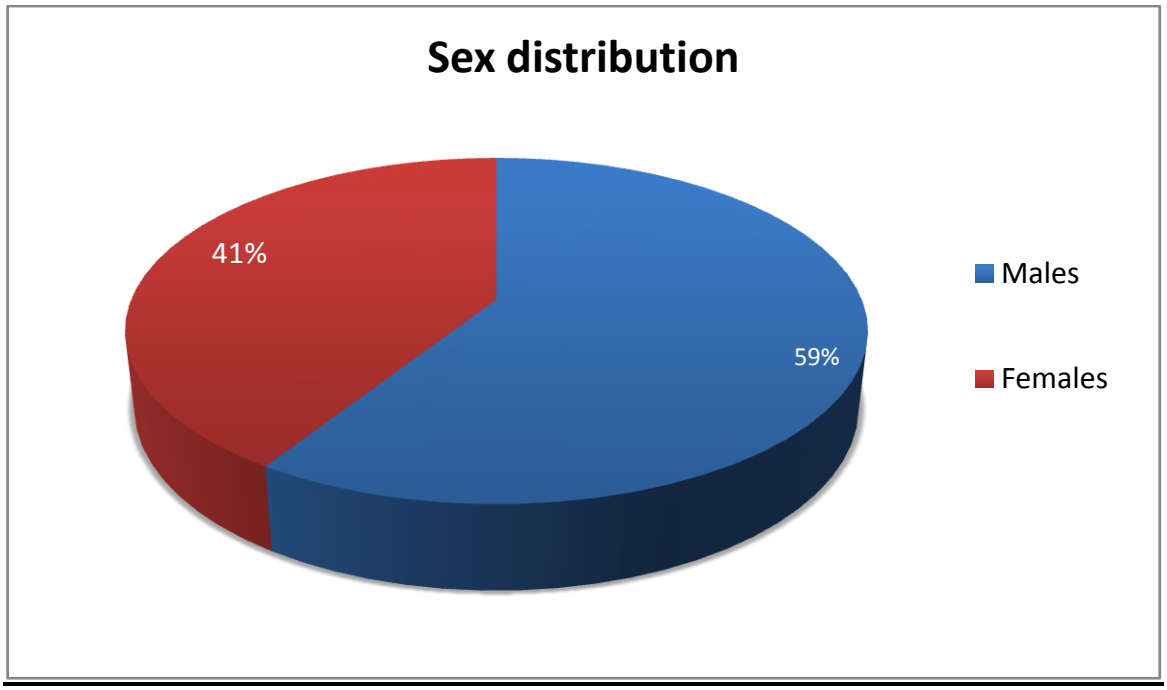
Demographic profilein nontraumatic myelopathy

The mean age of the hundred patients with nontraumatic myelopathy was 40.78 ± 16.56 years with age ranging from 14 to 70 years. Of them, 59 % were males and the rest 41% were females. All patients belonged to a low socioeconomic status.

Table 1: Age distribution in nontraumatic myelopathy

Nontraumatic Myelopathy	Frequency	Percent	Valid Percent	Cumulative Percent
11-20 years	18	18.0	18.0	18.0
21-30 years	13	13.0	13.0	31.0
31-40 years	16	16.0	16.0	47.0
41-50 years	21	21.0	21.0	68.0
51-60 years	20	20.0	20.0	88.0
61-70 years	12	12.0	12.0	100.0
Total	100	100.0	100.0	

Fig 1: Sex distribution in nontraumatic myelopathy



Clinical characteristics in nontraumatic myelopathy

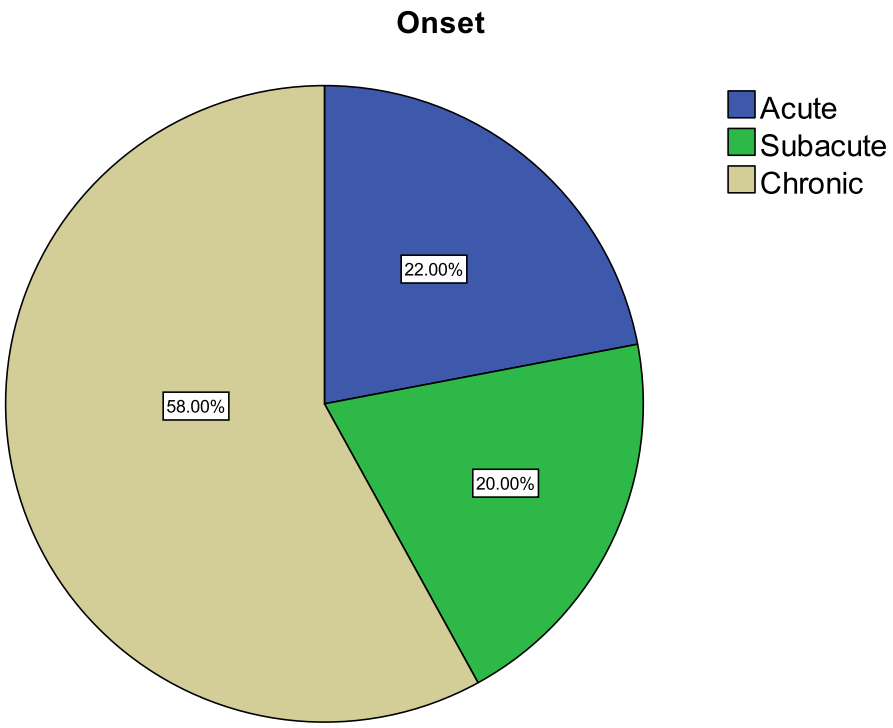
48 patients with nontraumatic myelopathy had paraparesis at presentation whereas 46% were quadriparetic. One patient presented with brachial monoparesis. 4 patients did not manifest with any weakness and had presented to the hospital with non-motor complaints only.

Table 2: Pattern of weakness at presentation

Weakness	Frequency	Percent	Valid Percent	Cumulative Percent
Asymptomatic	4	4.0	4.0	4.0
Crural monoparesis	1	1.0	1.0	5.0
Paraparesis	48	48.0	48.0	53.0
Quadriparesis	46	46.0	46.0	99.0
Brachial monoparesis	1	1.0	1.0	100.0
Total	100	100.0	100.0	

Onset of illness was acute in 22 % patients. Subacute onset was noted in another 20 % whereas 58 patients had a chronic and progressive course prior to presenting to the hospital.

Fig 2: Onset of illness in nontraumatic myelopathy

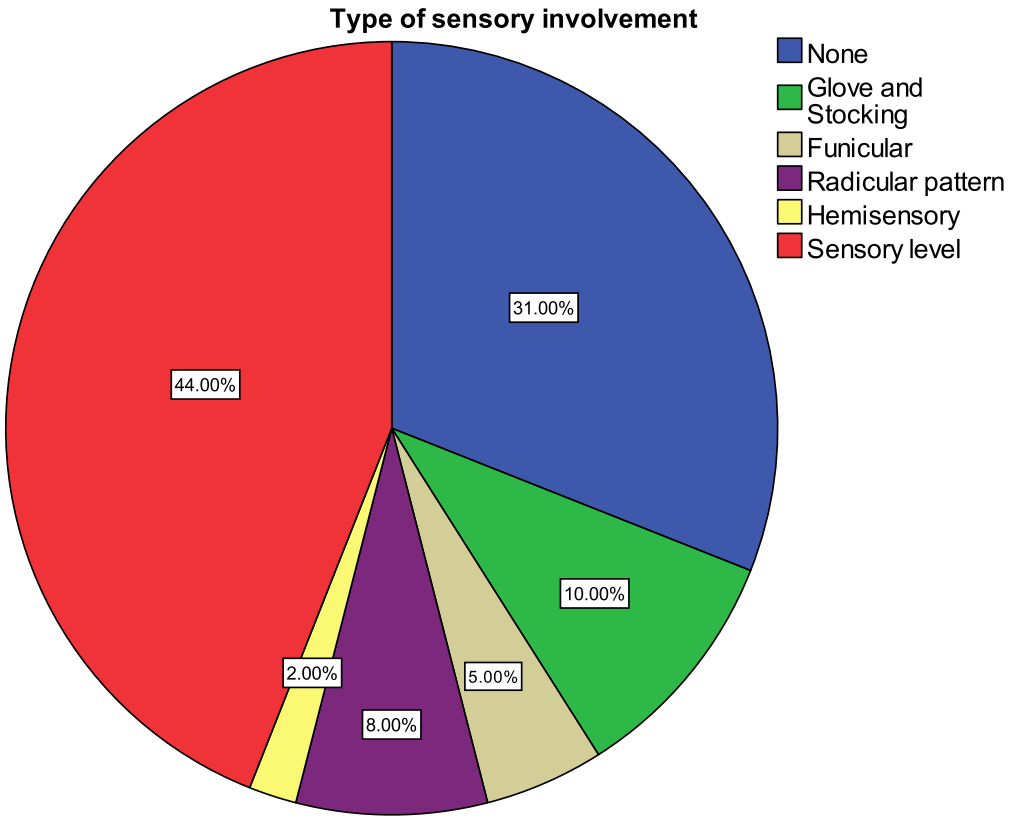


95% of the patients had both proximal and distal limb weakness and 2 patients presented with distal predominant weakness in addition to clinical evidence of myelopathy. The presenting motor weakness was symmetric in 46% of patients and asymmetric in 48% of the patients.

Of the hundred cases, sensory complaints were present in 69 patients. In those patients, 44 patients presented with a sensory level, 10 patients manifested with glove and stocking type of sensory loss, 8

patients had radicular pattern of sensory loss, 5 patients manifested with diffuse funicular pain, and only 2 patients had hemisensory loss.

Fig 3: Sensory complaints



Bowel disturbances were present in 11 % of the patients with only one patient manifesting as bowel incontinence whereas the rest of the patients with bowel disturbances had constipation as their predominant bowel related complaint.

In contrast, urinary sphincter disturbances were seen in a higher number of the patients with nontraumatic myelopathy. 14% presented

with acute urinary retention, 34 % had complaints of urgency, 5 % had hesitancy, and only one patient had urinary incontinence at presentation.

Table 3: Spectrum ofbladder related complaints

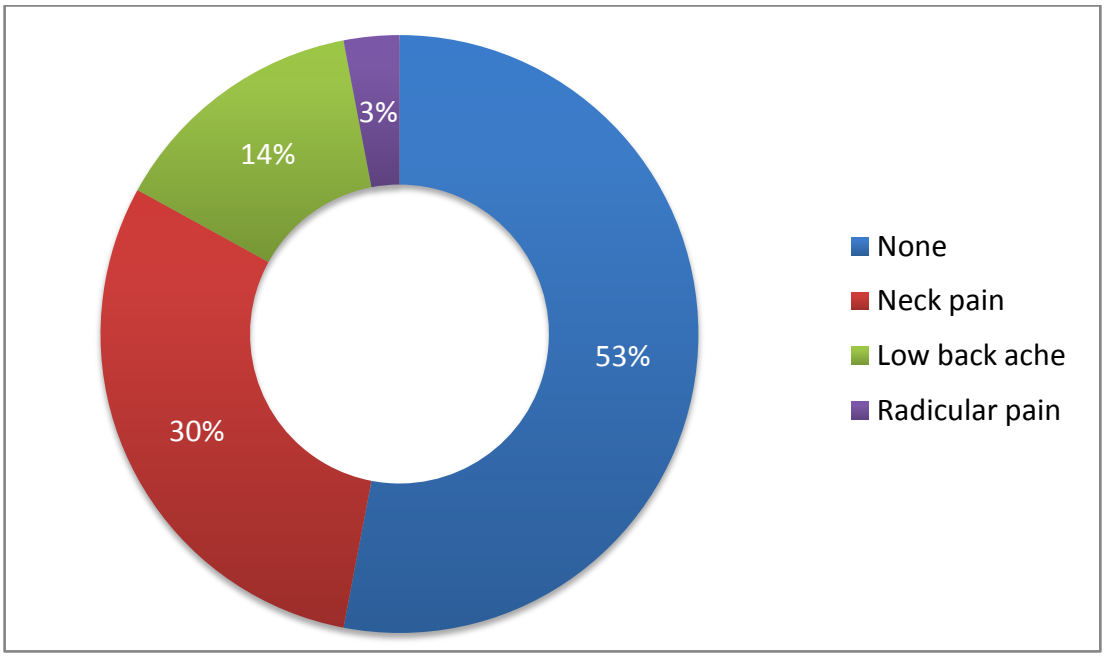
Bladder symptoms	Frequency	Percent	Valid Percent	Cumulative Percent
None	44	44.0	44.0	44.0
Urgency	34	34.0	34.0	78.0
Incontinence	2	2.0	2.0	80.0
Retention	14	14.0	14.0	94.0
Hesitancy	5	5.0	5.0	99.0
Frequency	1	1.0	1.0	100.0
Total	100	100.0	100.0	

Only three patients manifested with speech disturbances. One had a spastic quality of speech and the other two had cerebellar pattern.

Visual symptoms were present in 9% of patients with 7 patients having acute loss of vision and two patients presenting with progressive blurring of vision. All the patients with visual complaints belonged to the non-compressive group. None of the recruited patients had any symptoms pertaining to auditory involvement, although one patient was found to have bilateral sensori-neural hearing loss after clinical examination and audiological assessment.

Neck pain was the most frequent form of vertebral complaint manifesting in 30% of patients, followed by low back ache which was seen in 14% of the nontraumatic myelopathy patients.

Fig 4: Vertebral symptoms reported by patients



The patients reported a history of trauma in 4% of total cases, whereas three patients had a past history of similar episodes prior to the current presentation. 3 patients had co-existing fever, 2 patients had anemia with past history of prior blood transfusions, and one patient had significant cachexia.

Co-morbid conditions were present in 19 % of cases which were diabetes mellitus (3%), systemic hypertension (7 %), tuberculosis (4 %), HIV (3%), and hyperlipidemia (1 %).

21 % reported history of alcohol abuse and 7% had the habit of chronic smoking. 4 % also reported exposure to high risk sexual partners. Only 2% had significant family history. Both were noted to be cases of hereditary spastic paraparesis.

All patients had Glasgow Coma scale (GCS) scores greater than 8 at admission. None presented with any alteration of sensorium. Only one patient who was severely vitamin B12 deficient presented with dementia.

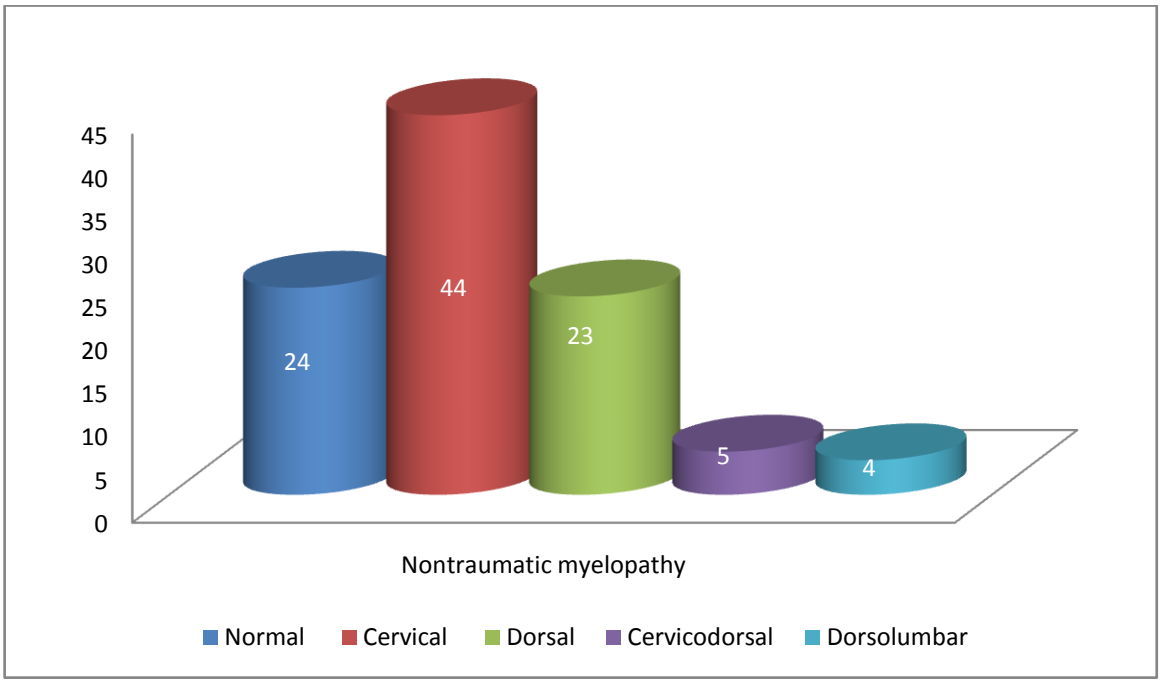
Examination revealed optic neuropathy in 10% of cases, with all cases belonging to non-compressive group. One patient was noted to have bilateral sensorineural hearing loss. Wasting with reduced bulk of musculature was seen in 4% of cases. Spasticity was noted in 74% of patients while 17% were hypotonic. It was also seen that 51% of patients had a positive Romberg sign, 10% had peripheral neuropathy, 10% had radicular involvement, and 7% had cerebellar signs.

Investigative and etiologic profile in nontraumatic myelopathy

Investigations showed anemia in 19% of the nontraumatic myelopathy cases and one patient manifested with bicytopenia. Other routine blood investigations did not reveal any statistically significant abnormality. Four patients had evidence of tuberculosis on radiologic

evaluation and another three were reactive for HIV – ELISA. None of the patients with HIV had co-existing tuberculosis. Syphilis serology by VDRL technique was non-reactive in all patients. CSF examination was done in all 48 patients with noncompressive myelopathy in which 9 patients showed elevated protein levels and one was oligoclonal band positive.

Fig 5: MRI findings in nontraumatic myelopathy



MRI spine was abnormal in 76% of the total patients. All the patients with normal MRI picture belonged to the noncompressive myelopathy group. MRI spine showed no lesions in 24 patients, cervical cord lesions in 44 patients, dorsal cord lesions in 23 patients. Analysis of the distribution of lesions in the MRI positive individuals showed 44

patients (57.89 %) having the pathology in the cervical cord. In those patients with cervical cord lesions, majority (84.1%) belonged to the compressive myelopathy group. Longitudinally extending transverse myelitis (LETM) lesions were noted in 11% of patients. All patients with LETM belonged to the noncompressive group only.

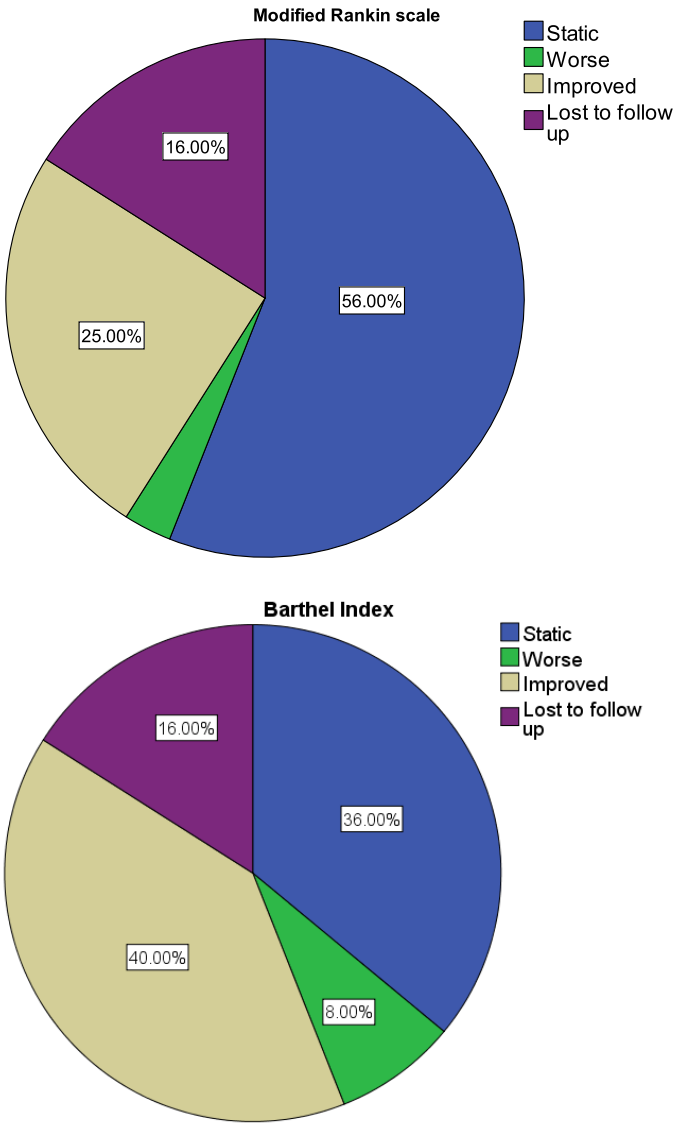
Out of the 100 patients with nontraumatic myelopathy, 59 patients received medical management and 41 patients underwent surgery. All those who underwent surgery had compressive myelopathy. 11 % of the nontraumatic myelopathy patients who had compressive myelopathy received conservative medical management due to their lack of fitness for surgery or their unwillingness to undergo surgical intervention. Twenty eight out of 48 patients were managed with steroids in the noncompressive myelopathy group. Demyelinating disorders like acute transverse myelitis, multiple sclerosis and NMO spectrum disorders were the common etiologies requiring use of steroids.

Functional outcome in nontraumatic myelopathy

The mean Barthel index score at initial presentation was 51.15 $\pm$ 19.67 with a range of 5 to 100. The corresponding mean mRS score was 3.29$\pm$0.81 with values ranging between 0 and 5 in the patients with

nontraumatic myelopathy. None of the patients enrolled in the study died during the hospital stay.

Fig 6: Functional outcome at 6 months



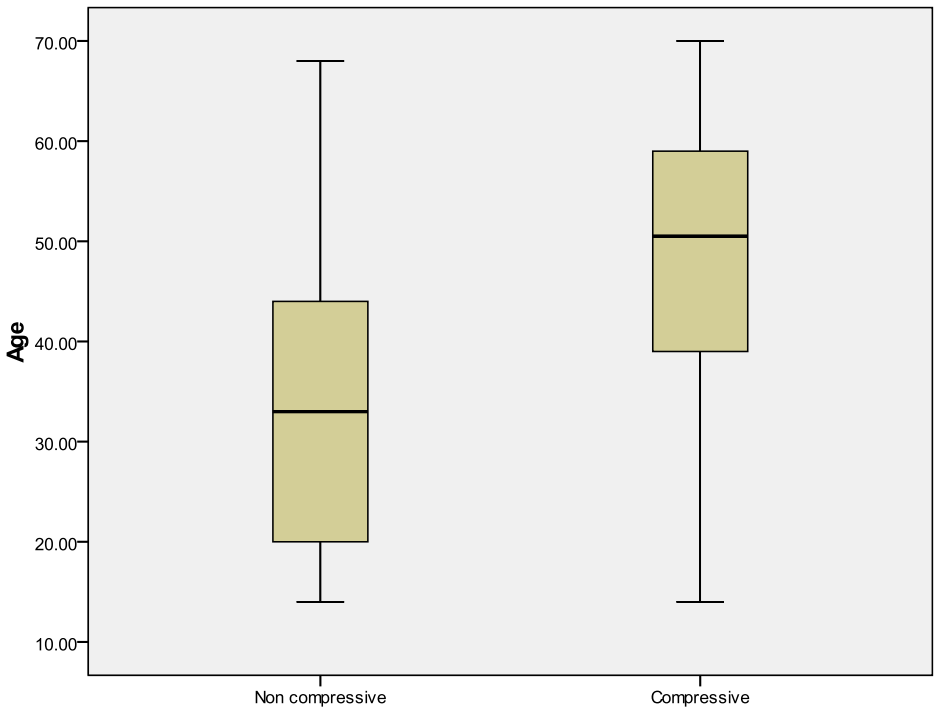
16 patients were lost to followup at the end of 6 months. Of them, 4 patients belonged to noncompressive myelopathy group and 12 patients belonged to compressive myelopathy group. Evaulation at 6 months

revealed that average Barthel index was 55.77± 19.56 and average mRS score was 3.02± 0.86. At the end of 6 months, it was noted that 40 % and 25 % patients had improved in their Barthel index and mRS scores respectively, whereas 36 % and 56 % of the patients had remained static in their Barthel index and mRS scores respectively. 8 % had paradoxically worsened Barthel index scores.

Compressive myelopathy versus noncompressive myelopathy:

The mean age of patients with non compressive myelopathy was 34.27± 15.66 years whereas the mean age was 46.78± 15.12 years.

Fig 7: Age spectrum in both groups



Out of the patients with nontraumatic myelopathy, 59% were males. Of the males, 55.9% belonged to the compressive myelopathy group. Interestingly, 53.7% of the females belonged to the noncompressive group which was not statistically significant.

Table 4: Sex distribution in both groups

			Group			
			Non compressive	Compressive	Total	p- value
Sex	Male	Count	26	33	59	0.345
		% within Sex	44.1%	55.9%	100.0%	
	Female	Count	22	19	41	
		% within Sex	53.7%	46.3%	100.0%	
	Total	Count	48	52	100	
		% within Sex	48.0%	52.0%	100.0%	

The onset was acute in 22 patients, all (100%) of them belonging to the non compressive group, whereas chronic course of illness was noted in 58 patients, with 18 patients (31%) belonging to the noncompressive group and the rest 69% (40 patients) in compressive group. The difference between the two groups was statistically significant ($p<0.05$).

Table 5: Onset of illness in both groups

			Group			
			NCM	CM	Total	p- value
Onset	Acute	Count	22	0	22	**<0.01****
		%	100.0%	.0%	100.0%	
	Sub-acute	Count	8	12	20	
		%	40.0%	60.0%	100.0%	
	Chronic	Count	18	40	58	
		%	31.0%	69.0%	100.0%	
	Total	Count	48	52	100	
		%	48.0%	52.0%	100.0%	

***- p<0.05; **- p<0.01**

Comparing the pattern of weakness between compressive and non compressive revealed that 35 patients (72.9 %) of the total 48 paraparetic patients belonged to the non-compressive group, whereas 38 (82.6%) of the 46 patients with quadriparesis belonged to the compressive myelopathy group, and the difference between the two groups was alsostatistically significant ($p<0.05$).

The varied etiological spectrum in compressive and non-compressive myelopathies is elaborated in the Table 5 and Table 6.

Table 5:Aetiologic diagnosis in noncompressive myelopathy

Non compressive myelopathy	Frequency	Percent
Acute transverse myelitis	23	47. 91
-Idiopathic ATM	7	14.58
-MS Spectrum	5	10.42
-NMO Spectrum	11	22.91
HIV related	3	6.25
B12 deficiency	6	12.5
HSP	8	16.66
TB Myeloradiculitis	1	2.09
Hirayama	1	2.09
Unclassified	6	12.5
Total	48	100.0

Table 6:Aetiologic diagnosis in compressive myelopathy

Compressive myelopathy	Frequency	Percent
Cervical spondylotic myelopathy	29	55.76
-Spondylotic cervical canal stenosis	17	32.69
-IVDP	6	11.54
-OPLL	4	7.69
-Hypertropic ligamentum flavum	2	3.84
Pott’s spine	3	5.78
CVJ anomaly	1	1.92
Atlantoaxial dislocation	1	1.92
C1-C2 subluxation	3	5.78
Extramedullary SOL	7	13.46
Intramedullary SOL	6	11.54
Dural AVM	1	1.92
Metastasis	1	1.92
Total	52	100

The compressive and non-compressive myelopathy patients in our study showed differences in clinical presentation as well as location of radiological lesion including muscle tone at presentation, pattern of weakness and bladder disturbances between the two groups showed statistical significance.94.1% of patients with hypotonia belonged to the noncompressive myelopathy group whereas 67.6% of patients with spasticity belonged to the compressive group, the difference of which showed statistical significance ($p<0.01$). Symmetric weakness was significantly seen more frequently in noncompressive myelopathy.

Fig 8: Sensory complaints between both groups

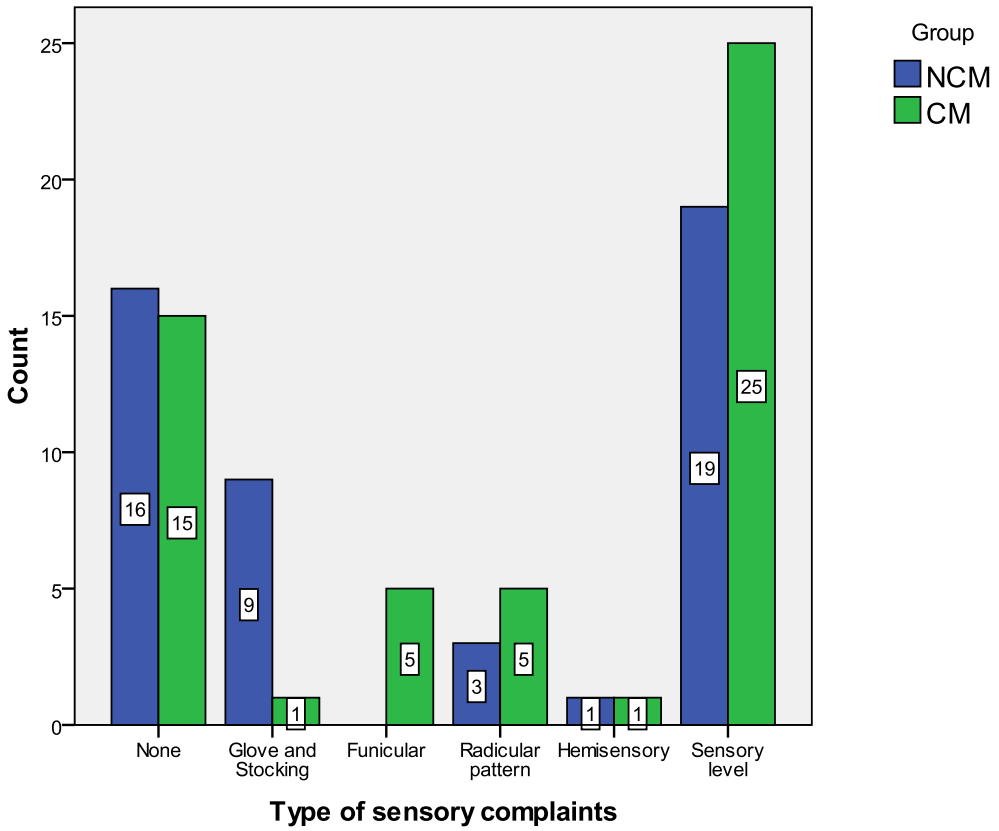


Table 7: Spectrum of bladder complaints in both groups

Bladder disturbances		Group		Total	p- value
		NCM	CM		
None	Count (%)	21	23	44	**0.01***
	Percent	47.7%	52.3%	100.0%	
Urgency	Count	9	25	34	
	Percent	26.5%	73.5%	100.0%	
Incontinence	Count	1	1	2	
	Percent	50.0%	50.0%	100.0%	
Retention	Count	12	2	14	
	Percent	85.7%	14.3%	100.0%	
Hesitancy	Count	5	0	5	
	Percent	100.0%	0%	100.0%	
Frequency	Count	0	1	1	
	Percent	.0%	100.0%	100.0%	
Total	Count	48	52	100	
	Percent	48.0%	52.0%	100.0%	

***- p<0.05; **- p<0.01**

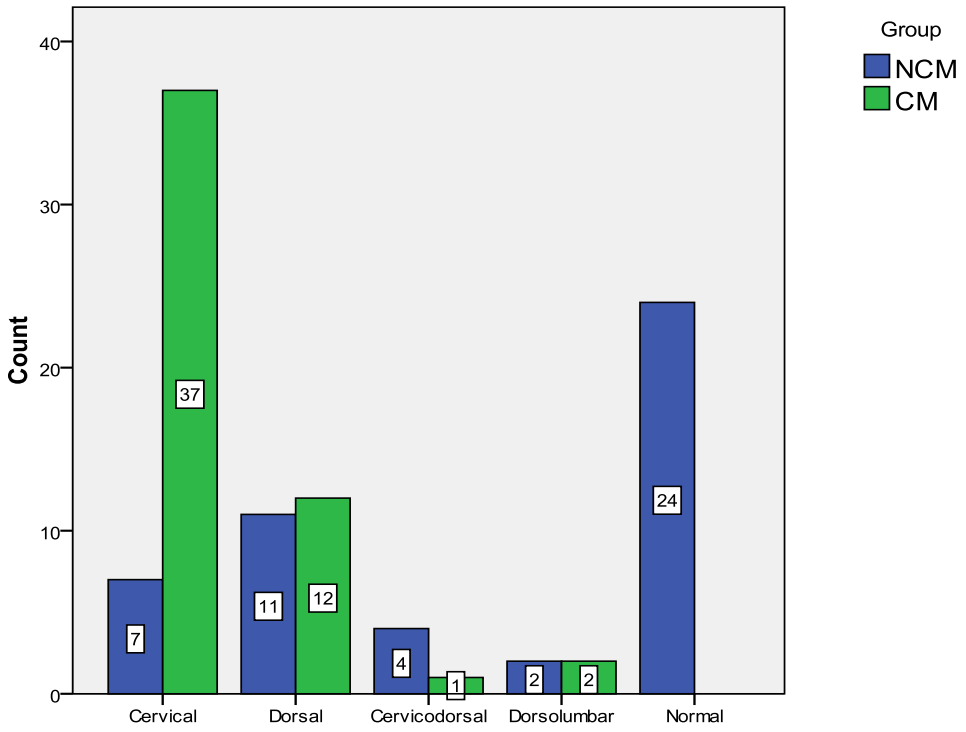
The location of MRI lesion in compressive and noncompressive myelopathy also showed statistical significance with cervical lesions dominating compressive myelopathy and dorsal cord lesions more commonly seen in non-compressive myelopathy.

Table 8: MRI lesion location between two groups

MRI lesion location	Group		Total	p- value
	NCM	CM		
Cervical	7	37	44	
Dorsal	11	12	23	
Cervicodorsal	4	1	5	**<0.01****
Dorsolumbar	2	2	4	
Normal	24	0	24	
Total	48	52	100	

***- p<0.05; **- p<0.01**

Fig 9: MRI lesion location between two groups



Functional outcome and predictors:

The functional outcome in patients with nontraumatic myelopathy assessed using Barthel index and modified Rankin scale was done on the

84 followed-up patients at the end of 6 months, which are depicted below in Fig 9 and Fig 10.

Fig 9: Functional outcome of Barthel index at 6 months

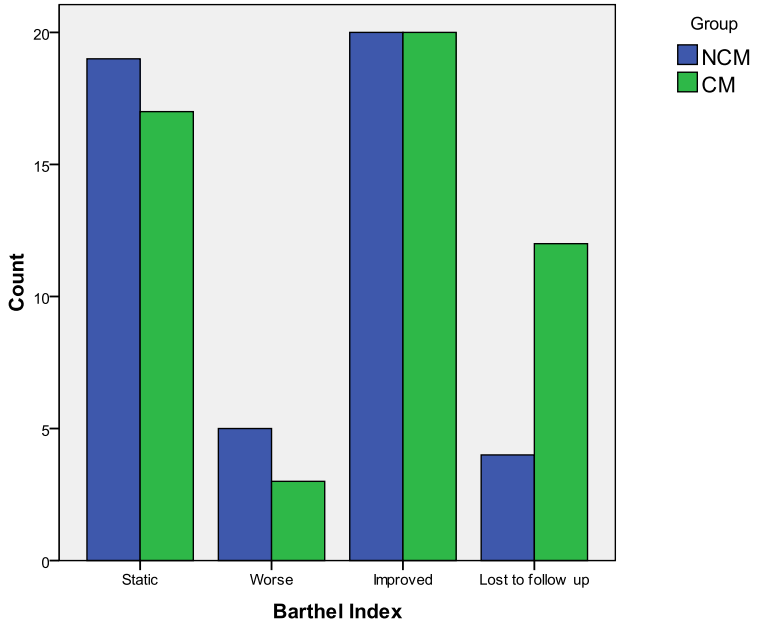


Fig 10: Functional outcome of modified Rankinscale at 6 months

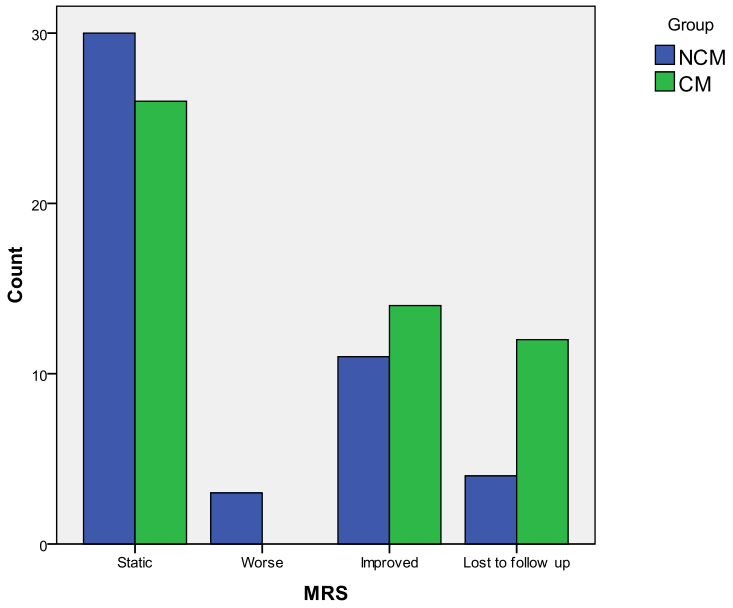


Table 8: Multivariate analysis on impact of specific factors on initial Barthel index and mRS

Effect		Value	F	Hypothesis df	Error df	P value
Intercept	Pillai's Trace	.902	362.067	2.000	79.000	.000
	Wilks' Lambda	.098	362.067	2.000	79.000	.000
	Hotelling's Trace	9.166	362.067	2.000	79.000	.000
	Roy's Largest Root	9.166	362.067	2.000	79.000	.000
Acute onset	Pillai's Trace	.019	.785	2.000	79.000	.459
	Wilks' Lambda	.981	.785	2.000	79.000	.459
	Hotelling's Trace	.020	.785	2.000	79.000	.459
	Roy's Largest Root	.020	.785	2.000	79.000	.459
Abnormal MRI Spine	Pillai's Trace	.054	2.241	2.000	79.000	.113
	Wilks' Lambda	.946	2.241	2.000	79.000	.113
	Hotelling's Trace	.057	2.241	2.000	79.000	.113
	Roy's Largest Root	.057	2.241	2.000	79.000	.113
Presence of sensory level	Pillai's Trace	.000	.001	2.000	79.000	.999
	Wilks' Lambda	1.000	.001	2.000	79.000	.999
	Hotelling's Trace	.000	.001	2.000	79.000	.999
	Roy's Largest Root	.000	.001	2.000	79.000	.999
Paraparesis	Pillai's Trace	.064	2.679	2.000	79.000	.075
	Wilks' Lambda	.936	2.679	2.000	79.000	.075
	Hotelling's Trace	.068	2.679	2.000	79.000	.075
	Roy's Largest Root	.068	2.679	2.000	79.000	.075
Quadriparesis	Pillai's Trace	.228	11.689	2.000	79.000	**.000****
	Wilks' Lambda	.772	11.689	2.000	79.000	**.000****
	Hotelling's Trace	.296	11.689	2.000	79.000	**.000****
	Roy's Largest Root	.296	11.689	2.000	79.000	**.000****
MRI lesion in cervical cord	Pillai's Trace	.034	1.379	2.000	79.000	.258
	Wilks' Lambda	.966	1.379	2.000	79.000	.258
	Hotelling's Trace	.035	1.379	2.000	79.000	.258
	Roy's Largest Root	.035	1.379	2.000	79.000	.258

Bladder disturbances	Pillai's Trace	.348	4.215	8.000	160.000	**.000****
	Wilks' Lambda	.655	4.656	8.000	158.000	**.000****
	Hotelling's Trace	.522	5.094	8.000	156.000	**.000****
	Roy's Largest Root	.514	10.272	4.000	80.000	**.000****
Diagnosis of ATM or cervical spondylosis	Pillai's Trace	.054	1.107	4.000	160.000	.355
	Wilks' Lambda	.946	1.108	4.000	158.000	.355
	Hotelling's Trace	.057	1.108	4.000	156.000	.355
	Roy's Largest Root	.056	2.252	2.000	80.000	.112
LETM	Pillai's Trace	.116	5.165	2.000	79.000	**.008****
	Wilks' Lambda	.884	5.165	2.000	79.000	**.008****
	Hotelling's Trace	.131	5.165	2.000	79.000	**.008****
	Roy's Largest Root	.131	5.165	2.000	79.000	**.008****
Treatment Modality	Pillai's Trace	.082	1.714	4.000	160.000	.149
	Wilks' Lambda	.918	1.729	4.000	158.000	.146
	Hotelling's Trace	.089	1.743	4.000	156.000	.143
	Roy's Largest Root	.088	3.532	2.000	80.000	.034
Group	Pillai's Trace	.000		.000	.000	.
	Wilks' Lambda	1.000		.000	79.500	.
	Hotelling's Trace	.000		.000	2.000	.
	Roy's Largest Root	.000	.000	2.000	78.000	1.000

***- p<0.05; **- p<0.01**

The MANOVA model was used to assess the impact of several disease related factors on the functional status at presentation which showed that there was a statistically significant difference in the Barthel Index and mRS between the groups who have and do not have bladder disturbance, quadriparesis or LETM. No statistically significant differences in the BI and mRSwere noted in the other factors.

Table 9: Multivariate analysis on impact of specific factors on 6-month Barthel index and mRS

Effect		Value	F	Hypothesis df	Error df	P value
Intercept	Pillai's Trace	.890	255.642	2.000	63.000	.000
	Wilks' Lambda	.110	255.642	2.000	63.000	.000
	Hotelling's Trace	8.116	255.642	2.000	63.000	.000
	Roy's Largest Root	8.116	255.642	2.000	63.000	.000
Acuteonset	Pillai's Trace	.011	.364	2.000	63.000	.697
	Wilks' Lambda	.989	.364	2.000	63.000	.697
	Hotelling's Trace	.012	.364	2.000	63.000	.697
	Roy's Largest Root	.012	.364	2.000	63.000	.697
Abnormal MRISpine	Pillai's Trace	.015	.466	2.000	63.000	.630
	Wilks' Lambda	.985	.466	2.000	63.000	.630
	Hotelling's Trace	.015	.466	2.000	63.000	.630
	Roy's Largest Root	.015	.466	2.000	63.000	.630
Presenceofsensorylevel	Pillai's Trace	.025	.806	2.000	63.000	.451
	Wilks' Lambda	.975	.806	2.000	63.000	.451
	Hotelling's Trace	.026	.806	2.000	63.000	.451
	Roy's Largest Root	.026	.806	2.000	63.000	.451
Paraparesis	Pillai's Trace	.014	.454	2.000	63.000	.637
	Wilks' Lambda	.986	.454	2.000	63.000	.637
	Hotelling's Trace	.014	.454	2.000	63.000	.637
	Roy's Largest Root	.014	.454	2.000	63.000	.637
Quadriparesis	Pillai's Trace	.102	3.589	2.000	63.000	**.033***
	Wilks' Lambda	.898	3.589	2.000	63.000	**.033***
	Hotelling's Trace	.114	3.589	2.000	63.000	**.033***
	Roy's Largest Root	.114	3.589	2.000	63.000	**.033***
MRIlesion in cervical cord	Pillai's Trace	.005	.168	2.000	63.000	.846
	Wilks' Lambda	.995	.168	2.000	63.000	.846
	Hotelling's Trace	.005	.168	2.000	63.000	.846
	Roy's Largest Root	.005	.168	2.000	63.000	.846

Bladder disturbances	Pillai's Trace	.339	3.268	8.000	128.000	**.002****
	Wilks' Lambda	.671	3.483	8.000	126.000	**.001****
	Hotelling's Trace	.477	3.694	8.000	124.000	**.001****
	Roy's Largest Root	.444	7.097	4.000	64.000	**.000****
Diagnosis of ATM or cervical spondylosis	Pillai's Trace	.091	1.528	4.000	128.000	.198
	Wilks' Lambda	.910	1.527	4.000	126.000	.198
	Hotelling's Trace	.098	1.525	4.000	124.000	.199
	Roy's Largest Root	.088	2.829	2.000	64.000	.066
LETM	Pillai's Trace	.023	.746	2.000	63.000	.479
	Wilks' Lambda	.977	.746	2.000	63.000	.479
	Hotelling's Trace	.024	.746	2.000	63.000	.479
	Roy's Largest Root	.024	.746	2.000	63.000	.479
Treatment Modality	Pillai's Trace	.038	.626	4.000	128.000	.644
	Wilks' Lambda	.962	.619	4.000	126.000	.650
	Hotelling's Trace	.039	.611	4.000	124.000	.656
	Roy's Largest Root	.031	.994	2.000	64.000	.376
Group	Pillai's Trace	.000		.000	.000	.
	Wilks' Lambda	1.000		.000	63.500	.
	Hotelling's Trace	.000		.000	2.000	.
	Roy's Largest Root	.000	.000	2.000	62.000	1.000

***- p<0.05; **- p<0.01**

The MANOVA model used to assess the impact of the same disease related factors on the functional outcome at 6 months showed that there was a statistically significant difference in the Barthel Index and mRS between the groups who have and do not have bladder disturbance and quadriparesis. No statistically significant differences in the BI and mRSwere noted in the other factors including LETM which had initially shown significance for functional status at presentation.

DISCUSSION

Patients with nontraumatic myelopathy are broadly subdivided as compressive myelopathy and noncompressive myelopathy based on the evidence of clinical and neuroradiological features of any spinal cord lesion (extrinsic or intrinsic) causing compression. The clinical features as well as the etiological spectrum in either group are known to differ in varying levels in the prior studies. There is scanty information on the functional outcome of patients with nontraumatic myelopathy as a whole group in the current literature.This study was undertaken to study the clinical spectrum of patients with nontraumatic myelopathy and assess their functional outcome at 6 months.

Out of the previous studies on nontraumatic myelopathies, only one study from India assesses the aetiologic spectrum in nontraumatic myelopathy, examining them as a whole group. According to Chaurasia et al [13], the most common etiology of compressive myelopathy was tuberculosis comprising 35.71% of the group, which is in contrast to our findings which shows that majority of compressive myelopathy was contributed by cervical degenerative spondylotic myelopathy (55.76%) alone. Tuberculosis as a cause of compressive myelopathy was seen in only 5.78% of the compressive myelopathy patients. In the

noncompressive group, Chaurasia et al[13] described that 21.79% of cases were due to acute transverse myelitis whereas our findings show that acute transverse myelitis comprised 47.91% of the cases.

Another Indian study by Yadav et al [14] describing the spectrum of compressive myelopathies also showed that spinal tuberculosis was the commonest cause (24.6%) followed by spinal metastases (17.4%). Ossified posterior longitudinal ligament (OPLL) constituted 7.8% of the total cases which correlated with our study population which had OPLL in 7.69% of the cases.

Prabhakar et al [15] reported the clinical and radiological findings in 57 Indian patients with noncompressive myelopathy having a mean age of 34.45 years which was similar demographically to our population whose mean age was 34.27 years. He reported that 81% were symmetrical with 54.38 % of the cases being acute transverse myelitis. 12.28 % of their patients were diagnosed to have vitamin B12 deficiency which was similar to the 12.5% of vitamin B12 deficiency in our study population.

A hospital based study on nontraumatic myelopathies by Lekoubou Looti et al[18]in Cameroon showed similarities in the demographic profile

of their study cases with our patients. However, 83.7% of their patients manifested with paraparesis and only 16.3% with quadriparesis at presentation, which is quite in contrast to our findings of nearly equal cases of paraparesis (48%) and quadriparesis (46%). Also, 89.8% of their cases had a sensory level on examination as against our study group who manifested with a definite sensory level in only 44% of the cases. While 80.7% of their study subjects had sphincter disturbances, only 56% of our patients reported bladder related complaints. The most common cause of nontraumatic myelopathy in their cohort was primary or secondary spinal tumors which accounted for 24.5% of the total cases whereas our study had maximum proportion of cases of cervical spondylotic myelopathy.

A Nigerian study on the profile of nontraumatic paraplegia[19] also reported tuberculosis as the commonest causes accounting to 44.7% of the patients. Also 14.1% of their patients were found to be positive in HIV screening, whereas our study showed 3% seropositivity for HIV in the nontraumatic myelopathy patients. Interestingly, Modi et al [20] who assessed the prevalence of HIV in nontraumatic myelopathy in HIV endemic South African hospital noted a high prevalence of HIV in 51.54% of the admitted cases.

Das et al [16] who studied the profile of non-compressive myelopathy in Eastern India showed acute presentation in 48.78% and chronic presentation in 32.92%. Etiological diagnosis was established in71.95% patients and transverse myelitis was diagnosed in 29.26%.

Western studies have shown cervical spondylotic myelopathy as the commonest cause of compressive myelopathy. Moore et al [21] who studied 585 patients with spastic parparesis and tetraparesis found that spondylotic myelopathy was the commonest accounting for 24 % of the total cases. Most of his cases were of compressive etiology and multiple sclerosis was detected in 9.1 % of the cases. He reported arteriovenous malformation (AVM) in 0.9% of his cases which parallels our study cohort who had AVM in 1% of the population.

Studies on functional outcome in nontraumatic myelopathy are not available in the current literature, although outcome assesement in compressive and noncompressive myelopathy of specific etiologies have been described.

Christensen et al[33] described the longterm follow-up of 29 cases of acute transverse myelopathy and noted that one third had a good outcome, while one third had poor outcome. Back-pain and signs of spinal shock were found to indicate worse outcome in his study. This is in contrast to our study in which mRS showed improvement in 25 % and

worsening in 3%, whereas Barthel index showed improvement in 40 % and worsening in 8% of the patients.

One interesting study by Ebner et al[38], has shown that patients diagnosed to have extended intramedullary lesions have a worse neurological status in the perioperative as well as inthe 3-month followup. In contrast, our study which attempted to assess the impact of specific disease related factors associated with the functional status at presentation and 6 month functional outcome found that although LETM was found to correlate with functional status at admission, it did not have any significant association with the functional outcome at 6 months. However, quadriparesis and bladder disturbances were found to have significant association with functional status at presentation as well as at 6 month followup.

CONCLUSION

1. The spectrum of nontraumatic myelopathy in our study population showed equal distribution of compressive as well as non-compressive myelopathy.
2. While the commonest cause of compressive myelopathy was cervical spondylotic myelopathy, demyelinating diseases with acute transverse myelitis presentation was the most common cause of non-compressive myelopathy.
3. The most common location for cord lesion was in the cervical cord based on radiological evaluation.
4. Significant number of patients remained static in their functional status at the end of 6 months.
5. Quadriparesis and bladder symptoms at initial presentation significantly showed association with the 6-month functional outcome, whereas LETM lesions on MRI showed significant association with the functional status at first presentation.

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PROFORMA

"A STUDY OF THE CLINICAL SPECTRUM AND FUNCTIONAL OUTCOME OF PATIENTS WITH NONTRAUMATIC MYELOPATHY"

PATIENT INFORMATION:

SERIAL NO.

NAME: AGE ……. YEARS

GENDER: MALE/FEMALE MIN NO. OP/IP NO:

ADDRESS: PHONE NO:

DISTRICT: OCCUPATION:

SOCIOECONOMIC STATUS: EDUCATION:

HISTORY & PRESENTATION:

Primary complaints Duration (D/W/M/Y)

1. -
2. -
3. -
4. –

- Distal weakness Duration
  - Tripping on toes
  - Difficulty in holding slippers
  - Unaware slippage of chappals
- Proximal weakness
  - Buckling of knees
  - Difficulty in getting up from squatting
- Trunk muscle weakness
  - Difficulty in rolling over
- Neck muscle weakness
  - Difficulty in lifting head above pillow
- Sensory complaints Duration Location
  - Parasthesias
  - Burning sensation
  - Numbness
  - Loss of hot/cold sensation
- Bladder and bowel
  - Urgency
  - Urge incontinence

    - Hesistancy
    - Precipitancy
    - Increased frequency
    - Urine retention
    - Painful retention of urine
- Visual
    - Loss of color vision
    - Transient visual obscuration
    - Night blindness
    - Progressive loss of vision
- Auditory
    - Tinnitus
    - Loss of hearing
- Vertebral symptoms
    - Neck pain
    - Low back pain
    - Radiating pain
    - Kyphosis or scoliosis

PAST HISTORY:

- Trauma
- Vision loss
- Radiation exposure
- Previous similar episodes

ASSOCIATED CONDITIONS:

- Diarrhea
- Anemia or blood transfusions
- Cachexia
- Previous hypercoagulable episodes

COMORBIDITIES

DM /Hypertension /CAD /Stroke /Hyperlipidemia /Liver disease /Kidney disease/Thyroid disease /Rheumatological illness /Medications for systemic illnesses (with duration)/ Others_____

PERSONAL HISTORY

Alcoholism /smoking /tobacco chewing/ substance abuse (mention form and type)
Diet- Vegetarian / Non vegetarian (type and frequency)
Drinking water source (mention)

FAMILY HISTORY

DM /HT / CAD /Hyperlipidemia /similar complaints
Others_____

EXAMINATION:

Temperature
Pulse
BP mmHg
Pallor/Icterus/Lymphadenopathy/Clubbing/Pedal oedema
Carotid bruit
Peripheral pulsation
Thyroid swelling
Neurocutaneous markers
Facial dysmorphism

CENTRAL NERVOUS SYSTEM:
HMF:
Sensorium: GCS: E V M
Speech
MMSE:
Cranial nerves: Right Left

1. Olfactory nerve:
2. Optic nerve
3. Extraocular movements: (3,4,6)
4. Trigeminal nerve:
5. Facial nerve
6. Vestibulocochlear nerve:
7. Glossopharyngeal and vagal nerve
8. Accesory nerve:
9. Hypoglossal nerve:

Superficial reflexes Right Left

- Corneal
- Conjunctival
- Abdominal
- Cremasteric
- Plantar

Spino motor system:

- Bulk
- Tone- UL
- Tone- LL

Power		Right	Left
Neck	Flexion		
	Extension		
Shoulder	Abduction		
	Adduction		
	Flexion		
	Extension		
Elbow	Flexion		

	Extension		
Wrist	Flexion		
	Extension		
Handgrip			
Hip	Abduction		
	Adduction		
	Flexion		
	Extension		
Knee	Flexion		
	Extension		
Ankle	Flexion		
	Dorsiflexion		
Extensor hallucislongus			
Flexor hallucislongus			
Deep tendon reflexes			
Biceps			
Triceps			
Supinator			
Finger flexion			
Wartenburg			
Hoffmans			
Knee (±clonus)			
Ankle (±clonus)			

Sensory: Right Left

- Pain
- Touch
- Temperature
- Vibration
- Position sense
- Joint sense
- Rombergs sign

Cerebellar Signs:

- Hypotonia
- Nystagmus
- Titubation
- Gait
- Stance ataxia
- Tandem walking
- Finger nose incoordination
- Past pointing
- Rebound phenomenon

- Intention tremors
- Heel knee test

Meningeal signs:

SLR:

Others

CVS:

RS:

ABDOMEN:

BARTHEL INDEX

FEEDING
0 = unable
5 = needs help cutting, spreading butter, etc., or requires modified diet
10 = independent _____
BATHING
0 = dependent
5 = independent (or in shower) _____
GROOMING
0 = needs to help with personal care
5 = independent face/hair/teeth/shaving (implements provided) _____
DRESSING
0 = dependent
5 = needs help but can do about half unaided
10 = independent (including buttons, zips, laces, etc.) _____
BOWELS
0 = incontinent (or needs to be given enemas)
5 = occasional accident
10 = continent _____
BLADDER
0 = incontinent, or catheterized and unable to manage alone
5 = occasional accident
10 = continent _____
TOILET USE
0 = dependent
5 = needs some help, but can do something alone
10 = independent (on and off, dressing, wiping) _____

TRANSFERS (BED TO CHAIR AND BACK)
0 = unable, no sitting balance
5 = major help (one or two people, physical), can sit
10 = minor help (verbal or physical)
15 = independent _____
MOBILITY (ON LEVEL SURFACES)
0 = immobile or < 50 yards
5 = wheelchair independent, including corners, > 50 yards
10 = walks with help of one person (verbal or physical) > 50 yards
15 = independent (but may use any aid; for example, stick) > 50 yards _____
STAIRS
0 = unable
5 = needs help (verbal, physical, carrying aid)
10 = independent _____

Date: _____ **TOTAL (0–100): _____**

Follow up date: _____ **TOTAL (0–100): _____**

MODIFIED RANKIN SCALE

Score Description

0 No symptoms at all
1 No significant disability despite symptoms; able to carry out all usual duties and activities
2 Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
3 Moderate disability; requiring some help, but able to walk without assistance
4 Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5 Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6 Dead

Date: _____ **TOTAL (0–6): _____**

Follow up date: _____ **TOTAL (0–6): _____**

LAB INVESTIGATIONS

Hb % gm% / PCV
TC / DC
ESR
Platelets
Peripheral smear

Mantoux

Blood sugar

Blood urea /Serum creatinine
Serum electrolytes

Liver function test

ECG

X ray Chest

X-Ray Spine

USG Abdomen

Thyroid profile

Serum Vitamin B12 levels

Serum homocysteine levels

Bone marrow examination

Upper GI endoscopy

Antral biopsy

Serum copper levels

ANA/ds DNA
c-ANCA/ p-ANCA
ACL-IgG/IgM
Lupus anticoagulant
HIV

VDRL

Serum ACE levels

Nerve conduction studies for associated neuropathy

Evoked potentials

CSF:

- Cell count
- Cytology
- Protein
- Sugar
- VDRL
- Gram stain
- AFB
- Oligoclonal bands

MRI SPINE:

FINAL DIAGNOSIS:

ACUTE/SUBACUTE/CHRONIC

COMPRESSIVE/NONCOMPRESSIVE

CERVICAL/THORACIC/LUMBAR/SACRAL

COMPLICATIONS, IF ANY

ETIOLOGY DETERMINED

MASTER CHART

S.NO	NAME	AGE	SEX	ONSET	WEAKNESS PATTTERN	LOCATION	PATTERN OF SENSORY LOSS	BOWEL INV	BLADDER INV	SPEECH	VISUAL SYMPTOMS	VERTEBRAL SYMPTOMS	PAST HISTORY	ASSOSCIATED HISTORY	CO-MORBIDITIES	PERSONAL HISTORY	FAMILY HISTORY	BULK	TONE	ROMBERG
1	ABITHA	16.00	2.00	1.00	3.00	4.00	7.00	1.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
2	AKSHAYA	18.00	2.00	1.00	4.00	4.00	7.00	1.00	5.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00
3	ASHOK KUMAR	42.00	1.00	1.00	3.00	4.00	7.00	1.00	4.00	1.00	1.00	3.00	3.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
4	BABY	17.00	2.00	1.00	4.00	4.00	7.00	1.00	4.00	1.00	1.00	2.00	1.00	6.00	1.00	1.00	1.00	1.00	2.00	3.00
5	BOOPALAN	34.00	1.00	3.00	3.00	4.00	2.00	1.00	2.00	1.00	1.00	1.00	1.00	3.00	12.00	6.00	1.00	1.00	3.00	2.00
6	GANESAN	15.00	1.00	2.00	3.00	4.00	5.00	1.00	1.00	1.00	1.00	4.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	1.00
7	GEETHA	23.00	2.00	2.00	1.00	1.00	1.00	1.00	2.00	1.00	2.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
8	GNANAVEL	61.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	2.00	2.00	6.00	1.00	3.00	1.00
9	HOUSAR NAHEEMA	18.00	2.00	1.00	3.00	4.00	7.00	1.00	2.00	1.00	4.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
10	JAYALAKSHMI	20.00	2.00	2.00	3.00	4.00	5.00	1.00	2.00	1.00	1.00	3.00	1.00	6.00	13.00	1.00	1.00	2.00	2.00	3.00
11	KARUPAYEE	40.00	2.00	1.00	4.00	4.00	7.00	1.00	4.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
12	KUMAR	31.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
13	KUMARI	20.00	2.00	1.00	3.00	4.00	7.00	1.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
14	MALLIKA	24.00	2.00	2.00	3.00	4.00	5.00	1.00	2.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	2.00
15	MANIKANDAN	15.00	1.00	3.00	3.00	4.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
16	NAGALAKSHMI	14.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
17	NAINA MOHAMMED	20.00	2.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
18	NARESHBABU	33.00	1.00	3.00	3.00	4.00	2.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	6.00	1.00	1.00	3.00	2.00
19	PADMA	62.00	2.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	4.00	1.00	1.00	1.00	3.00	1.00
20	PALANI	37.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	6.00	1.00	3.00	1.00
21	PALANISAMY	40.00	1.00	1.00	3.00	4.00	7.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00
22	PARANJOTHI	39.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	1.00
23	PARVATHY	50.00	2.00	1.00	3.00	4.00	7.00	1.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
24	RAJA	32.00	1.00	1.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	2.00	1.00	1.00	2.00	1.00
25	RANJITHKUMAR	19.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
26	SADAGOPAN	49.00	1.00	1.00	4.00	4.00	7.00	1.00	4.00	1.00	1.00	2.00	1.00	1.00	12.00	2.00	1.00	1.00	2.00	3.00
27	SANGEETHA	38.00	2.00	1.00	3.00	4.00	7.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
28	SAROJA	65.00	2.00	3.00	4.00	4.00	2.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
29	SASI	17.00	2.00	3.00	3.00	4.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
30	SATHISH	35.00	1.00	3.00	3.00	4.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
31	SAVITHRIAMMAL	50.00	2.00	1.00	2.00	4.00	7.00	3.00	4.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
32	SEETHARAMAN	44.00	1.00	2.00	3.00	4.00	7.00	3.00	4.00	1.00	2.00	1.00	5.00	5.00	1.00	1.00	1.00	1.00	3.00	2.00
33	SEKAR	39.00	1.00	3.00	3.00	4.00	2.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	6.00	1.00	1.00	3.00	2.00
34	SELVAM	22.00	1.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
35	SELVAM	19.00	1.00	1.00	4.00	4.00	7.00	1.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
36	SELVARAJ	47.00	1.00	1.00	3.00	4.00	7.00	1.00	4.00	1.00	1.00	3.00	1.00	6.00	1.00	1.00	1.00	1.00	3.00	2.00
37	SHANAVAS	49.00	1.00	1.00	4.00	4.00	7.00	3.00	4.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00
38	SHEIKH MANSOOR	22.00	1.00	2.00	5.00	3.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00
39	SOKKAMAL	56.00	2.00	1.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
40	SRINIVASAN	68.00	1.00	2.00	3.00	4.00	1.00	2.00	3.00	1.00	1.00	1.00	1.00	3.00	1.00	2.00	1.00	1.00	4.00	3.00
41	STELLA	23.00	2.00	1.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00
42	SUBRAMANI	68.00	1.00	3.00	3.00	4.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
43	THIRUPATHY	44.00	1.00	1.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	3.00
44	VALARMATHY	32.00	2.00	3.00	3.00	4.00	2.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	12.00	6.00	1.00	1.00	3.00	2.00
45	VENKATASUBRAMANI	33.00	1.00	2.00	3.00	4.00	1.00	1.00	2.00	1.00	4.00	1.00	5.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
46	VENKATESAN	44.00	1.00	3.00	3.00	4.00	2.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
47	VIJAYA	16.00	2.00	1.00	1.00	4.00	6.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
48	VIJAYALAKSHMI	25.00	2.00	1.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00

MASTER CHART

S.NO	NAME	AGE	SEX	ONSET	WEAKNESS PATTTERN	LOCATION	PATTERN OF SENSORY LOSS	BOWEL INV	BLADDER INV	SPEECH	VISUAL SYMPTOMS	VERTEBRAL SYMPTOMS	PAST HISTORY	ASSOSCIATED HISTORY	CO-MORBIDITIES	PERSONAL HISTORY	FAMILY HISTORY	BULK	TONE	ROMBERG
49	ANNAMALAI	40.00	1.00	2.00	3.00	4.00	7.00	1.00	2.00	1.00	1.00	4.00	1.00	4.00	13.00	2.00	1.00	1.00	3.00	2.00
50	ARJUNAN	52.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	2.00	1.00	3.00	2.00	1.00	1.00	3.00	2.00
51	ARUMUGAM	45.00	1.00	3.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	6.00	3.00	1.00	1.00	3.00	2.00
52	BEEMAKUMAR	58.00	1.00	3.00	4.00	4.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
53	CHINNASAMY	50.00	1.00	3.00	4.00	4.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
54	DHANABAKKIYAM	61.00	2.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
55	JEYAMMAL	50.00	2.00	3.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
56	KANNAN	33.00	1.00	3.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
57	KANNIYAMMAL	67.00	2.00	3.00	3.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
58	KESAVAN	41.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00	1.00	1.00	3.00	2.00
59	KODANDAPANI	63.00	1.00	3.00	4.00	4.00	7.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	3.00	1.00	1.00	3.00	2.00
60	KOTI REDDY	65.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
61	KRISHNAMOORTHY	60.00	1.00	3.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	2.00	2.00	1.00	3.00	1.00	1.00	1.00	3.00	2.00
62	KUMAR	58.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	2.00
63	LAVANYA	23.00	2.00	2.00	3.00	4.00	3.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
64	MAHENDRAN	60.00	1.00	3.00	4.00	4.00	7.00	3.00	2.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00	1.00	1.00	3.00	2.00
65	MALLIGA	46.00	2.00	3.00	4.00	4.00	7.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
66	MANI	45.00	1.00	3.00	1.00	1.00	1.00	1.00	8.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
67	MANIBALA	60.00	1.00	2.00	3.00	4.00	3.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	1.00
68	MANICKAM	60.00	1.00	3.00	3.00	4.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	1.00
69	MARIMUTHU	55.00	1.00	3.00	4.00	4.00	5.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
70	MEERA	49.00	2.00	3.00	3.00	4.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	1.00
71	MEERAVATHY	63.00	2.00	3.00	4.00	4.00	2.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
72	MUNUSAMY	16.00	1.00	3.00	4.00	4.00	1.00	1.00	4.00	2.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
73	MUTHU	27.00	1.00	2.00	4.00	4.00	3.00	3.00	3.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
74	MUTHURAJ	14.00	1.00	3.00	4.00	3.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
75	NATARAJAN	60.00	1.00	3.00	4.00	4.00	7.00	3.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
76	NAZEENA	27.00	2.00	3.00	4.00	4.00	1.00	3.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
77	PADMANABHAN	23.00	1.00	3.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
78	PALANINAICKAR	60.00	1.00	2.00	4.00	4.00	1.00	3.00	1.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	3.00	2.00
79	PANEERSELVAM	57.00	1.00	3.00	4.00	4.00	7.00	3.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
80	PERIYASAMY	50.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
81	PERUMAL	55.00	1.00	2.00	4.00	4.00	7.00	1.00	4.00	1.00	1.00	2.00	1.00	1.00	13.00	1.00	1.00	1.00	3.00	1.00
82	PONMANI	45.00	2.00	3.00	4.00	4.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
83	PREMA	38.00	2.00	3.00	4.00	4.00	5.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	3.00	2.00
84	RAFIKA	52.00	2.00	2.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
85	RAMACHANDRAN	57.00	1.00	3.00	4.00	4.00	7.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
86	RAMAMOORTHY	62.00	1.00	2.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00	1.00	1.00	3.00	3.00
87	RANGANATHAN	55.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
88	SANGEETHA	29.00	2.00	2.00	3.00	4.00	7.00	1.00	2.00	1.00	1.00	4.00	1.00	1.00	13.00	1.00	1.00	1.00	3.00	2.00
89	SARAVANAN	45.00	1.00	2.00	4.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	1.00
90	SATYAVAMITHRA	51.00	2.00	2.00	3.00	4.00	3.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
91	SELVARAJ	52.00	1.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
92	SELVI	45.00	2.00	3.00	4.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
93	SHANTHI	29.00	2.00	3.00	4.00	4.00	7.00	1.00	2.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	1.00	1.00	3.00	2.00
94	SUNDARAMMAL	70.00	2.00	3.00	4.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
95	SUNDARAMOORTHY	56.00	1.00	3.00	4.00	4.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00	1.00	3.00	2.00
96	SURYA	15.00	1.00	2.00	4.00	4.00	6.00	1.00	1.00	1.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
97	THANGAMMA	17.00	2.00	3.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
98	THANGAMMAL	53.00	2.00	3.00	3.00	4.00	7.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00
99	VENKATASUBRAMANI	48.00	1.00	3.00	4.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	1.00
100	VINOTHINI	21.00	2.00	3.00	3.00	4.00	7.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	3.00	2.00

MASTER CHART

S.NO	NAME	HEMO GRAM	TB EVIDENCE	HIV	CSF	MRI SPINE	MRI LESION LOCATION	LET M	ASSOCIATED DEFICITS	DIAGNOSTIC CLASSIFICATION	GROSS DIAGNOSTIC SUBGROUPING	INTERVENTION DONE	TREATMENT STRATERGY	INITIAL BARTHEL INDEX	BARTHEL INDEX 6 MONTH	BI OUTCOME	INITIAL MRS	MRS 6 MONTH	MRS OUTCOME	CM vs NCM
1	ABITHA	1.00	1.00	1.00	2.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	30.00	55.00	3.00	4.00	3.00	3.00	2.00
2	AKSHAYA	1.00	1.00	1.00	1.00	2.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	35.00	45.00	3.00	4.00	3.00	3.00	2.00
3	ASHOK KUMAR	1.00	1.00	1.00	1.00	2.00	2.00	1.00	2.00	3.00	1.00	1.00	1.00	25.00	25.00	1.00	4.00	4.00	1.00	2.00
4	BABY	1.00	1.00	1.00	2.00	2.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	10.00	20.00	3.00	4.00	3.00	3.00	2.00
5	BOOPALAN	2.00	1.00	2.00	1.00	1.00	6.00	2.00	3.00	4.00	2.00	1.00	2.00	65.00	LTFW	4.00	3.00	LTFW	4.00	2.00
6	GANESAN	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	1.00	1.00	1.00	1.00	60.00	60.00	1.00	3.00	3.00	1.00	2.00
7	GEETHA	1.00	1.00	1.00	1.00	2.00	1.00	1.00	2.00	3.00	2.00	1.00	1.00	95.00	95.00	1.00	1.00	1.00	1.00	2.00
8	GNANAVEL	2.00	1.00	1.00	5.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	75.00	75.00	1.00	2.00	2.00	1.00	2.00
9	HOUSAR NAHEEMA	1.00	1.00	1.00	1.00	2.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
10	JAYALAKSHMI	2.00	2.00	1.00	2.00	1.00	6.00	2.00	1.00	7.00	2.00	1.00	1.00	65.00	55.00	2.00	2.00	3.00	2.00	2.00
11	KARUPAYEE	1.00	1.00	1.00	1.00	2.00	4.00	1.00	1.00	3.00	1.00	1.00	1.00	30.00	25.00	2.00	4.00	4.00	1.00	2.00
12	KUMAR	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	8.00	2.00	1.00	2.00	70.00	LTFW	4.00	3.00	LTFW	4.00	2.00
13	KUMARI	2.00	1.00	1.00	1.00	2.00	4.00	1.00	1.00	3.00	1.00	1.00	1.00	25.00	25.00	1.00	4.00	4.00	1.00	2.00
14	MALLIKA	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	8.00	2.00	1.00	1.00	60.00	70.00	3.00	3.00	2.00	3.00	2.00
15	MANIKANDAN	2.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	5.00	2.00	1.00	2.00	65.00	75.00	3.00	2.00	2.00	1.00	2.00
16	NAGALAKSHMI	1.00	1.00	1.00	1.00	2.00	1.00	1.00	2.00	3.00	1.00	1.00	1.00	100.00	70.00	2.00	0.00	2.00	2.00	2.00
17	NAINA MOHAMMED	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	75.00	75.00	1.00	2.00	2.00	1.00	2.00
18	NARESHBABU	1.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	5.00	2.00	1.00	2.00	65.00	70.00	3.00	3.00	2.00	3.00	2.00
19	PADMA	2.00	1.00	1.00	1.00	1.00	6.00	2.00	4.00	6.00	2.00	1.00	2.00	70.00	75.00	3.00	3.00	3.00	1.00	2.00
20	PALANI	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
21	PALANISAMY	1.00	1.00	1.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	65.00	70.00	3.00	3.00	3.00	1.00	2.00
22	PARANJOTHI	1.00	1.00	1.00	1.00	1.00	6.00	2.00	4.00	6.00	2.00	1.00	2.00	70.00	70.00	1.00	3.00	3.00	1.00	2.00
23	PARVATHY	1.00	1.00	1.00	2.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	35.00	45.00	3.00	4.00	3.00	1.00	2.00
24	RAJA	1.00	1.00	1.00	5.00	1.00	6.00	2.00	1.00	8.00	1.00	1.00	1.00	70.00	LTFW	4.00	3.00	LTFW	4.00	2.00
25	RANJITHKUMAR	2.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
26	SADAGOPAN	1.00	1.00	2.00	2.00	1.00	6.00	2.00	1.00	1.00	1.00	1.00	1.00	10.00	25.00	3.00	5.00	4.00	3.00	2.00
27	SANGEETHA	2.00	1.00	1.00	1.00	2.00	2.00	1.00	2.00	3.00	1.00	1.00	1.00	75.00	85.00	3.00	3.00	2.00	3.00	2.00
28	SAROJA	1.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	8.00	2.00	1.00	2.00	30.00	40.00	3.00	4.00	4.00	1.00	2.00
29	SASI	1.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	65.00	75.00	3.00	3.00	3.00	1.00	2.00
30	SATHISH	1.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	8.00	2.00	1.00	1.00	70.00	70.00	1.00	3.00	3.00	1.00	2.00
31	SAVITHRIAMMAL	2.00	1.00	1.00	1.00	2.00	5.00	1.00	1.00	3.00	1.00	1.00	1.00	25.00	35.00	3.00	4.00	3.00	3.00	2.00
32	SEETHARAMAN	1.00	1.00	1.00	1.00	2.00	2.00	1.00	2.00	3.00	1.00	1.00	1.00	30.00	30.00	1.00	4.00	4.00	1.00	2.00
33	SEKAR	1.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	5.00	2.00	1.00	2.00	60.00	70.00	3.00	3.00	2.00	3.00	2.00
34	SELVAM	2.00	1.00	1.00	1.00	1.00	6.00	2.00	1.00	6.00	2.00	1.00	2.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
35	SELVAM	1.00	1.00	1.00	2.00	2.00	4.00	1.00	1.00	1.00	1.00	1.00	1.00	10.00	5.00	2.00	5.00	5.00	1.00	2.00
36	SELVARAJ	2.00	1.00	1.00	2.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	25.00	35.00	3.00	4.00	4.00	1.00	2.00
37	SHANAVAS	1.00	1.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	1.00	5.00	5.00	1.00	5.00	5.00	1.00	2.00
38	SHEIKH MANSOOR	1.00	1.00	1.00	5.00	1.00	6.00	2.00	1.00	9.00	2.00	1.00	2.00	80.00	80.00	1.00	2.00	2.00	1.00	2.00
39	SOKKAMAL	2.00	1.00	1.00	1.00	2.00	2.00	1.00	1.00	3.00	1.00	1.00	1.00	60.00	LTFW	4.00	3.00	LTFW	4.00	2.00
40	SRINIVASAN	3.00	1.00	1.00	1.00	2.00	2.00	2.00	3.00	5.00	2.00	1.00	2.00	25.00	45.00	3.00	4.00	3.00	3.00	2.00
41	STELLA	1.00	1.00	1.00	1.00	2.00	2.00	2.00	1.00	3.00	1.00	1.00	1.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
42	SUBRAMANI	2.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	5.00	2.00	1.00	2.00	65.00	70.00	3.00	3.00	3.00	1.00	2.00
43	THIRUPATHY	1.00	1.00	1.00	1.00	2.00	4.00	1.00	1.00	3.00	1.00	1.00	1.00	60.00	75.00	3.00	3.00	2.00	3.00	2.00
44	VALARMATHY	2.00	1.00	2.00	1.00	1.00	6.00	2.00	3.00	4.00	2.00	1.00	2.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00
45	VENKATASUBRAMANI	1.00	1.00	1.00	1.00	2.00	1.00	2.00	2.00	2.00	2.00	1.00	1.00	60.00	50.00	2.00	3.00	4.00	2.00	2.00
46	VENKATESAN	1.00	1.00	1.00	1.00	1.00	6.00	2.00	3.00	5.00	2.00	1.00	2.00	70.00	70.00	1.00	3.00	3.00	1.00	2.00
47	VIJAYA	1.00	1.00	1.00	1.00	2.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	30.00	40.00	3.00	4.00	4.00	1.00	2.00
48	VIJAYALAKSHMI	1.00	1.00	1.00	2.00	2.00	5.00	2.00	1.00	8.00	1.00	1.00	1.00	65.00	65.00	1.00	3.00	3.00	1.00	2.00

MASTER CHART

S.NO	NAME	HEMO GRAM	TB EVIDENCE	HIV	CSF	MRI SPINE	MRI LESION LOCATION	LET M	ASSOCIATED DEFICITS	DIAGNOSTIC CLASSIFICATION	GROSS DIAGNOSTIC SUBGROUPING	INTERVENTION DONE	TREATMENT STRATERGY	INITIAL BARTHEL INDEX	BARTHEL INDEX 6 MONTH	BI OUTCOME	INITIAL MRS	MRS 6 MONTH	MRS OUTCOME	CM vs NCM
49	ANNAMALAI	2.00	2.00	1.00	5.00	2.00	2.00	2.00	1.00	3.00	4.00	1.00	4.00	60.00	75.00	3.00	3.00	2.00	3.00	1.00
50	ARJUNAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	40.00	50.00	3.00	4.00	3.00	3.00	1.00
51	ARUMUGAM	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	4.00	3.00	2.00	3.00	30.00	25.00	2.00	4.00	4.00	1.00	1.00
52	BEEMAKUMAR	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	5.00	3.00	2.00	3.00	35.00	LTFW	4.00	4.00	LTFW	4.00	1.00
53	CHINNASAMY	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	1.00	4.00	65.00	LTFW	4.00	3.00	LTFW	4.00	1.00
54	DHANABAKKIYAM	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	45.00	45.00	1.00	3.00	3.00	1.00	1.00
55	JEYAMMAL	1.00	1.00	1.00	5.00	2.00	5.00	2.00	1.00	9.00	4.00	2.00	3.00	70.00	70.00	1.00	3.00	3.00	1.00	1.00
56	KANNAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	30.00	45.00	3.00	4.00	3.00	3.00	1.00
57	KANNIYAMMAL	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	9.00	4.00	2.00	3.00	65.00	65.00	1.00	3.00	3.00	1.00	1.00
58	KESAVAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	35.00	LTFW	4.00	4.00	LTFW	4.00	1.00
59	KODANDAPANI	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	35.00	45.00	3.00	4.00	3.00	3.00	1.00
60	KOTI REDDY	1.00	1.00	1.00	4.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	30.00	30.00	1.00	4.00	4.00	1.00	1.00
61	KRISHNAMOORTHY	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	35.00	LTFW	4.00	4.00	LTFW	4.00	1.00
62	KUMAR	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	1.00	4.00	30.00	40.00	3.00	4.00	4.00	1.00	1.00
63	LAVANYA	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	10.00	4.00	2.00	3.00	65.00	60.00	2.00	3.00	3.00	1.00	1.00
64	MAHENDRAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	1.00	4.00	35.00	35.00	1.00	4.00	4.00	1.00	1.00
65	MALLIGA	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	35.00	35.00	1.00	4.00	4.00	1.00	1.00
66	MANI	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	9.00	4.00	2.00	3.00	90.00	100.00	3.00	1.00	0.00	3.00	1.00
67	MANIBALA	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	10.00	4.00	2.00	3.00	60.00	LTFW	4.00	3.00	LTFW	4.00	1.00
68	MANICKAM	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	10.00	4.00	2.00	3.00	65.00	70.00	3.00	3.00	2.00	3.00	1.00
69	MARIMUTHU	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	40.00	35.00	2.00	4.00	4.00	1.00	1.00
70	MEERA	1.00	1.00	1.00	5.00	2.00	5.00	2.00	1.00	9.00	4.00	2.00	3.00	70.00	80.00	3.00	3.00	2.00	3.00	1.00
71	MEERAVATHY	1.00	1.00	1.00	4.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	65.00	65.00	1.00	3.00	3.00	1.00	1.00
72	MUNUSAMY	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	8.00	4.00	2.00	3.00	30.00	50.00	3.00	4.00	3.00	3.00	1.00
73	MUTHU	1.00	1.00	1.00	5.00	2.00	4.00	2.00	1.00	10.00	4.00	2.00	3.00	70.00	70.00	1.00	3.00	3.00	1.00	1.00
74	MUTHURAJ	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	7.00	4.00	2.00	3.00	35.00	LTFW	4.00	4.00	LTFW	4.00	1.00
75	NATARAJAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	45.00	55.00	3.00	4.00	3.00	3.00	1.00
76	NAZEENA	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	6.00	4.00	2.00	3.00	35.00	35.00	1.00	4.00	4.00	1.00	1.00
77	PADMANABHAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	8.00	4.00	2.00	3.00	40.00	40.00	1.00	4.00	4.00	1.00	1.00
78	PALANINAICKAR	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	40.00	45.00	3.00	4.00	4.00	1.00	1.00
79	PANEERSELVAM	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	35.00	LTFW	4.00	4.00	LTFW	4.00	1.00
80	PERIYASAMY	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	4.00	3.00	2.00	3.00	40.00	40.00	1.00	4.00	4.00	1.00	1.00
81	PERUMAL	1.00	2.00	1.00	5.00	2.00	1.00	2.00	1.00	3.00	4.00	2.00	3.00	40.00	55.00	3.00	4.00	3.00	3.00	1.00
82	PONMANI	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	8.00	4.00	2.00	3.00	55.00	60.00	3.00	3.00	3.00	1.00	1.00
83	PREMA	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	4.00	3.00	2.00	3.00	45.00	45.00	1.00	3.00	3.00	1.00	1.00
84	RAFIKA	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	9.00	4.00	2.00	3.00	70.00	80.00	3.00	2.00	1.00	3.00	1.00
85	RAMACHANDRAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	9.00	4.00	2.00	3.00	40.00	LTFW	4.00	3.00	LTFW	4.00	1.00
86	RAMAMOORTHY	2.00	1.00	1.00	1.00	2.00	1.00	2.00	1.00	1.00	3.00	1.00	4.00	35.00	LTFW	4.00	3.00	LTFW	4.00	1.00
87	RANGANATHAN	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	1.00	4.00	65.00	75.00	3.00	3.00	2.00	3.00	1.00
88	SANGEETHA	2.00	2.00	1.00	5.00	2.00	2.00	2.00	1.00	3.00	4.00	1.00	4.00	75.00	80.00	3.00	2.00	2.00	1.00	1.00
89	SARAVANAN	1.00	1.00	1.00	1.00	2.00	1.00	2.00	1.00	1.00	3.00	1.00	4.00	65.00	65.00	1.00	3.00	3.00	1.00	1.00
90	SATYAVAMITHRA	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	10.00	4.00	2.00	3.00	65.00	65.00	1.00	3.00	3.00	1.00	1.00
91	SELVARAJ	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	40.00	55.00	3.00	4.00	3.00	3.00	1.00
92	SELVI	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	1.00	3.00	2.00	3.00	45.00	55.00	3.00	3.00	3.00	1.00	1.00
93	SHANTHI	2.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	45.00	LTFW	4.00	3.00	LTFW	4.00	1.00
94	SUNDARAMMAL	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	2.00	3.00	2.00	3.00	35.00	35.00	1.00	4.00	4.00	1.00	1.00
95	SUNDARAMOORTHY	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	4.00	3.00	1.00	4.00	40.00	LTFW	4.00	3.00	LTFW	4.00	1.00
96	SURYA	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	10.00	4.00	2.00	3.00	35.00	40.00	3.00	4.00	4.00	1.00	1.00
97	THANGAMMA	2.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	11.00	4.00	2.00	4.00	70.00	70.00	1.00	3.00	3.00	1.00	1.00
98	THANGAMMAL	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	5.00	4.00	2.00	3.00	70.00	75.00	3.00	3.00	2.00	3.00	1.00
99	VENKATASUBRAMANI	1.00	1.00	1.00	5.00	2.00	1.00	2.00	1.00	12.00	4.00	1.00	4.00	45.00	LTFW	4.00	3.00	LTFW	4.00	1.00
100	VINOTHINI	1.00	1.00	1.00	5.00	2.00	2.00	2.00	1.00	9.00	4.00	2.00	3.00	65.00	65.00	1.00	3.00	3.00	1.00	1.00

KEYS FOR MASTER CHART

Sex	1= Male; 2= Female
Onset	1= Acute; 2= Subacute; 3= Chronic
Weakness pattern	1= None; 2= Crural monoparesis; 3= Paraparesis; 4= Quadriparesis; 5= Brachial monoparesis
Weakness location	1= None; 2= Distal predominant; 3= Proximal, 4= Both distal and proximal
Pattern of sensory loss	1= None; 2= Glove and stocking pattern; 3= Funicular; 4= Mononeuritis distribution; 5= Radicular pattern; 6= Hemisensory; 7= Definite sensory level
Bowel involvement	1= None; 2= Incontinence; 3= Constipation
Bladder involvement	1= None; 2= Urgency; 3= Incontinence; 4= Retention; 5= Hesistancy; 6= Precipitancy; 7= Painful retention; 8= Frequency
Speech involvement	1= None; 2= Spastic; 3= Cerebellar
Visual symptoms	1= None; 2= Loss of vision; 3= Night blindness; 4= Progressive diminution of vision
Vertebral symptoms	1= None; 2= Neck pain; 3= Back pain; 4= Radicular pain
Past history	1= None; 2= Trauma; 3= Visual loss; 4= Radiation; 5= Previous similar episodes
Associated history	1= None; 2= Diarrhoea; 3= Anemia or prior transfusions; 4= Cachexia; 5= Prior hypercoagulable states; 6= Fever
Co-morbidities	1= None; 2= Diabetes mellitus; 3= Hypertension; 4= Coronary artery disease; 5= Stroke; 6= Hyperlipidemia;7= Liver disease; 8= Kidney disease; 9= Thyroid disease; 10= Rheumatological disease; 11= Chronic medications; 12= HIV; 13= TB
Personal history	1= None; 2= Alcoholism; 3= Smoking; 4= Tobacco use; 5= Substance abuse; 6= Exposure to high risk partners
Family history	1= None; 2= Diabetes mellitus; 3= Hypertension; 4= Coronary artery disease; 5= Hyperlipidemia 6= Similar illness
Bulk	1= Normal; 2= Reduced; 3= Hypertrophied
Tone	1= Normal; 2= Hypotonic; 3= Spasticity; 4= Rigidity
Romberg	1= Negative; 2= Positive; 3= Could not be tested
Hemogram	1= Normal; 2= Anemia; 3= Bicytopenia
TB evidence	1= None; 2= Present
HIV	1= Nonreactive; 2= Reactive
CSF	1= Normal; 2= Elevated protein; 3= Decreased sugar; 4= Oligoclonal bands; 5= Not done
MRI spine	1= Normal; 2= Abnormal
MRI lesion location	1= Cervical; 2= Dorsal; 3= Lumbar; 4= Cervicodorsal; 5= Dorsolumbar; 6= No lesions
LETM	1= Present; 2= Absent
Associated deficits	1= None; 2= Optic neuropathy; 3= Peripheral neuropathy; 4= Cerebellar involvement
Diagnostic classification/ NCM	1= Idiopathic ATM; 2= MS spectrum; 3= NMO spectrum; 4= HIV related; 5= B12 deficiency; 6= HSP; 7= TB myeloradiculitis; 8= Unclassified; 9= Hirayama

Diagnostic classification/ CM	1= Cervical spondylosis; 2= IVDP; 3= Pott's spine; 4= OPLL; 5= Hypertrophic ligamentum flavum; 6= CVJ anomaly; 7= Atlantoaxial dislocation; 8= C1-C2 subluxation; 9= Extramedullary SOL; 10= Intramedullary SOL; 11= Dural AVM; 12= Metastasis
Gross diagnostic subgrouping	1= ATM/ NCM; 2= Non ATM /NCM; 3= Cervical spondylosis/ CM; 4= Non cervical spondylosis/ CM
Intervention done	1= Medical; 2= Surgical
Treatment strategy	1= Steroids/NCM; 2= Non steroid management/ NCM; 3= Surgery/CM; 4= Conservative/ CM
BI ans mRS outcoem	1= Static; 2= Worse; 3= Improved; 4= Lost to folowup
LTFW	Lost to followup

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.D.Anusha
PG in Neurology
Madras Medical College,Chennai -3

Dear Dr.D.Anusha,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " A study of the clinical spectrum and functional outcome of patients with nontraumatic myelopathy" No.22112012.

The following members of Ethics Committee were present in the meeting held on 01.11.2012 conducted at Madras Medical College, Chennai -3.

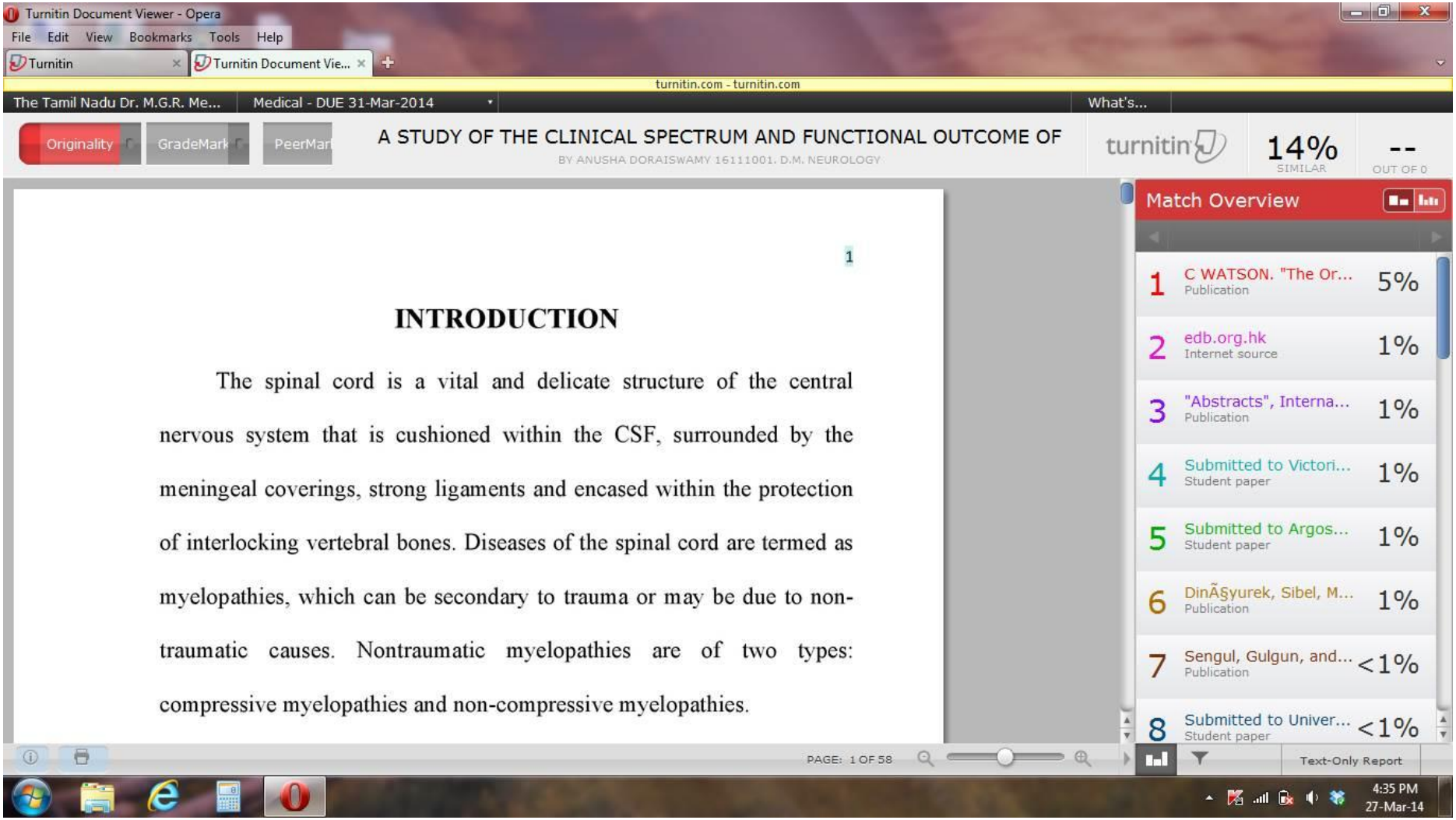
1.	Prof. R. Nandhini MD Director, Instt. of Pharmacology ,MMC, Ch-3	-- Member Secretary
2.	Prof. Reghu MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
3.	Prof. Shyamraj MD Director i/c , Instt. of Biochemistry , MMC, Ch-3	-- Member
4.	Prof. P. Karkuzhali. MD Prof., Instt. of Pathology, MMC, Ch-3	-- Member
5.	Prof. G.Muralidharan MS Prof of Surgery, MMC, Ch-3	-- Member
6.	Thiru. S. Govindsamy. BA, BL	-- Lawyer

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini 19/11/12
Member Secretary, Ethics Committee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INTRODUCTION
The spinal cord is a vital and delicate structure of the central nervous system that is cushioned within the CSF, surrounded by the meningeal coverings, strong ligaments and encased within the protection of interlocking vertebral bones. Diseases of the spinal cord are termed as myelopathies, which can be secondary to trauma or may be due to non-traumatic causes. Nontraumatic myelopathies are of two types: compressive myelopathies and non-compressive myelopathies.
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INTRODUCTION

The spinal cord is a vital and delicate structure of the central nervous system that is cushioned within the CSF, surrounded by the meningeal coverings, strong ligaments and encased within the protection of interlocking vertebral bones. Diseases of the spinal cord are termed as myelopathies, which can be secondary to trauma or may be also to non-traumatic causes. Nontraumatic myelopathies are of two types: compressive myelopathies and non-compressive myelopathies.

Myelopathies commonly present with motor and sensory deficits along with sphincter disturbances. The clinical presentation and causes of compressive myelopathies characteristically differ from those of non-compressive myelopathies, although rare presentations in either category can mimic each other [1] and pose a diagnostic dilemma to the astute clinician. The common causes of spinal cord compression are Pott's spine, fractures, infective abscess, arteriovenous malformations, spondylotic changes, spinal instability, tumours, multiple myeloma and metastases. The non-compressive myelopathies have wide and diverse etiologies like infective, inflammatory, demyelinating, vascular, hereditary causes or can be secondary to toxin exposure, metabolic disorders or nutritional deficiencies.