A STUDY ON THE CLINICAL, RADIOLOGICAL AND ETIOLOGICAL PROFILE OF NON-TRAUMATIC MYELOPATHIES IN SOUTH TAMILNADU

Dissertation submitted in partial fulfilment of the

Requirement for the award of the Degree of

DOCTOR OF MEDICINE

BRANCH I - GENERAL MEDICINE

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY,

CHENNAI,

TAMIL NADU

CERTIFICATE

This is to certify that the dissertation entitled "A STUDY ON THE CLINICAL, RADIOLOGICAL AND ETIOLOGICAL PROFILE OF NON-TRAUMATIC MYELOPATHIES IN SOUTH TAMILNADU" submitted by **Dr.Anu Elizabeth Mathew** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. degree Branch-I (General Medicine) is a bonafide research work carried out by her under my strict supervision and guidance during the academic year 2013-2016.

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DECLARATION

I, **Dr.Anu Elizabeth Mathew,** solemnly declare that, this dissertation **"A STUDY ON CLINICAL, RADIOLOGICAL AND ETIOLOGICAL PROFILE OF NON-TRAUMATIC MYELOPATHIES IN SOUTH TAMIL NADU"** is a bonafide record of work done by me at the Department of General Medicine, Tirunelveli Medical College, under the guidance of Professor **Dr.S.ALAGESAN M.D., D.M,** Department of General Medicine, Tirunelveli Medical college, during the academic year 2013-2016. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, and diploma to any other University, Board either in India or abroad.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Degree of Doctor of Medicine (M.D.), General Medicine Branch-I, examination to be held in April 2016.

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ABBREVIATIONS

ADEM	-	Acute Disseminated Encephalomyelitis
AON	-	Acute optic neuritis
APS	-	Anti Phospholipid Syndrome
ATM	-	Acute Transverse Myelitis
CMV	-	Cytomegalovirus
CIS	-	Clinically isolated Syndrome
CSF	-	Cerebrospinal Fluid
CV Jn	-	Craniocervical Junction
EMG	-	Electromyography
HIV	-	Human Immunodeficiency Virus
INO	-	Inter nuclear opthalmoplegia
IRIS	-	Immune reconstitution inflammatory syndrome
ITM	-	Idiopathic Transverse Myelitis
LETM	-	Longitudinally Extensive Transverse Myelitis
LMN	-	Lower Motor Neuron
MRI	-	Magnetic Resonance Imaging

MS	-	Multiple Sclerosis
NMO	-	Neuro Myelitis Optica
OCBs	-	Oligoclonal Bands
PCR	-	Polymerase chain reaction
PITM	-	Para infectious transverse myelitis
PLS	-	Posterolateral Sclerosis
SAIDs	-	Systemic Autoimmune Disorders
SCD	-	Subacute Combined Degeneration
SD	-	Standard Deviation
SLE	-	Systemic lupus erythematosus
STIR	-	Short Tau Inversion Recovery
UMN	-	Upper Motor Neuron

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NAME OF PRINCIPAL INVESTIGATOR: Dr. Anu Elizabeth Mathew, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate in MD General Medicine
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Dear Dr. Anu Elizabeth Mathew, *The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 14.05.14.*

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
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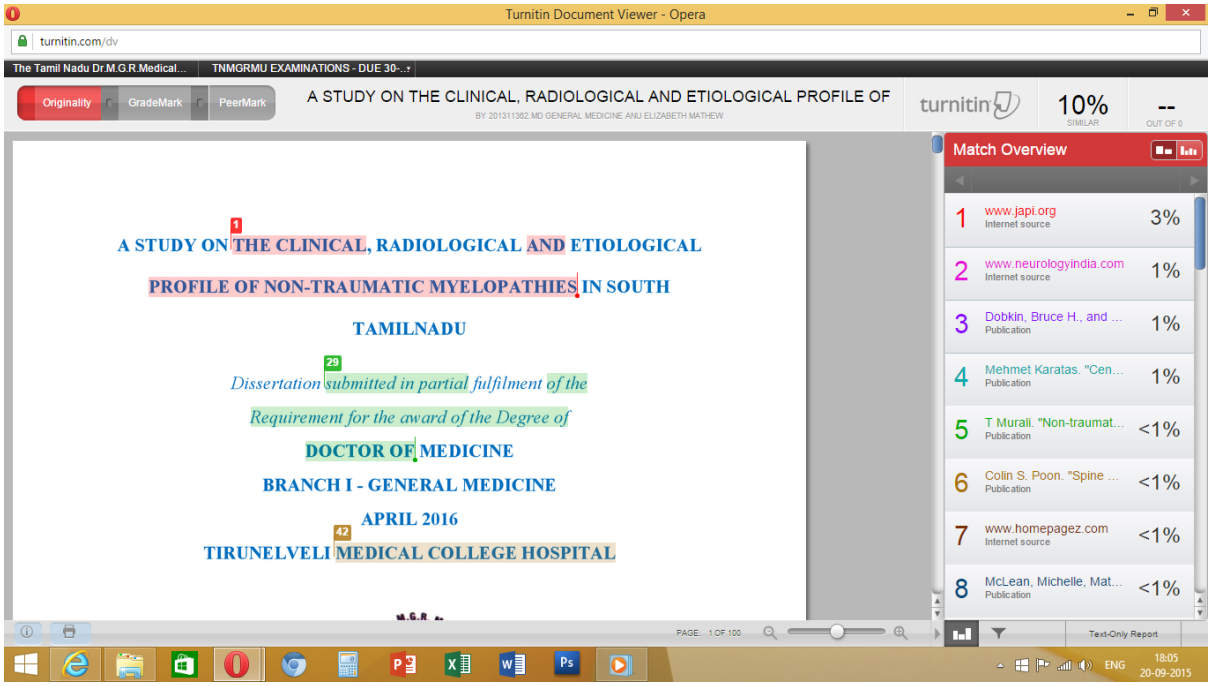
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INTRODUCTION

Quadriplegia and paraplegia resulting from non-traumatic myelopathy is a disabling and distressing neurological disease. The clinical presentation of spinal cord disease is varied. Myelopathies not only affects the motor, sensory and autonomic functions but also has serious psychosocial sequelae. The incidence of non-traumatic spinal cord lesions is difficult to determine because of the infrequent reporting. The causes can be classified as compressive and noncompressive. Among the compressive causes include pott's spine, tumors, disc prolapse, CV jn anomalies etc. Non compressive myelopathy encompasses a large range of disease entities ranging from demyelination, nutritional, toxic, infection, heredo-familial to degenerative conditions. Among this acute transverse myelitis has evoked considerable interest amongst neurologists as it strikes apparently healthy individuals in the prime of their lives, who are left with variable degree of sequale.

The disease spectrum is somewhat different in India as compared to western countries, where infections and nutritional causes are less common. The onset can be acute, subacute or insidious. In any case, they must be recognized as early as possible to prevent progression that can lead to permanent disability. Compressive lesions from neoplasms, degenerative disc disease, or infection may have to be managed surgically to relieve the cord compression in hopes of restoring normal function. Other primary neurologic diseases, such as multiple sclerosis, neuromyelitis optica, idiopathic transverse myelitis, and effects of infectious processes, may not be

amenable to surgical intervention. It is important to consider the age and gender of the patient when evaluating myelopathic patients. The temporal profile of the myelopathic features must be elucidated. Arriving at a diagnosis based on history and clinical examination alone may be difficult. With the advent of MRI which is a very sensitive imaging modality for lesions of the spinal cord, the yield for positive diagnosis has greatly increased. The incidence of non-traumatic spinal cord lesions is difficult to determine because of the infrequent reporting, but it is estimated to be equal to that of traumatic spinal cord injury[4]. Spinal tumors[1] and Pott's spine[2, 3]have been reported as the most common etiology of Non traumatic spinal cord lesions in different studies. Present study is aimed to identify the etiological profile of non-traumatic myelopathies of patients admitted in our Medical college Hospital which covers the population of South Tamil Nadu.

AIM OF THE STUDY

This study was aimed to identify the clinical and radiological profile of non-traumatic myelopathies in south Tamil Nadu population and the various etiologies associated with them.

REVIEW OF LITERATURE

The clinical presentation of spinal cord diseases is varied. The classical manifestation of spinal cord disease can confuse even the most astute clinician

An overview of the spinal cord anatomy

The spinal cord is elongated and nearly cylindrical, continuous with the medulla above and ending in a conical tip, the conus medullaris. The spinal cord occupies approximately the upper two-thirds of the vertebral canal, extending from the foramen magnum to a level that varies slightly from individual to individual but in adults lies between the lower border of L1 and the upper border of L2[5]. The filum terminale is a delicate filament of connective tissue that descends from the apex of the conus medullaris to the periosteum of the posterior surface of the first segment of the coccyx. The dentate ligaments extend along the lateral surface of the spinal cord, between the anterior and posterior nerve roots, from the pia to the duramater. They suspend the spinal cord in the vertebral canal. The general organization is the same throughout but there is some variability in detail at different segmental levels. The cord and vertebral column are of different lengths because of different fetal growth rates, so there is not absolute concordance between cord levels and vertebral levels; this discrepancy grows more significant at more caudal levels. Each spinal cord segment has anterior and posterior roots. The anterior roots convey motor and autonomic fibers into the peripheral nerve. Posterior roots bear ganglia composed of unipolar neurons, and the roots are made up of the central processes of these neurons.

The ganglion lies in the intervertebral foramen in close proximity to the anterior root. The anterior and posterior roots join just distal to the dorsal root ganglion to form the mixed spinal nerve. In the thoracolumbar region, white and gray rami connect the spinal nerve to the paravertebral sympathetic chain. The spinal cord ends in the conus medullaris. Roots from the lower cord segments descend to their exit foramina, forming the cauda equina.

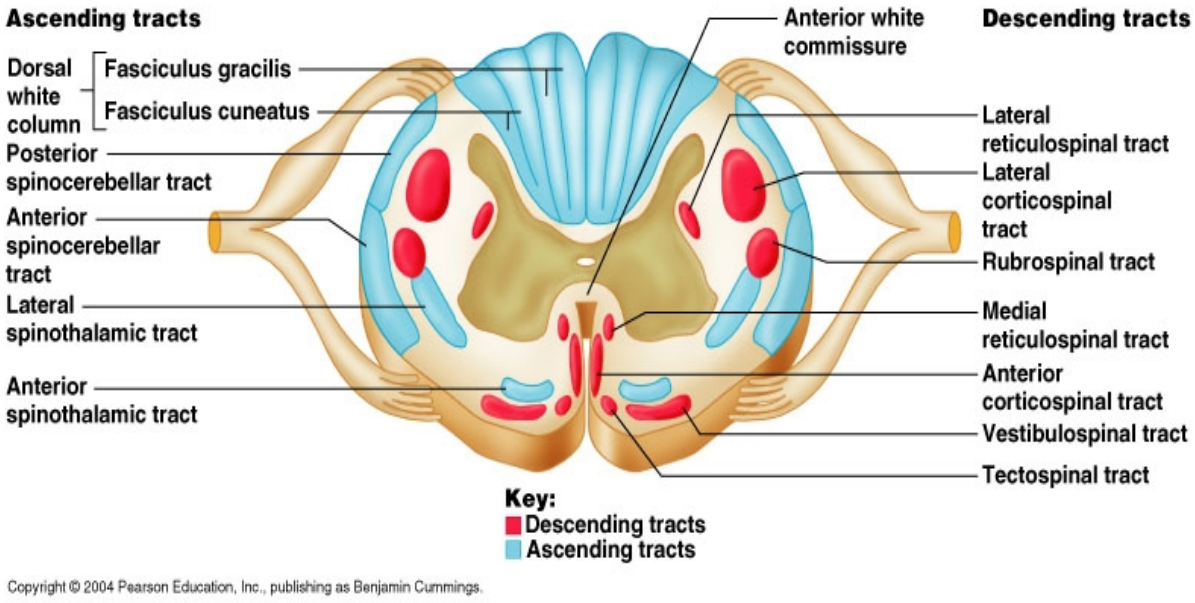


FIGURE: 1 Cord showing the cross section of the spinal cord with various descending and ascending tracts.

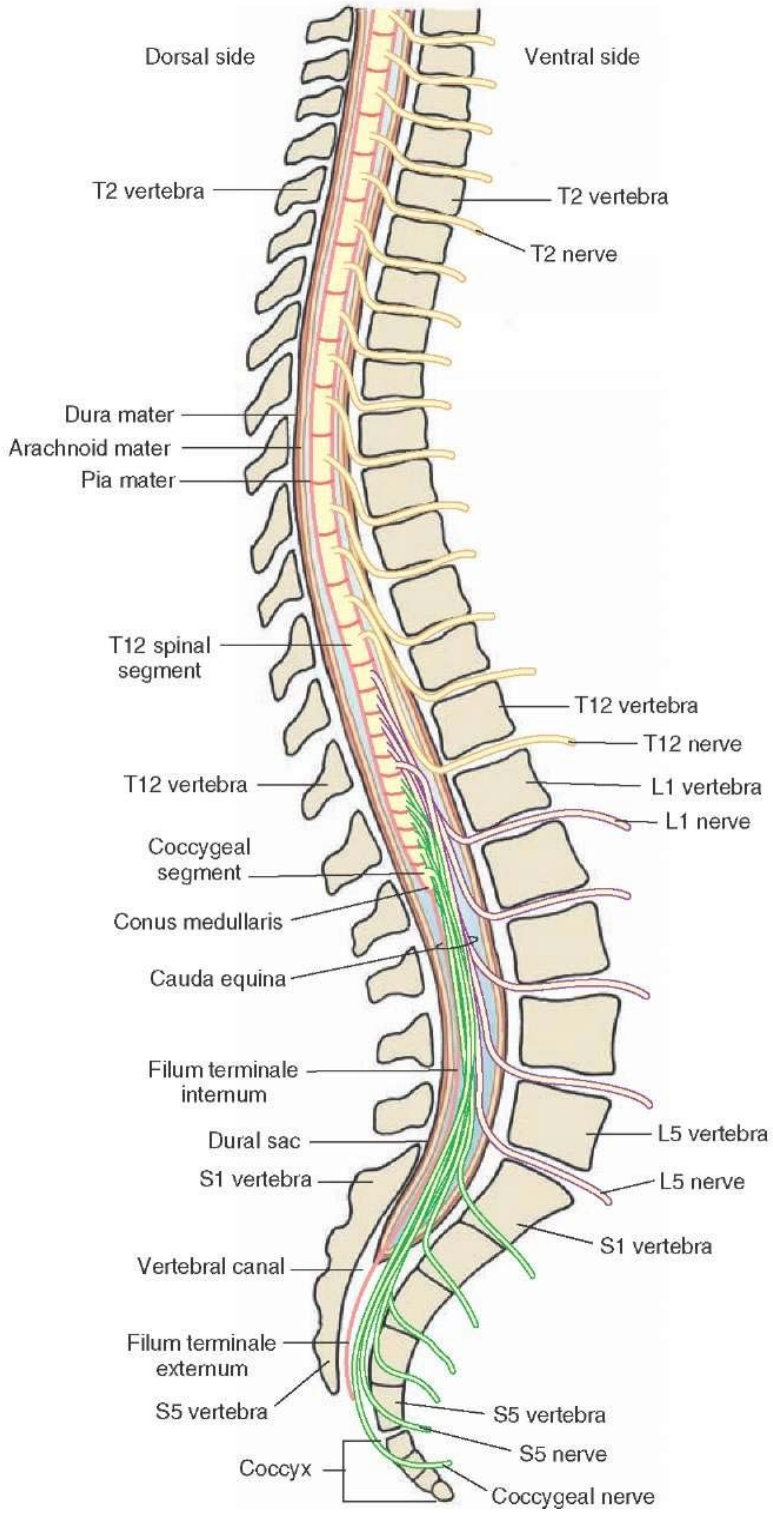


FIGURE: 2 Anatomy of the Spinal Cord

Common Spinal Cord Syndromes

Spinal Shock

A complete transverse cut of the cord results in complete loss of motor and sensory function below the level of lesion[6].

If the lesion is gradually developing, such as a benign neoplasm or cervical spondylosis, or if it's incomplete, then spinal reflexes such as exaggerated Deep Tendon Reflexes and Babinski's sign generally are present

On the contrary, if the lesion is sudden in onset, a condition known as spinal shock develops, in which there is transient loss of all spinal reflex activity below the level of the lesion along with motor paralysis and sensory loss.

Spinal shock is characterized by flaccid, areflexic paralysis of skeletal and smooth muscles. A total loss of autonomic functions occurs below the level of the lesion, which results in a loss of urinary bladder tone and paralytic ileus.

Because vasomotor tone is lost, dependent lower extremeties may become edematous and temperature regulation will be lost. Genital reflexes will be lost associated with total loss of sensation below the level of lesion

Incomplete lesions of the spinal cord

- Unilateral Transverse Lesion

A one sided lesion, otherwise known as hemisection of spinal cord produces a Brown Sequard Syndrome

In our daily practice, pure unilateral lesions are rare. The presentation of a patient with pure Brown Sequard's syndrome is that of ipsilateral weakness and loss of position and vibration below the level of the lesion, as well as contralateral loss of pain and temperature caudal to the lesion. Pain and temperature loss will manifest a few segments below the level of lesion[7].

At the level of lesion, there may be small area of anaesthesia, analgesia, and LMN weakness because the segmental afferent and efferent pathways are disrupted.

Trauma such as a bullet injury or stab wound is the most common cause of Brown Sequard Syndrome. Among the non-traumatic causes include spinal metastases and radiation necrosis

- Central Cord Syndrome

It is caused by an intra axial lesion disrupting the normal structures of the central or paracentral region of the Spinal Cord.

They can be either acute, which is usually caused by hemorrhage or contusion following trauma[8] or chronic, in which, cause could be a tumor or syringomyelia.

Contusions following trauma as well as syringomyelia, most commonly occur in the cervical spine and cervicothoracic junction. Spontaneous hematomyelia usually presents with acute onset of severe back or neck pain followed by paralysis.

When the cervical spine or cervicothoracic junction is the site of central cord syndrome, LMN type of weakness occurs in the upper extremities.

There is loss of sensation in the upper extremities of a dissociated type. This is caused by interruption in the decussating fibers. As a result of the lamination of the spinothalamic tract, sensation from the more caudal regions is preserved, with a cape like distribution of sensory loss with sacral sparing of pain and temperature

- Anterior Spinal Artery Syndrome

Infarction of the spinal cord has now become more common in recent years, partly because of the increased no: of invasive procedures such as vascular and thoracoabdominal surgery and revival after cardiac arrest and hypotension[9].

The anterior horns and anterolateral tracts are involved in this syndrome. The thoracic vascular watershed zone at about T6 is highly susceptible.

Corticospinal deficits develop below the level of infarction, also associated with dysfunction of autonomic pathways, causing bowel, bladder disturbances, sexual dysfunction, and a sensory disturbance develops in which posterior column function remains intact and the spinothalamic tracts are damaged. Initially, there is spinal shock with areflexia, which is followed later by spasticity. Anterior spinal artery

syndrome can be differentiated from acute central cord syndrome, by the sacral sparing that occurs in the latter.

- Anterior horn and pyramidal tract syndrome

Anterior horns and pyramidal tracts involvement with sparing of the sensory functions and autonomic nervous system are seen in motor neuron disease. Clinically, there is a combination of both LMN weakness with atrophy and fasciculation, fibrillation and denervation / renervation on electromyography, UMN signs with spasticity, exaggerated reflexes and Babinski's sign. Main diagnostic importance is the presence of LMN and UMN signs in the same muscle group. Alternatively, either the LMN or the UMN disturbance may predominate for months or years. Ultimately, as the LMN disease progresses, increasingly severe atrophy and progression from hyperreflexia to hyporeflexia occur.

- Combined Posterior and Lateral Column Disease

The clinical presentation is of loss of posterior column and lateral column. There will be sensory ataxia and may be bizarre in appearance. Even though Friedreich's ataxia may cause such a syndrome, the classical example is that of sub-acute combined degeneration of spinal cord caused by vitamin B12 deficiency[10].

CHARACTERISTIC CLINICAL FEATURES OF LESIONS AT DIFFERENT LEVELS

Spinal lesions at different levels often present with characteristic symptoms and signs referable to the involved segments. In extramedullary compression, disturbances at the segmental level herald the presentation .To the contrary, intramedullary lesions frequently do not present with segmental disturbances but with tract dysfunction.

Foramen Magnum

Foramen magnum lesions, which include trauma, tumors, syringomyelia, multiple sclerosis, atlanto-axial dislocation, Arnold – Chiari malformation, and bony abnormalities of the craniocervical junction, present a most challenging diagnostic problem for the clinician[11]. Neck pain or occipital pain, often increased by neck movement, is a common initial presentation. The pain often radiates to the shoulders or the same side arm. In the latter situation, the pain may simulate that of cervical spondylosis. Cranial nerve symptoms and signs are inconstant; nystagmus, often downbeating, impaired sensation over the upper face caused by involvement of the descending tract of cranial nerve V, and dysarthria, dysphonia, and dysphagia are present in some patients. Motor system involvement can present as spastic weakness. The corticospinal tract compression causes weakness that typically begins in the ipsilateral arm and involves weakness of the ipsilateral legs followed by weakness of the contralateral leg and then the arm.

Sometimes tumors of the foramen magnum tumors may cause signs of LMN like atrophy, and depressed reflexes in the arms and hands. The mechanism of this LMN disturbance below the level of the tumor is uncertain but possibly is secondary to circulatory disturbances affecting the distribution of the anterior spinal artery.

Sensory disturbances like pain and numbness are initial manifestations of foramen magnum tumors. Paresthesias and pain affecting the same upper limb first involved by spastic weakness is an early finding. The sensory disturbances are often of the dissociated type, so patients have preserved tactile sensation with loss of pain and temperature sensation. A suspended sensory loss also can occur or loss of vibration sense over the clavicles in others. This pattern may be due the secondary syrinx, which can direct attention away from the causative lesion at the cervicomedullary junction. Magnetic resonance imaging has become the test of choice for imaging of the cervicomedullary junction.

Upper Cervical spine

Compressive lesions at the cervical spine almost have similar characteristics to those at the foramen magnum. Pain in the neck, back of head, or shoulder is a common presenting complaint. With progressive compression, upper extremity weakness becomes apparent on the side of the pain[12]. When upper motor neuron findings develop in the leg, a spinal hemiplegia develops. Weakness can then progress to the contralateral lower extremity, and then the contralateral upper extremity.

Lower cervical and upper thoracic Spine

Spinal cord and root compressions at C5-T1 betray their presence by radicular symptoms at the affected level in the form of pain, later reflex, sensory and motor changes. With intramedullary neoplasms, pain is common but localization is diffuse and less typically radicular.

Thoracic levels

The thoracic dermatome landmarks that guide localization are nipple (T4), umbilicus (T10), inguinal ligament (L1). The relatively narrow vertebral canal and the vascular watershed area at T6 make the thoracic spinal cord segment extremely vulnerable to compression

Conus Medullaris and Cauda Equina

Lesions of the conus medullaris and cauda equina cause similar symptoms and signs including local, referred, and radicular pain, loss of buttock and leg sensations, leg weakness and sphincter disturbances.

Classification of diseases of the spinal cord

Differential diagnosis of diseases affecting the spinal cord[13]

1. Compressive lesions

- Non-neoplastic

  Trauma

Spondylosis

Spinal stenosis

Intervertebral disc herniation

Infectious disorders (eg. abscess, tuberculosis)

Inflammatory (e,g., rheumatoid arthritis, ankylosing spondylitis)

Syringomyelia

- Neoplastic

Epidural

Intradural extramedullary (e.g., meningioma, neurofibroma, and leptomeningeal metastasis)

Intramedullary

Non compressive myelopathies

Demyelinating

Viral myelitis

Vitamin B12 deficiency

Infarction

Toxic myelopathies

Auto immune diseases

Acute transverse myelitis of unknown cause

TRANSVERSE MYELITIS

Transverse myelitis includes a diverse spectrum characterized by acute or subacute spinal cord dysfunction resulting in plegia, a sensory level, and autonomic impairment below the level of lesion[14,15,16] .Etiologies of transverse myelitis are parainfectious, paraneoplastic, drug/toxin induced, systemic autoimmune disorders(SAIDs), and acquired demyelinating disorders like multiple sclerosis (MS) or neuromyelitis optica (NMO). Isolated transverse myelitis is a diagnostic dilemma, as it is common in both MS and NMO, but can also be the initial manifestation of SAIDs.

Clinical presentation

Age is an important consideration when evaluating myelopathies. Whereas older patients are likely to suffer from spinal cord infarction; Female patients are likely to suffer from transverse myelitis. Also the temporal profile is important, transverse myelitis typically has an acute to subacute onset, with neurological deficits reaching nadir within a few weeks. An apoplectic onset with deficits reaching the nadir in less than 4 hours indicates a vascular etiology. An insidious, progressive course in which the deficits continue to worsen beyond 4 weeks is not suggestive of Transverse Myelitis. Clinically, Transverse myelitis can present as one of the several syndromes of spinal cord. Acute complete Transverse myelitis (ACTM) manifests as paresis / plegia, sensory dysfunction and autonomic dysfunction below the level of lesion. Acute partial transverse myelitis (APTM) presents with asymmetric

manifestations or deficits pertaining to particular anatomic tracts; manifestations can be hemi-cord, central cord, or posterior column syndrome. Acutely, limb tone and muscle stretch reflexes may be diminished and even absent. Clinically spinal shock may persist for days to weeks, with a mean duration of 4to 6 weeks following an insult[17].

Some report a circumferential band of dysthesia, attributable to the dermatomes just rostral to the sensory level, around their trunk. Lhermitte phenomenon suggests an intrinsic cervical spinal cord lesion, typically affecting the dorsal columns.

Autonomic dysfunction is almost always present in the form of bladder, sexual, gastrointestinal, cardiovascular, and thermoregulatory functions.

An antecedent infection or prior vaccination suggests acute disseminated encephalomyelitis (ADEM) or parainfectious transverse myelitis. Women are at higher risk of acquired demyelinating diseases and SAIDs with the exception of Behcet disease and ankylosing spondylitis .A history of relapsing – remitting attacks of neurologic deficits, for e.g., acute optic neuritis (AON) or inter nuclear opthalmoparesis (INO), suggest Multiple sclerosis. NMO causes attacks of severe AON and brainstem lesions resulting in intractable nausea, vomiting, hiccups[18-21], attacks of NMO are more devastating[22]. Treatment with interferon beta 1-a would dramatically worsen NMO. Auto immune disorders, in particular SLE, Behcet disease, Ankylosing Spondylitis, Sjogren's syndrome and Antiphopholipid syndrome

(APS), are known causes of Transverse myelitis. In some cases Transverse myelitis may be the initial manifestation.

Evaluation and diagnosis

Magnetic resonance imaging (MRI) of the entire spinal axis is necessary to exclude any structural lesions, particularly those requiring emergent neurosurgical interventions. The most sensitive MRI sequence for detecting spinal cord sequence are short-tau inversion recovery (STIR) fast spin- echo and T2-weighted fast – spin sequences[23]. Based on the clinical and radiological data, Transverse myelitis can first be classified into longitudinally limited and longitudinally extensive Transverse myelitis (LETM). Longitudinally limited can be further classified as ACTM or APTM.ACTM causes a complete cord syndrome, and on axial section either cause a full thickness involvement or more involvement of the central cord. Patients with APTM are at increased risk of recurrence and transition to MS. On the contrary, patients with ACTM carries a lower risk of transition to clinically definite multiple sclerosis (CDMS) and is usually associated with other causes .LETM refers to lesions that extend over 3 or more vertebral segments; on axial sections, it typically involves more than two-thirds of the spinal cord thickness, maximally involving the central portion[24].

Serum vitamin B12 level, thyroid function tests, syphilis, and HIV serology should be obtained to check for potentially treatable cause of myelopathy. Vitamin E, serum copper, and ceruloplasmin levels are checked for those at risk of deficiency.

Serum paraneoplastic profiles should be performed in suspected cases of paraneoplastic Transverse myelitis. Cerebrospinal fluid (CSF) analysis is essential for all cases of transverse myelitis(TM).CSF cell count, differential, protein, glucose, oligoclonal bands (OCBs) and IgG index should also be obtained in all cases of transverse myelitis. OCBs are good in detecting the conversion to MS

An opthalmological evaluation is necessary to search for other diagnostic clues. Electrophysiological tests may be very useful in assessing patients with TM.EMG evidence of anterior horn involvement suggests worse prognosis for recovery. Somatosensory evoked potentials may offer evidence of a myelopathy in the presence of a normal spinal cord MRI.

Cause of Transverse Myelitis

Multiple Sclerosis

MS is a progressive neurologic disorder, first attacks of MS, called clinically isolated syndrome (CIS), usually consist of AON, APTM, or brainstem syndromes. Transverse myelitis in MS commonly presents with sensory phenomenon. MRI of the spine typically reveals an asymmetrically placed lesion less than 2 segments in length with a predilection for the cervicothoracic cord[16, 25].The important investigation that predicts the progression to CDMS is the brain MRI, followed by presence of OCBs in CSF. In patients with normal MRI, the presence of OCBs and or an elevated IgG index places a higher risk of developing MS

Neuromyelitis Optica

NMO is diagnosed based on the revised Wingerchuk[26] criteria requiring the presence of optic neuritis and TM as well as 2 of 3 of the following: NMO antibodies, LETM, and/or brain lesions inconsistent with MS.

Many autoimmune diseases can coexist with NMO leading to diagnostic confusion, which are Sjogren's syndrome, SLE, type1 Diabetes mellitus, ulcerative colitis, idiopathic thrombocytopenic purpura, myasthenia gravis, rheumatoid arthritis, celiac disease and Raynaud phenomenon. So it is necessary that all patients with SAIDs who present with TM undergo testing for NMO IgG

Parainfectious Transverse Myelitis (PITM)

Parainfectious TM refers to TM associated with antecedent infection. The antecedent event has typically resolved before the onset of TM and is therefore difficult to demonstrate the offending organism in the spinal cord parenchyma.

The hepatitis viruses may cause TM through post infectious, immune mediated, inflammatory mechanisms[27]. Hepatitis A virus and Hepatitis B virus infection have been associated with immune mediated Transverse Myelitis. Hepatitis C virus has been implicated as the most common Hepatitis virus in TM. The most common extra pulmonary manifestation of Mycoplasma pneumonia infection attribute to the CNS, of which transverse myelitis is the most debilitating manifestation

Campylobacter jejuni infection has classically been associated with GBS, due to the molecular mimicry between bacterial lipopolysaccharides and human gangliosides, but it has also been associated with transverse myelitis[28-30], & ADEM[31-33].

Paraneoplastic TM

Collapsin response mediator protein- 5 (CRMP 5 IgG) antibodies, seen in small cell lung, is the antibody most commonly associated with transverse myelitis. It usually presents with a subacute, progressive predominantly motor myelopathy with increased CSF protein, elevated IgG index with mild pleocytosis. MRI will show T2 hyperintense lesions with enhancement with gadolinium.

Idiopathic TM

The mean age of incidence is between 30 – 40 years, with female preponderance[34, 35]. The MRI usually demonstrates a central lesion, extending over 2 segments and extending more than two- thirds of spinal cord with predilection towards thoracic cord. ITM can recur in about one third of cases. Recurrence is more with male gender, age more than 50 years, negative CSF oligoclonal bands. They are associated with a poor outcome. The response to corticosteroid therapy is poor. One third of patients with idiopathic acute transverse myelitis recover with little or no sequelae

PseudoExacerbation

It is a phenomenom in which the symptoms worsen temporarily; in demyelinating disorders, Uhthoff phenomenon is the common underlying cause of pseudoexacerbation. Increased body temperature for e.g. in hot weather, hot baths, febrile illness, stress, dehydration can result in pseudoexacerbation .Even metabolic or physiologic abnormalities can worsen prior neurological deficits. Thus worsening does not always indicate relapse and the treatment should be directed against the cause (e.g. treating the UTI)

Management

Once transverse myelitis is diagnosed, immunotherapy should be started to retard the inflammatory process. Although randomized trials are lacking, high dose iv corticosteroids should be started as soon as possible in all cases of transverse myelitis[36]. If no response to steroids, plasmapheresis should be initiated; with the rationale of removing the humeral factors causing Transverse myelitis. The regimen consists of 1.5 plasma volumes for five treatments over ten days[37].

In lesions extending into the medulla, respiratory failure can occur and therefore respiratory function should be aggressively monitored. In cases of TM caused by autoimmune diseases, starting of long term immunomodulatory therapies would help in preventing future attacks

Neurorehabilitation

Successful neurorehabilitation requires a multidisciplinary approach that incorporates a goal directed program tailored for the patient's needs, through assessment by a general physician, physiotherapist, speech therapist, occupational therapist and psychologist

Bladder Dysfunction

Bladder dysfunction is one of the most disabling consequences of transverse myelitis; among it urinary tract infection is the most common medical problem in myelopathic patients. Three forms of bladder dysfunction occurs in transverse myelitis which are detrusor overactivity, detrusor sphincter dyssynergia and hypocontractile bladder. In acute TM, urinary retention occurs from a "shocked" bladder often necessitating bladder catheter insertion. Later on it is followed by detrusor hyperreflexia characterized by frequency, urgency, and urge incontinence. Treatment options available for detrusor sphincter dyssynergia are alpha 1 adrenergic antagonist, clean intermittent catheterization, neuromodulation etc.

Neurogenic bowel Dysfunction

Bowel dysfunction is a source of considerable psychosocial disability, affecting the quality of life. It can manifest as fecal incontinence or constipation. Psychiatric disturbances and medications also contribute to bowel dysfunction.

Autonomic Dysregulation

Autonomic dysfunction can occur in both the acute and chronic phases of Transverse myelitis, and is present in lesions above the upper thoracic segments.

Orthostatic hypotension

Orthostatic hypotension occurs in both the acute and chronic stages of Transverse myelitis. It is defined as a drop in systolic blood pressure of 20 mm Hg or more or a drop in diastolic blood pressure of 10 mm Hg or more when the subjects stands from a supine position. This occurs due to loss of reflex vasoconstriction due to loss of sympathetic nervous activity leading to pooling of blood in the abdominal organs and legs, ultimately leading to reduced cardiac output

Vascular Diseases of the Spinal Cord

Vascular diseases of spinal cord are rare, when compared to cerebrovascular events, can cause significant neurologic morbidity. It includes structural causes like infarction, dural arteriovenous fistula, arteriovenous malformation, hematomyelia, inflammatory causes like primary and secondary vasculitides and genetic abnormalities. Several vascular spinal disorders present as neurologic emergencies.

Spinal Cord Infarction

Spinal cord infarction is a rare cause of acute myelopathy, representing only 1% of all strokes and constitutes 5 % of all myelopathies[38]. Syphilitic arteritis was the

most common cause of spinal cord infarction in the early twenthieth century[39] which is now replaced by atherosclerotic disease and surgery of aorta[40-43]. Other reported causes include decompression sickness[44], systemic hypotension[45], spinal trauma, rare causes include vertebral angiography[46], sympathectomy[47], abdominal aortography[48], lumbar epidural anesthesia[49], single radicular artery ligation, renal artery embolization, intra-aortic balloon pump counterpulsation[50], portocaval shunt placement

Clinical presentation

Patients presents with acute weakness, urinary retention, and pain, in the descending order of frequency across series[38, 40, 41, 43]. The most common site involved is the thoracolumbar region followed by the mid thoracic segment. Weakness typically tends to progress over minutes to hours; nadir is reached within 12 hrs of symptom onset.

Anterior spinal artery syndrome is clinically characterized by rapid onset of symmetric motor weakness and spinothalamic sensory deficit below the level of lesion in association with autonomic involvement. The weakness is often flaccid associated with absent tendon reflexes, showing the involvement of anterior horns. Patients may experience respiratory distress (phrenic nerve palsy, C3- C5), orthostatic hypotension (greater splanchnic nerve palsy, T4 to T9), and urologic dysfunction[51]

Effective treatment of spinal cord injury depends on rapid diagnosis. During the perioperative stage, management starts with serial neurologic examinations

beginning soon as one gets up from general anaesthesia. If spinal cord injury is suspected, the mean arterial pressure should be maintained above 90 m Hg, using hemodynamic augmentation with volume and/ or pressors. Emergent neuroimaging of the spine should be done to rule out compression of spinal cord particularly from epidural hematoma. MRI is he preferred modality. Diffusion weighted imaging has now become increasingly popular for the diagnosis of spinal cord infarction[52], although it is less sensitive s that of brain. MRI can also be normal in acute stages, so absence of any abnormalities should not dissuade from the diagnosis. If there is no clinical improvement after hemodynamic augmentation, placement of a lumbar drain should be considered to maintain cord perfusion.

Prognosis

Spinal cord infarction is immediately life threatening, but a significant minority of severely affected patients can have a good outcome. Severity of the impairment and the presence of peripheral vascular disease were independently associated with poor prognosis after spinal cord infarction.

Metabolic, Nutritional, and Toxic Myelopathies

Disorders affecting the spinal cord can either occur acutely or can be insidious in onset. Various nutritional, metabolic, and toxic causes can cause myelopathy and myeloneuropathy, and requires a different approach to diagnoses and treatment[53].

Vitamin B 12 deficiency

Leichtenstern[54] and Lichtheim[55] first described pathologic abnormalities in the dorsal and lateral columns of the spinal cord in patients with megaloblastic anemia in the late 19th century. Neurologic deficits of pernicious anemia were well described at that time and included subacute combined degeneration of spinal cord (SCD), peripheral neuropathy, cognitive symptoms as well as dementia.

Vitamin B12 deficiency can produce overlapping clinical syndromes of peripheral neuropathy, SCD, optic atrophy, autonomic symptoms, mood and behavioral changes, psychosis and dementia. The neurologic symptoms are usually insidious in onset and consist of only vague symptoms, such as fatigue and generalized weakness. Autonomic symptoms consist of urinary frequency, constipation, or erectile dysfunction in men. Gait abnormalities usually occur and the presence suggest sensory ataxia. In addition to that, there may be spasticity, hyperreflexia, loss of position and vibration sense, and presence of pathologic reflexes. Distal paresthesias are suggestive of peripheral neuropathy. SCD can also associated with megaloblastic anemia with raised mean corpuscular volumes and hyper segmented polymorphonuclear leukocytes[56].

The diagnosis depends on a high index of suspicion. A Low serum cobalamin levels is all that is needed for diagnosis. Homocysteine and methylmalonic acid levels are elevated in about one third of patients with normal levels of cobalamin. Intrinsic factor and parietal cell antibodies may be done if there is associated anemia. MRI of

the lower cervical and thoracic spinal cord may show increased T2 – weighted signal in the posterior and lateral columns of the spinal cord. Cobalamin deficiencies occur in conditions such as pernicious anemia, malabsorption syndromes, gastric surgery, H2 antagonists and metformin, parasitic infestation by fish tapeworm.

Treatment is with high doses of cobalamin given intramuscularly. Exposure to Nitrous oxide can give rise to a similar myelopathic picture in individuals with mild cobalamin deficiency. It interferes with the metabolic pathway that produces methionine synthase, which is vitamin B12 dependent. This leads to loss of myelin cohesion and vacuolization of the spinal cord. MRI shows T2 hyperintensities in the posterior and lateral columns. Treatment is with large doses of cobalamin.

FOLATE DEFICIENCY

Folate deficiency can also cause myelopathy, peripheral neuropathy, optic atrophy, and cognitive problems. Folate deficiencies can as a result of gastrointestinal disease, alcoholism, and drugs such as methotrexate and trimethoprim.

COPPER DEFICIENCY

Copper deficiency produce similar neurologic manifestations as subacute combined degeneration, patients present with gait abnormalities related to a sensory ataxia and spasticity due to posterior and lateral column involvement. Paresthesias in the hands and feet are common. The diagnosis of myelopathy related to copper deficiency depends on demonstration of low serum copper and low ceruloplasmin levels. MRI shows abnormalities with increased signal intensities in the posterior and

lateral columns. The abnormalities are seen in cervical cord and are similar to those seen in cobalamin deficiency[58].

The causes of copper deficiency seem to be varied. The most common cause is due to abnormalities in copper absorption as in those who have undergone previous gastric surgery, particularly bariatic surgery. Treatment consists of supplementation of elemental copper that is given orally beginning with 8 mg/day for one week, 6 mg/ day for one week, and maintenance on 2 mg/day.

VITAMIN E DEFICIENCY

Vitamin E is absorbed in the intestine as alpha tocopherol and is bound to the alpha tocopherol transport protein. In adults, deficiency is due to malabsorption syndromes such as celiac disease, cystic fibrosis, cholestasis and various other disorders.

Neurologic manifestations of vitamin E deficiency vary and include spinocerebellar syndromes and peripheral neuropathy resulting in gait abnormalities, decreased tendon reflexes, impairment of position and vibratory sensation, gaze palsies, and retinopathy. Diagnosis is based on low vitamin E levels. MRI will show hyperintensities in posterior columns. Treatment is by replacement of Vitamin E daily at a dose of 800 to 1200 mg/day.

TOXIC MYELOPATHIES

Exposure to various toxins can result in myelopathy. Two of these in less developed countries are lathyrism and konzo. Lathyrism is caused by atoxic amino acid, beta- N- oxalylamino- L- alanine contained in the grass pea, Lathyrus sativus. This toxin causes irreversible spastic paraparesis by causing degenerative changes in the spinal cord. Examination may show spasticity of limbs with hyper reflexia that causes a spastic gait disorder. The condition can be prevented by avoiding consumption of the chicking pea and combining it with other cereals.[59]

MYELOPATHY RELATED TO MEDICATIONS, OTHER TOXINS

Various drugs and chemicals have been implicated to cause myelopathy and myeloneuropathy. Organophosphate poisoning is known to cause myelopathy[60]. They are used as pesticides and are available in many home settings. A major chemical attributed for causing these is triorthocresyl phosphate. This compound has been used as an adulterant in various cooking oils. Myelopathic symptoms start to occur late in the course of the pathologic state. The signs and symptoms of acute organophosphate poisoning is followed by a period of latency, after which a progressive phase may follow, during which patients starts developing signs and symptoms of motor – sensory neuropathy in the extremities. A stationary phase then follows after there is evidence of spasticity with paraparesis and quadriparesis. There is usually some delay in the onset of disease after exposure to organophosphates. Measurement of cholinesterase activity in RBCs can help confirm the diagnosis.

Various chemotherapeutic agents are known to cause myelopathy and neuropathy. Drugs include cisplatin, cladarabine, vincristine, cytosine arabinoside, and intrathecal methotrexate. Administration of these drugs intrathecally can cause myelopathy either related to the agent itself or to preservatives and diluents used in these agents. Another entity is the radiation myelopathy, which occurs months to years after radiation that involves the spinal cord.

Heroin abuse can lead to myelopathy[61]. The onset is acute, with evidence of T2 weighted hyperintensities in the spinal cord resembling that of transverse myelitis. The pathophysiological mechanism causing this is not known, but could be due vasculitis, direct toxicity, or hypersensitivity reaction.

TABLE: 1MRI findings of clinical condition

Clinical condition	MRI findings
Vitamin B12 Deficiency Myelopathy	T2 hyper intensities in the posterior and lateral columns more in the cervical and upper thoracic cord
Nitrous oxide intoxication	Similar picture as above
Copper deficiency Myelopathy	T2 hyperintensities involving posterior columns.
Heroin induced Myelopathy	Cord hyperintensities in T2 and Flair affecting posterior and lateral columns, lesions in pontomedullary region and in ventral pons.
Organophosphate poisoning	Atrophy of spinal cord
Hepatic myelopathy	Symmetric demyelination of lateral corticospinal tracts, spinocerebellar tracts and posterior columns
Vitamin E deficiency	T2 hyperintensity in posterior columns with cerebellar atrophy
Lathyrism	No specific MRI findings

Spinal Cord Tumors

Spinal cord tumors are uncommon neoplasms that without treatment, can cause significant neurologic morbidity and mortality. The classification of spine tumors is based on the use of myelography with three main groups 1) Extramedullary extradural 2) Intradural extramedullary 3) Intradural intramedullary. Spinal tumors are classified either inside the dura or outside[62].

The most common primary extramedullary neoplasms are neurofibromas and meningiomas, which together constitute about fifty percent of all intraspinal neoplasms[63]. They are often intradural than extradural. Neurofibromas have a predilection for thoracic region and meningiomas are more evenly found over the vertical extent of the cord. Sarcomas, vascular tumors, chordomas, and epidermoid tumors are the other primary extramedullary tumors. Primary intramedullary tumors of the spinal cord have the same cytology as that of primary brain tumors, but they vary in their proportion. Astrocytomas constitute the majority of intramedullary spine tumors, if one excludes tumors arising from filum terminale which are formed by ependymomas. Of the remaining, hemangioblastomas account for about 2 to 7 % and the minority being intramedullary metastases. Intramedullary growths invade as well as distort the tracts in the spinal cord white matter. As the cord is compressed by the invading tumor from within or without, the free space in the cord is thereafter consumed, causing the CSF below the lesion to be isolated from the remaining circulating fluid above the lesion. There is xanthochromia and clotting of CSF known

as Froin syndrome. The most useful imaging modality is the MRI, which gives information about the site of the tumor and its extension into the subarachnoid space

Secondary spinal cord tumors can also be classified into intramedullary and extramedullary types. Extradural metastases eg carcinoma, lymphoma, myeloma are the most common of all spinal tumors. Metastases at extradural site arise from either hematogenous deposits or extension from tumors of the vertebral bodies or extension of paraspinal tumor via intervertebral foramina. The intradural type takes the form of a meningeal carcinomatosis or lymphomatosis.

Intramedullary metastases are not uncommon. In a retrospective study done by Costigan and Winkelman in patients in patients with systemic cancer[64], Bronchogenic carcinoma was the main source. Diagnosis is aided by the MRI imaging which shows extensive contiguous edema. Differentiation is from meningeal carcinomatosis, paraneoplastic necrotizing myelopathy and radiation myelopathy.

Clinical symptoms

Patients with spinal cord tumors present with any of the three spinal cord syndromes

1) Sensorimotor spinal tract syndrome
2) A painful radicular – spinal cord syndrome
3) Intramedullary syringomyelic syndrome.

Pain in the back may be the first symptom or dominate the clinical picture in some cases of extramedullary neoplasms. The pain is usually worse on lying down or after several hours after assuming recumbent posture.

Sensorimotor spinal tract syndrome

The signs of compression consist of an asymmetric spastic paraparesis with involvement of arms with cervical lesions[63]. There will be a sensory level below which the sensations will be lost along with posterior column signs and finally a spastic bladder with weak voluntary control. The onset of compression is often gradual and the course progressive over a period of weeks or months, frequently with back pain.

In extradural lesions weakness usually develops over a period of days to weeks, but can progress in a more rapid manner. The disturbance can be motor or sensory and the distribution asymmetrical. With thoracic lesions one leg usually becomes weak and stiff followed by the other leg. Pain and temperature sensations are affected more than tactile sensations. Bladder and bowel gets involved concurrently with paralysis of the legs. Recovery from the motor and sensory symptoms is usual, if the compression is relieved, often in the reverse order of appearance

Radicular- spinal Cord syndrome

Here there is associated radicular pain, described as dull aching with superimposed sharp stabs or knife like which radiate in the distal direction, intensified

by coughing, sneezing, or straining. Percussion tenderness over the growth is found in about half of the cases.

Intramedullary Syringomyelic Syndrome

Intramedullary tumors can present nonspecific symptom. Pain in the back is common and is invariably present in the tumors of the filum terminale. Ependymomas and astrocytomas, which are the two most common intramedullary tumors, give rise to a mixed sensorimotor tract syndrome. When the tumor involves the central gray matter, a central cord, or *syringomyelic syndrome*[65]can result. The main features are segmental or dissociated sensory loss, anterior horn cell involvement leading to atrophy, early incontinence, and late corticospinal tract involvement. On sensory examination, there may be sacral sparing but is of much less value in distinguishing from extramedullary lesions. The dissociated sensory loss over several segments on the trunk is a more reliable sign of an intramedullary lesion. Rarely, an extramedullary tumor may give rise to syringomyelic syndrome, probably by causing vascular insufficiency in the central portion of the cord.

Special Spinal Syndromes

Tumors of the foramen magnum can produce unusual spinal cord syndromes. They produce a quadriparesis associated with pain in the back of the head and stiffness of neck, weakness and atrophy of the muscles of the hand and dorsal neck muscles, marked unsteadiness, and variable sensory changes. If they spread intracranially, there may be lower cranial nerve involvement along with cerebellar signs. Slowly growing tumors such as meningiomas characteristically produce a clockwise

progression of weakness beginning in one limb and proceeding to the adjacent one. Tumors at the level of the lower thoracic and the first lumbar vertebrae may result in mixed cauda equina and spinal cord syndromes. An extensor plantar response indicates that the spinal cord is involved above the fifth lumbar segment. Lesions of the cauda equina, which is usually difficult to distinguish from those of the lumbosacral plexuses presents in the early stages by sciatica and low backache, which is often associated with a bilaterally asymmetrical, areflexic paralysis, segmental sensory loss and sphincteric disturbances. These must be distinguished from lesions of the conus medullaris(lower sacral segments of the spinal cord), in which there are early involvement of bladder and bowel (urinary retention and constipation), backpain, symmetrical sensory loss over the sacral dermatomes, lack of tone in anal sphincter with absent anal and bulbocavernosus reflexes, impotence, and sometimes weakness of leg muscles. Sensory disturbances may precede motor and reflex changes by months. Very rarely, for unknown reasons, tumors of the thoracolumbar cord, invariably intramedullary may be associated with markedly increased spinal fluid protein and hydrocephalus; these respond to shunting and removal of the spinal tumor[66].

Differential Diagnosis

To arrive at a diagnosis of spinal tumors by clinical grounds alone is difficult. In their initial stages they must be differentiated from other diseases that cause pain over certain parts of the body, i.e., diseases affecting the gallbladder, kidney, pancreas, stomach, intestinal tract, pleura, etc. Pain localized to a dermatome;

increasing on sneezing, coughing, and straining, and the finding of segmental sensory changes and alteration in motor, reflex, or sensory function in the legs will provide clues to the presence of a spinal cord radicular lesion. MRI will aid in the final diagnosis. The segmental level of lesion should be found out. Initially, the sensory and motor deficits may be most pronounced in the parts of the body farther away from the lesion, i.e., in the feet for lumbosacral segments. Later the levels ascend, but they will still be at a level few segments below the lesion. In determining the level of the lesion, the location of back pain, root pain, and atrophic paralysis are of great help than sensory level. After the vertebral and segmental levels of the lesion are found, next step is in determining whether the lesion is extradural, intradural-extramedullary, or intramedullary. In the presence of a visible or palpable spinal deformity or radiographic evidence of vertebral destruction, we can assume the lesion as extradural or if there is a root involvement and motor change preceding the sensory loss with late bladder involvement. Distinguishing between intradural extramedulary and intramedulary tumor on clinical grounds alone is often difficult.

Extradural tumors, including both primary and secondary, must be distinguished from cervical spondylosis, tuberculous granuloma, sarcoidosis, arteriovenous malformations of the cord and certain chronic pyogenic or fungal granulomatous lesions. In the thoracic region, a ruptured disc or eventration of the cord through a dural tear may be possibility. In the lower back, i.e., over the cauda equina, one should also distinguish between a tumor and protruded intervertebral disc.

With intradural extramedullary lesions, the important diagnostic considerations are neurofibroma, meningioma, meningeal carcinomatosis, cholesteatoma, a meningomyelitic process, or adhesive arachnoiditis. CSF cytology studies and imaging with MRI are the essential laboratory aids. Intramedullary lesions are mostly gliomas, ependymomas, or vascular malformations or, in the background of a known carcinoma, intramedullary metastases.

Treatment

Treatment depends on the nature of the tumor and the clinical condition of the patient. Early diagnosis is needed as once neurological symptoms occur, it portrays a poor prognosis.

TABLE 2

Cord syndromes caused by spinal cord tumors

Complete cord transection	Metastatic epidural disease, intramedullary cord metastasis, pathologic fractures
Hemitransection of the cord	Astrocytoma, ganglioglioma, nerve sheath tumor, meningioma, and hemangioblastoma
Ventral cord syndrome[67]	Anterior epidural metastatic disease, radiation myelopathy, astrocytoma
Central cord syndrome	Intramedullary astrocytoma, ependymoma, metastases
Posterior cord syndrome	Epidural metastases, hemangioblastomas, astrocytoma
Conus medullaris syndrome	Ependymoma, syringomyelia, lymphoma, astrocytoma
Cauda equina syndrome	Nerve sheath tumors, leptomeningeal disease, myxopapillary ependymoma, paraganglioma, epidural metastasis,meningiomas[68,69]

SPINAL CORD INFECTIONS

Suspecting Spinal Cord Infection: A Diagnostic Approach[70]

The Physician approaching patients with a potential spinal cord infection should follow a systemic strategy to optimize appropriate choice of diagnostic and therapeutic options.

Demographic clues

Demographic clues help to generate an initial approximation of possible pathogens in vulnerable patient populations

Pace of illness

Pace of illness in spinal cord infections can be acute, subacute, or chronically progressive but usually not episodic or fluctuating, the latter occurring with cases of recurrent herpes simplex virus 2 myelitis or subarachnoid neurocysticercosis. Compressive myelopathies such as those associated with tuberculosis (TB) or pyogenic epidural abscess may appear to have an acute onset, but detailed historical inquiry discloses a more indolent pain syndrome preceding neurological deterioration. Chronic presentations characterize a wide range of infections, including human immunodeficiency virus (HIV), human T-lymphotropic virus 1 (HTLV-1), syphilis, hepatitis C. The list of organisms causing acute flaccid paralysis includes polio virus 1/2/3; Coxsackie A and Enteroviruses 70, 71, 93 and 94; varicella- zoster virus; and syphilitic arteritis etc.

Physical examination should be directed to the systemic manifestations of systemic infection that prompts investigation for an infectious cause.

Imaging

The initial imaging of choice in any spinal cord infection will be MRI. Normal spinal MRI is often seen in many spinal infections[71]. Abnormal MRI is almost always seen in spinal cord neurocysticercosis, schistosomiasis, Aspergillosis, Tuberculosis and pyogenic epidural abscess.

Serum and CSF Diagnostic Testing

In general, the presence of pleocytosis greater than 50 leukocytes per microlitre raises suspicion of a possible infection. Although a predominantly neutrophilic pleocytosis enhances suspicion of infection, this can also occur in noninfectious myelitis such as neuromyelitis optica. The absence of white blood cells in CSF does not exclude infection. The sensitivity of CSF tests depends on proper test choice with respect to the onset of illness. A reasonable wide spectrum screening includes CSF PCR testing for CMV, varicella zoster virus, herpes simplex virus-1 and 2, and cryptococcal antigen and blood testing for HIV, syphilis etc.

Spinal cord infections consideration in at- risk patient populations

Infectious myelopathies in patients with cancer

Cancer and its treatment pose an array of noninfectious differential diagnostic concerns that complicate the evaluation for potential spinal cord infection. These concerns include

1. Recurrence of disease or spread to epidural space from bony metastases
2. Treatment related myelopathies that mimic infection, such as radiation myelitis or superficial siderosis, and toxicity from chemotherapeutic agents, such as cytarabine, methotrexate, thiotepa, and cisplatin.[72]
3. Paraneoplastic myelopathies with selective spinothalamic tract or dorsal column dysfunction

HIV/AIDS

Patients with HIV are susceptible to a variety of spinal cord pathogens.

Clinical challenges includes:

1. Atypical CSF cell counts: Patients with very low CD4counts will not be able to mount an appropriate CSF pleocytosis, and spinal fluid may show unusual differential count, or total absence of cells[73]
2. Impaired humoral response causes insensitive serologic titres.
3. Neuroimaging may prove difficult to interpret because HIV patients have a higher risk for CNS neoplasms, including lymphoma and metastatic disease.

SELECTED SPINAL INFECTIONS

Bacterial infections

Spinal epidural abscess

Spinal epidural abscess is a medical and sometimes surgical emergency. Risk factors include recent barrier breach, such as IV drug abuse, spinal surgery, intrathecal pumps, and skin and soft tissue infections, and systemic conditions, such as diabetes mellitus, renal failure, alcoholism, and bacteremia/endocarditis. The main routes of infection are hematogenous dissemination or contiguous spread from osteomyelitis or muscle or soft tissue abscess[74,75].

Clinical presentation includes back pain that usually only lasts a few days but may be more indolent. There is often fever and elevated white blood count, C- reactive protein and erythrocyte sedimentation rate. Common organisms are S aureus, gram negative rods, streptococci, and mycobacterium tuberculosis. Antibiotic coverage should target gram positive organisms with vancomycin and gram- negative bacilli with piperacillin-tazobactum, cefotaxim, and meropenem. Negative prognostic features include MRSA infection, motor deficits, and age older than 50 years, cervical and thoracic level involvement, delayed diagnosis, concurrent sepsis, diabetes, and prior spinal surgery. Distinction between dorsal or ventral spinal epidural abscess is important; the former causes compression earlier and warrants surgical intervention whereas ventral abscesses responds well to conservative antibiotic management.

TB Spine

Potts disease or tuberculous spondylitis, is the most common skeletal manifestation of TB. Potts disease is increasing in prevalence due to development of drug resistant strains, and /HIV infection[76].

Tuberculous spondylitis can cause neurologic symptoms via direct extension from the vertebrae to the spinal cord[77-79].

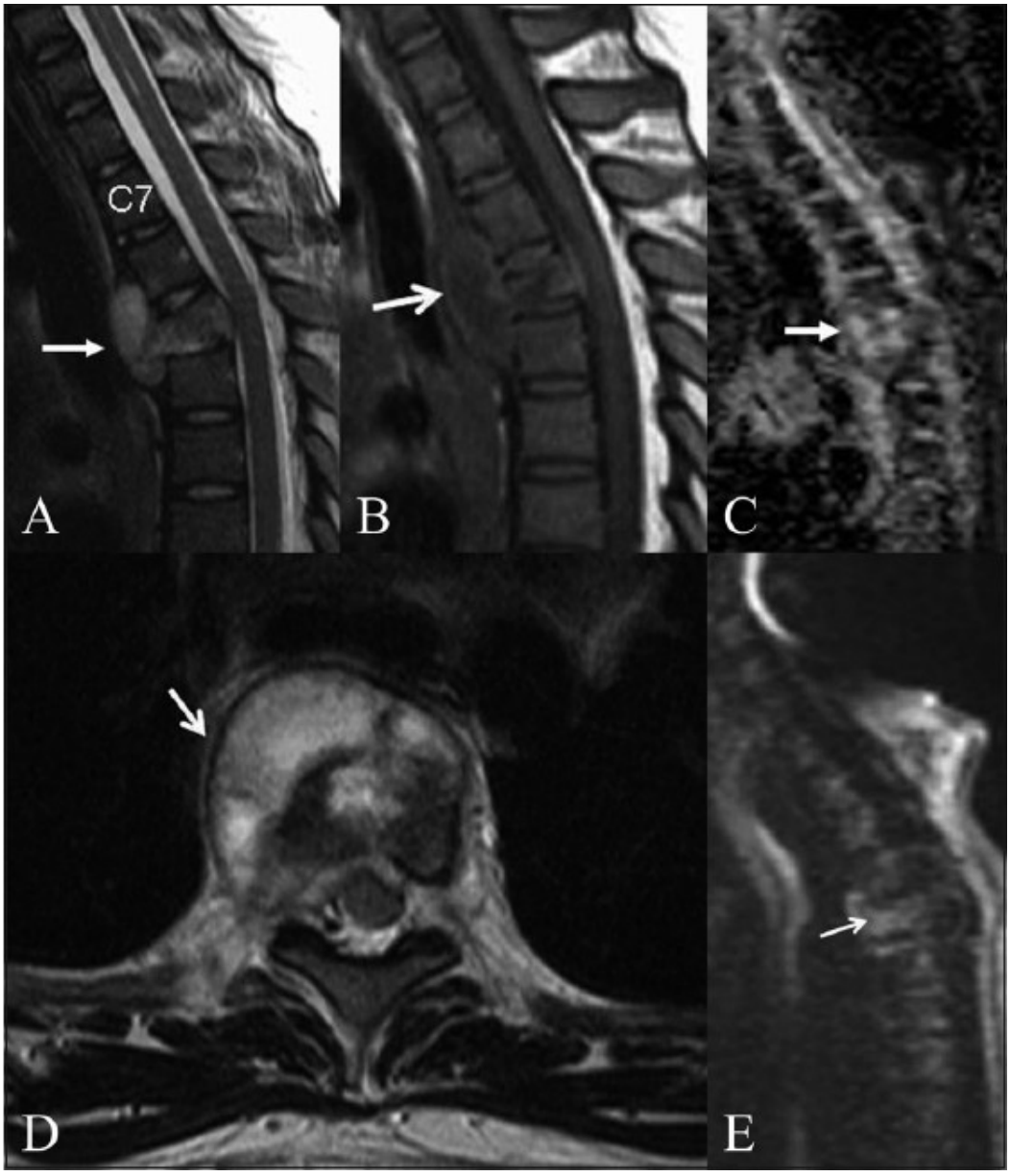


FIGURE: 3 Tuberculosis of the spine. T2W (A), T1W (B) axial MRI images in a patient with upper dorsal spine tuberculosis show a small prevertebral abscess (arrow). Wedging of the D2 vertebral body is seen with marrow involvement. Sagittal ADC map (C) sagittal and T2W (D) and diffusion (E) images show increased diffusion (arrow) in the involved vertebra (ADC: 1.35 × 10– 3 mm2/s).

In the absence of skeletal lesions, Tuberculosis can appear as simultaneous spinal cord and root inflammation and spinal arachnoiditis is the most common

primary cord manifestation of TB.CSF is typically inflammatory with parameters in the range of tuberculous meningitis. Hypoglycorrhahia, with CSF sugar less than fifty percent of concomitant blood sugar, is a hallmark. Protein levels may increase to a level causing blockage of CSF flow. Inflammation may lead to arterial compromise and ischemia, and syringomyelia may develop as a late complication.

Spinal tuberculomas rarely show CSF abnormalities and may cause confusion with cord neoplasm.

A clinical phenomenon characteristic of spinal tuberculosis as well as other spinal infections, such as toxoplasmosis, is the therapeutic paradox. Some patients become symptomatic from spinal cord diseases only after tuberculous therapy is initiated for extra spinal disease or there may be paradoxic growth of known spinal tuberculomas after treatment initiation. This phenomenon reflects the recovery of patients delayed hypersensitivity response and increased immune system activity directed at mycobacterial antigens liberated with antimicrobial treatment. As such, it is a version of IRIS, and the vigorous inflammatory response may be detrimental to patient's neurologic recovery. Corticosteroids can be used as adjuvant treatment with considerable ongoing debate about when to institute antiretroviral therapy in HIV patients with active TB.

CERVICAL SPONDYLOSIS

Symptomatology

The characteristic syndrome consists of varying combinations of the following: (1) painful, stiff neck or pain in the neck, shoulders, and upper arms (brachialgia) that may beaching or radicular (stabs of sharp and radiating pain evoked by movement); asymmetric or unilateral; (2) numbness and paresthesias mainly of the hands; and (3) spastic leg weakness with Babinski signs, unsteadiness of gait, and a Romberg sign. The numbness and paresthesias are occasionally the earliest symptoms and typically involve the distal limbs, especially the hands[80]. Variations of these symptoms are elaborated later. Each of the components may occur separately, or they may occur in several combinations and sequences[81].

Pathologic Changes

The fundamental lesion is generated initially by a fraying of the annulus fibrosus, with extrusion of disc material into the spinal canal. The disc becomes covered with fibrous tissue or partly calcified, thereby forming a transverse osteophytic “spondylitic bar” or there may be simply central bulging of the annulus without extrusion of nuclear material. The latter changes, unlike ruptured discs that occur mainly at the C5-6 or C6-7 interspace, often involve higher interspaces and almost invariably occur at several adjacent levels. The dura- mater may be thickened and adherent to the posterior longitudinal ligament at affected levels. The underlying pia-arachnoid is also thickened and the adjacent ligamentous hypertrophy contributes

to compression of the cord or the nerve roots. This series of pathologic changes is often ascribed to hypertrophic osteoarthritis.

Anomalies at the Craniocervical Junction

Of these, congenital *fusion of the atlas and foramen magnum* is the most common. Fusion of the second and third cervical vertebrae is a common associated anomaly but does not seem to be of clinical significance[80].

Abnormalities of the Odontoid Process

These were found in 17cases of McCrae's series. There may be complete separation of the odontoid from the axis or chronic *atlantoaxial dislocation* (atlas displaced anteriorly in relation to the axis). These abnormalities may be congenital or the result of injury and are known causes of acute or chronic spinal cord compression and stiffness of the neck.

In all the congenital anomalies of the foramen magnum and the upper cervical spine there is a high incidence of syringomeylia. All patients whose symptoms might be explained by a lesion in the cervicocranial region (particularly patients in whom MS and foramen magnum tumor are suspected) require careful radiologic examination.

In mucopolysaccharidosis IV, or the Morquio syndrome, a typical feature is the absence or severe hypoplasia of the odontoid process. This abnormality, combined with laxity or redundancy of the surrounding ligaments, results in atlantoaxial subluxation and compression of the spinal cord. Affected children refuse to walk or develop spastic weakness of the limbs. Early in life they excrete an excess of keratan

sulfate, but this may no longer be detectable in adult life. In certain of the mucopolysaccharidoses, there is a true pachymeningiopathy—great thickening of the dura in the basal cisterns and high cervical region with spinal cord compression. Surgical decompression and spinal immobilization has been curative.

Platybasia and Basilar Invagination

Platybasia refers to a flattening of the base of the skull (the angle formed by intersection of the plane of the clivus and the plane of the anterior fossa is greater than 135 degrees). *Basilar impression* or *invagination* has a somewhat different meaning—namely, an upward bulging of the occipital condyles; if the condyles, which bear the thrust of the spine, are displaced above the plane of the foramen magnum, basilar invagination is present. Each of these abnormalities may be congenital or acquired (as in Paget disease); frequently they are combined. They give rise to a characteristic shortness of the neck and a combination of cerebellar and spinal signs.

MATERIALS AND METHODS

STUDY DESIGN

This is a prospective study conducted on a sample South Tamilnadu population admitted in the Department of Medicine and Neurology during the period of 2014 to 2015.The study included a standardized proforma and detailed neurological examination. Study population consisted of 50 patients admitted with myelopathies, in which history of trauma was excluded. The study population included 25 males and 25females.

CRITERIA FOR SELECTION OF PATIENTS

All cases with no history of trauma

METHODS

Patients were clinically evaluated and relevant routine biochemical analysis and appropriate neuroimaging studies were carried in all patients. Patients were categorized first according to their onset of deficit. Those within 7 days were considered acute, less than 4 weeks considered subacute, more than 4 weeks –chronic. MRI was done in all cases. Cases were classified clinically into complete / incomplete myelopathy and the latter into compressive/ non compressive myelopathy. All cases with no obvious compression visible on MRI underwent further investigations which included serum HIV, VDRL, Mantoux, ESR, X-ray chest, ANA, serum B12 assay. CSF examination was done to rule out secondary causes.

Criterion for diagnosis of acute transverse myelitis modified from Berman *et al* were as follows : 1) acutely or subacutely developing motor, sensory and sphincteric disturbance, 2) sensory level, 3) no clinical or laboratory evidence compression of spinal cord ,4) absence of other known neurological illness and 5) lack of progression over 4 weeks[82].

Six patients with spinal cord lesions suggestive of Longitudinally extensive transverse myelitis (hyperintense spinal cord signal changes in T2- weighted images, extending over 3-4 vertebral segments and in central 2/3rd of spinal cord) were subjected to visual evoked potential (VEP) study. In cases of compressive etiology relevant investigations were done to rule out secondaries in spine and other causes.

An oral consent was taken from all patients for a detailed clinical history and examination and the required laboratory investigations. The details collected from each patient were entered in the proforma. (Annexure-1).

STATISTICAL ANALYSIS

This study used the mean and the median as the measures of central tendency, standard deviation as a measure of dispersion to characterize the study population by age. In addition to this, only simple percentages were used to characterize the population under study by various parameters. The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012).

OBSERVATIONS AND RESULTS

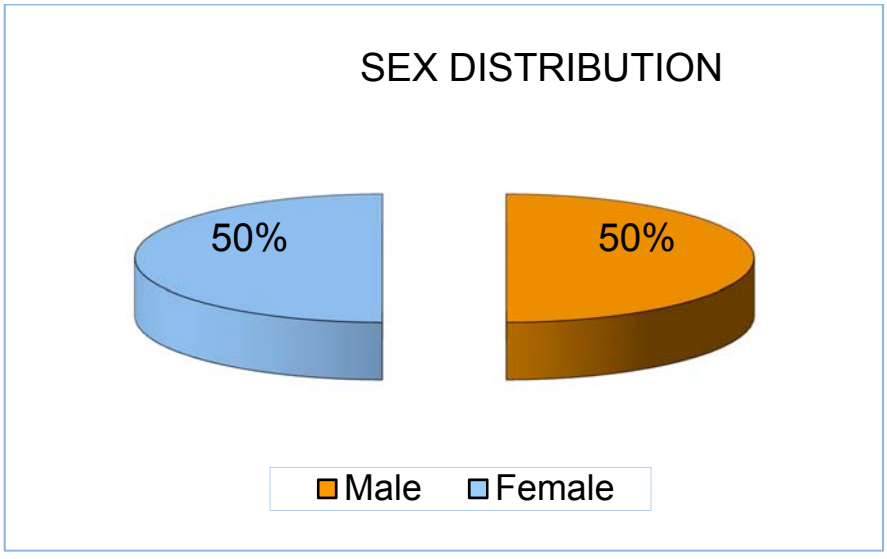
The study contained 50 patients admitted in the medical and neurological wards.

SEX DISTRIBUTION IN THE STUDY POPULATION:

Table 3: Sex Distribution

Sex	Cases	
	No	%
Male	25	50
Female	25	50
Total	**50**	**100**

Figure 4: Sex Distribution



Both females and males were equally affected.

AGE DISTRIBUTION IN THE STUDY POPULATION:

In the study the youngest patient was a 13 year old girl and the oldest patient was a 64 year old male.17 cases (34%) had an age more than 50 years. The mean age group was 43 years.

Table 4: Age Distribution

Age Group	Cases	
	No	**%**
up to 20 yrs	3	6
21 - 30 yrs	8	16
31 - 40 yrs	9	18
41 – 50 yrs	13	24
51 – 60 yrs	10	20
61 – 70 yrs	7	14
Total	**50**	**100**
Range	**13 – 65**	
Mean	**43.32**	
SD	**14.54**	

Figure 5.1: Age Distribution

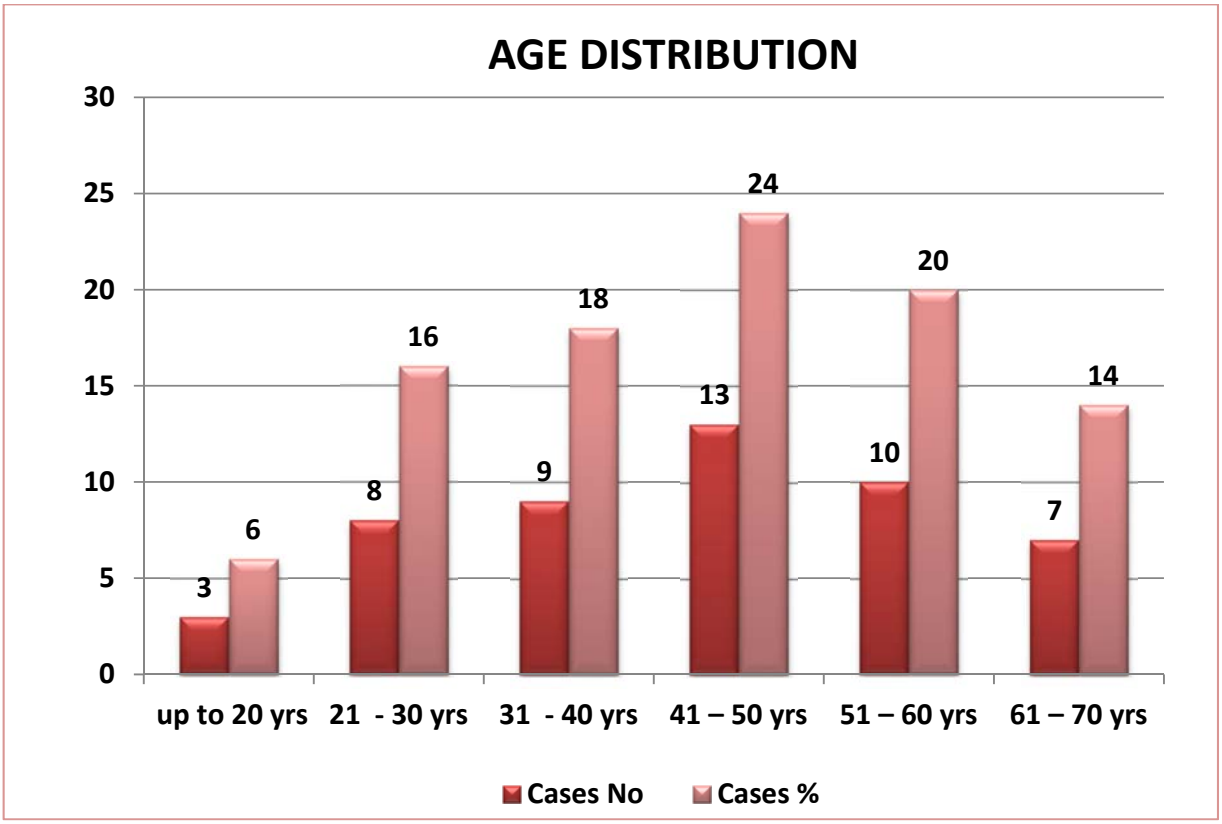
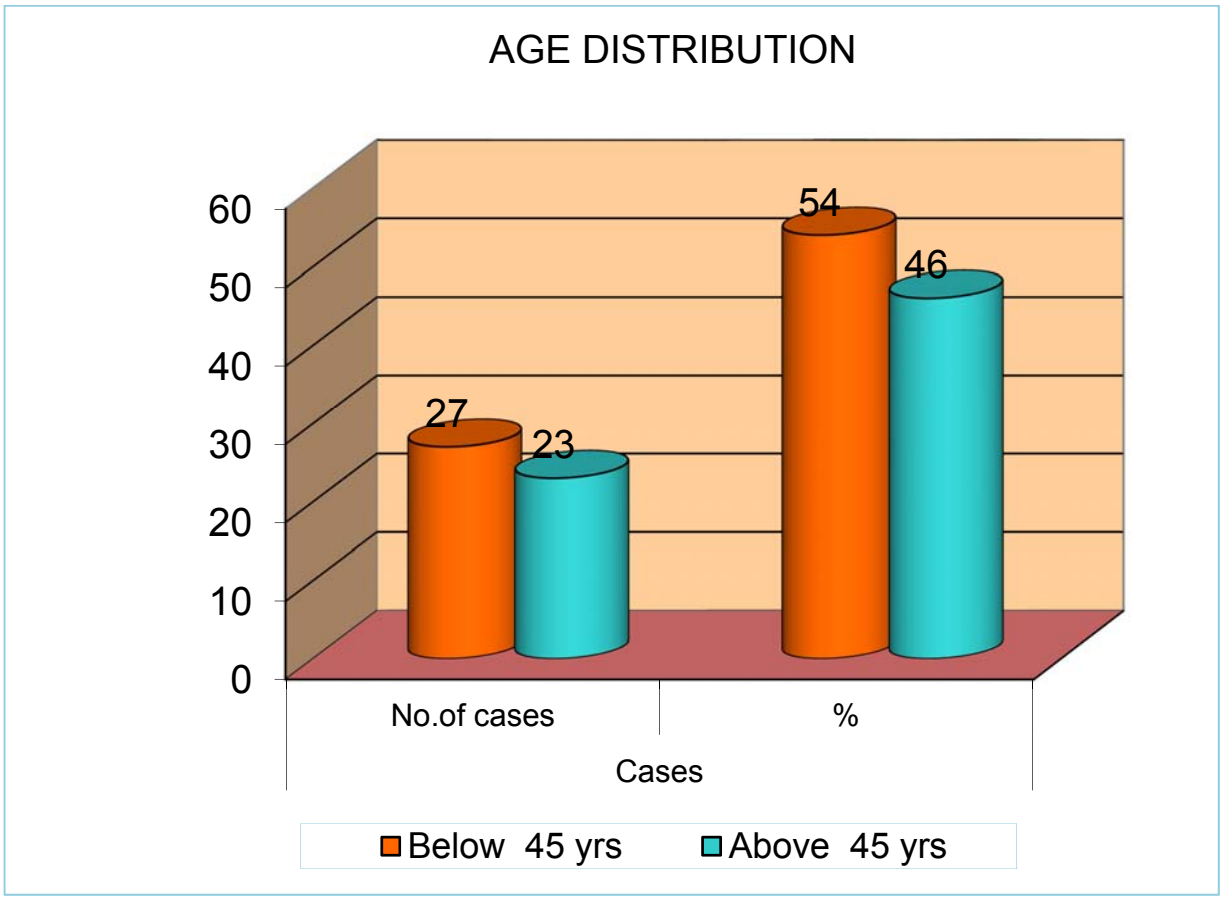


Figure 5.2: AGE DISTRIBUTION

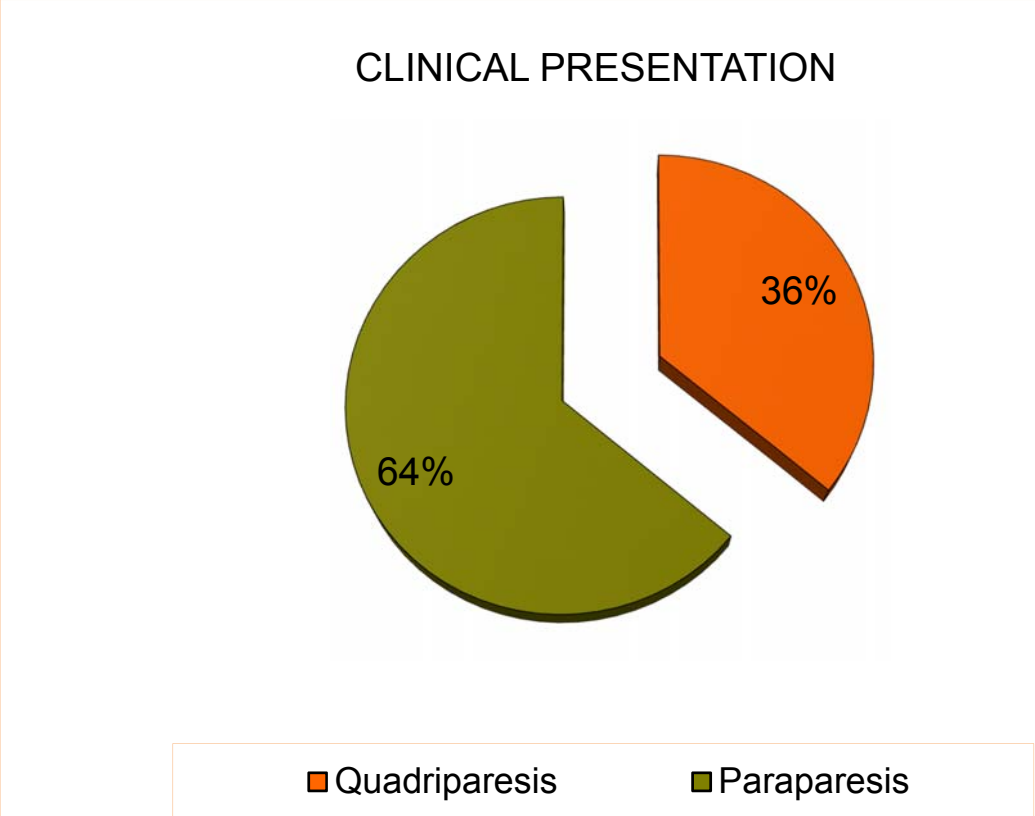


54% of the population were below 45 years, which highlights the brunt of the illness in the young

Table 5: Clinical Presentation

Clinical Presentation	Cases	
	No	%
Quadriparesis	18	36
Paraparesis	32	64
Total	**50**	**100**

Figure 6: Clinical Presentation

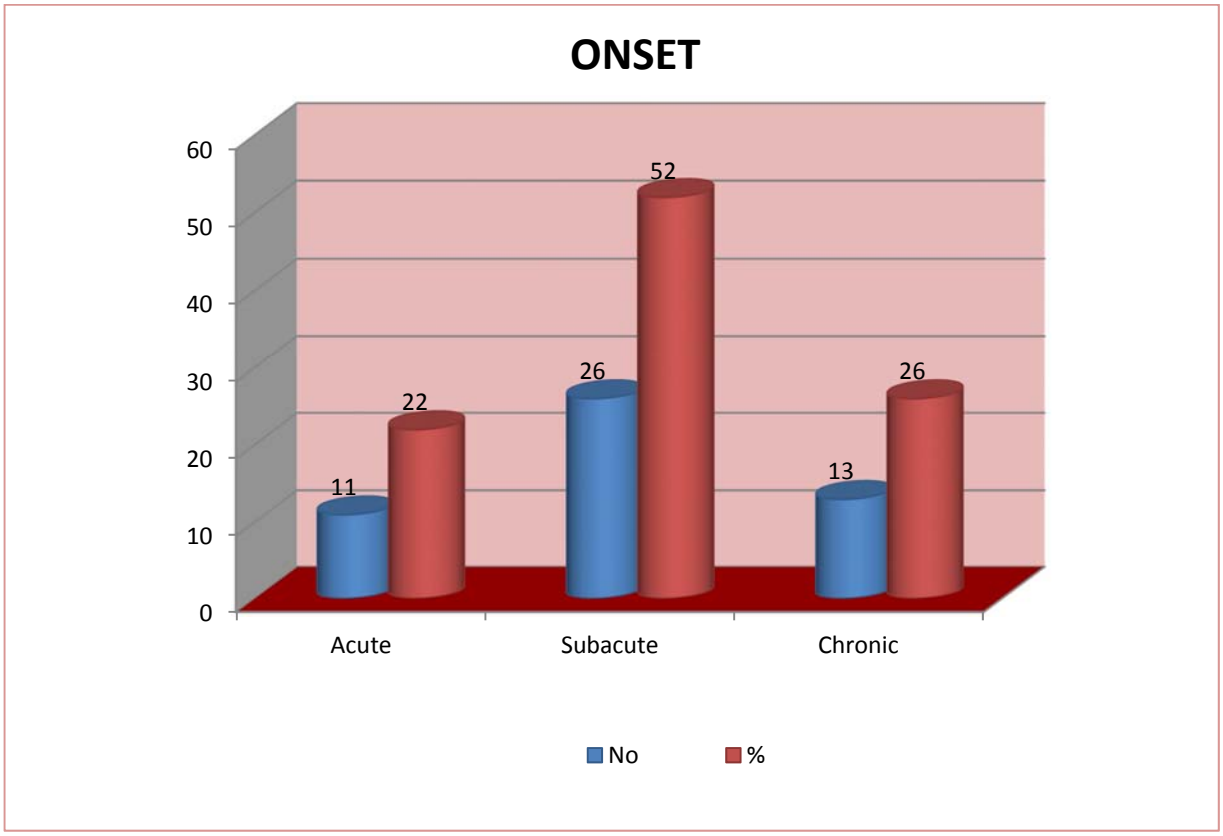


64% of the patients presented with Quadriparesis, 36 % with Paraparesis

Table 6: Onset of illness

Onset	Cases	
	No	%
Acute	11	22
Subacute	26	52
Chronic	13	26
Total	**50**	**100**

Figure 7: Onset of illness

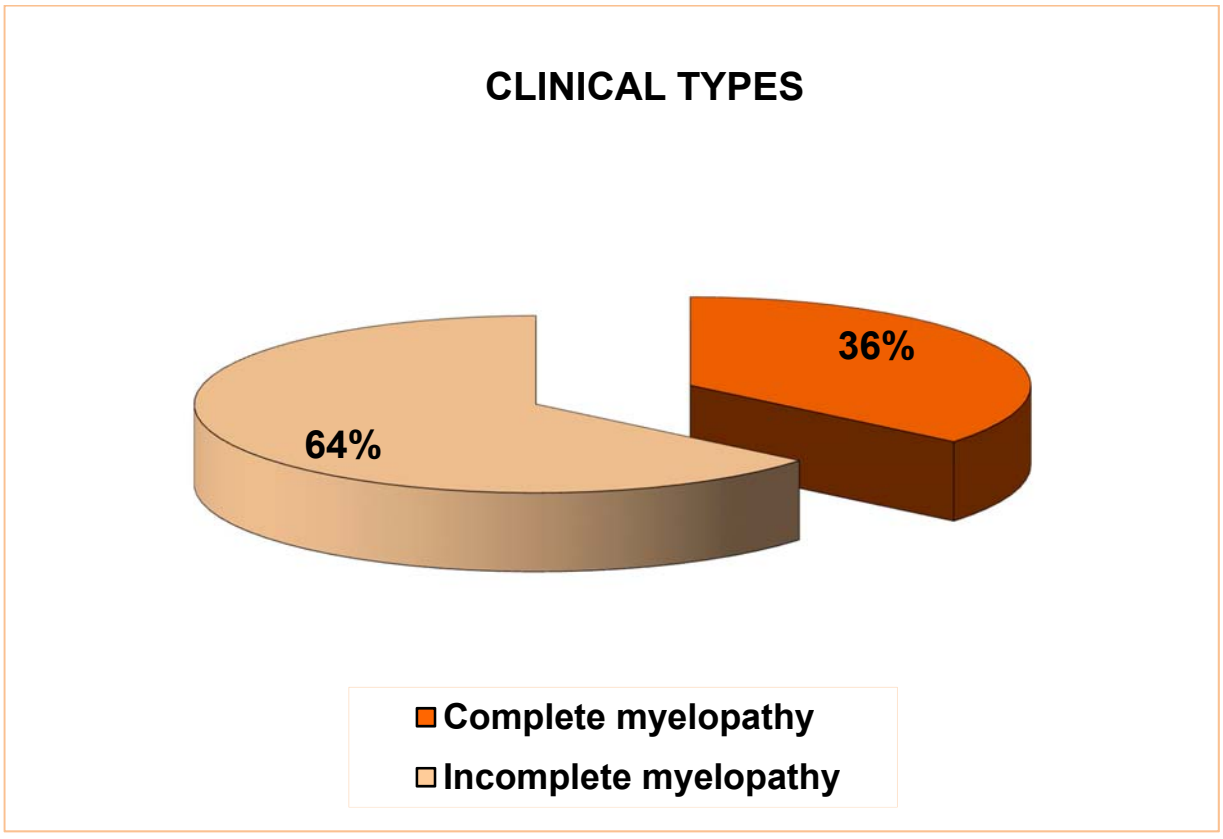


52% of the cases has presented with sub-acute onset of weakness, followed by 26% with insidious onset followed by 22% with acute onset

Table 7: Clinical Types

Clinical Types	Cases	
	No	%
Complete myelopathy	18	36
Incomplete myelopathy	32	64
Total	**50**	**100**

Figure 8: Clinical Types

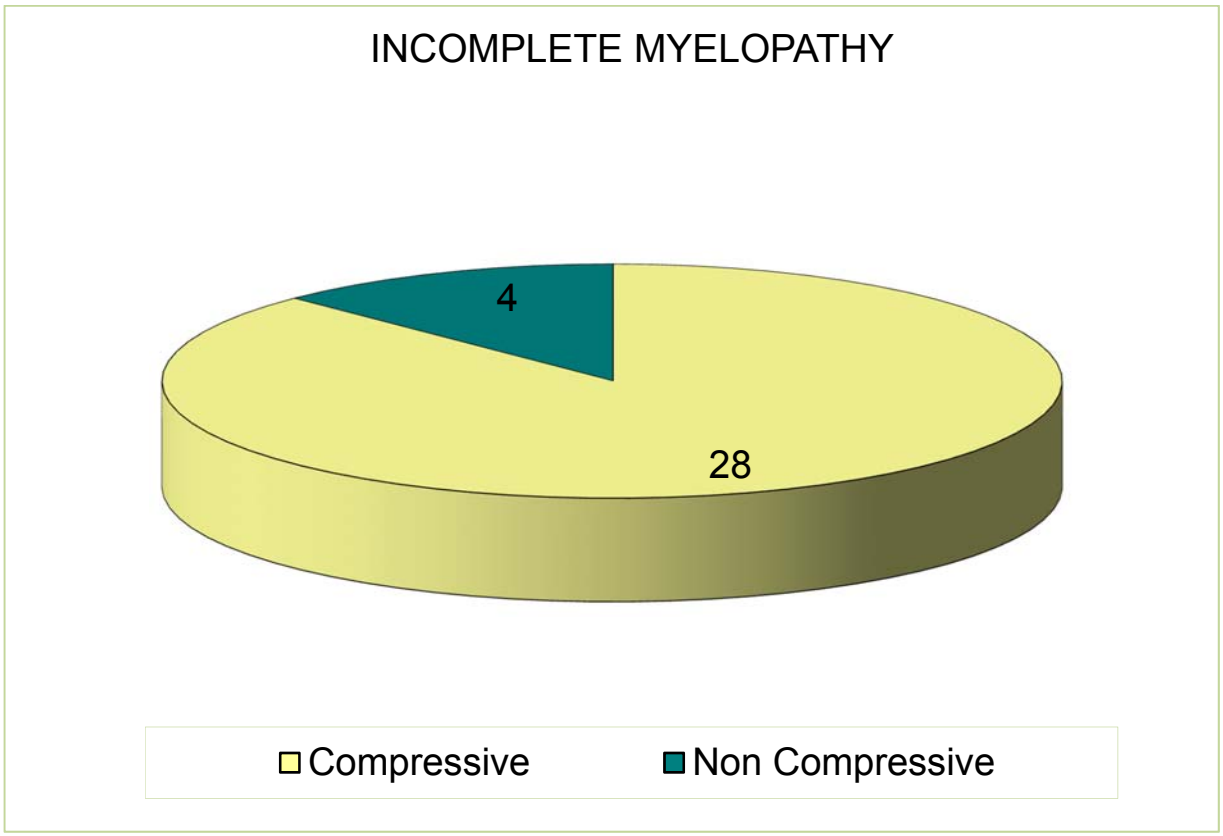


64% of the study population presented with the clinical picture of complete myelopathy

Table 7.1: Incomplete Myelopathy

Incomplete myelopathy	Cases	
	No	**%**
Compressive	28	87.5
Non Compressive	4	12.5
Total	**32**	**100**

Figure 8.1: Incomplete Myelopathy

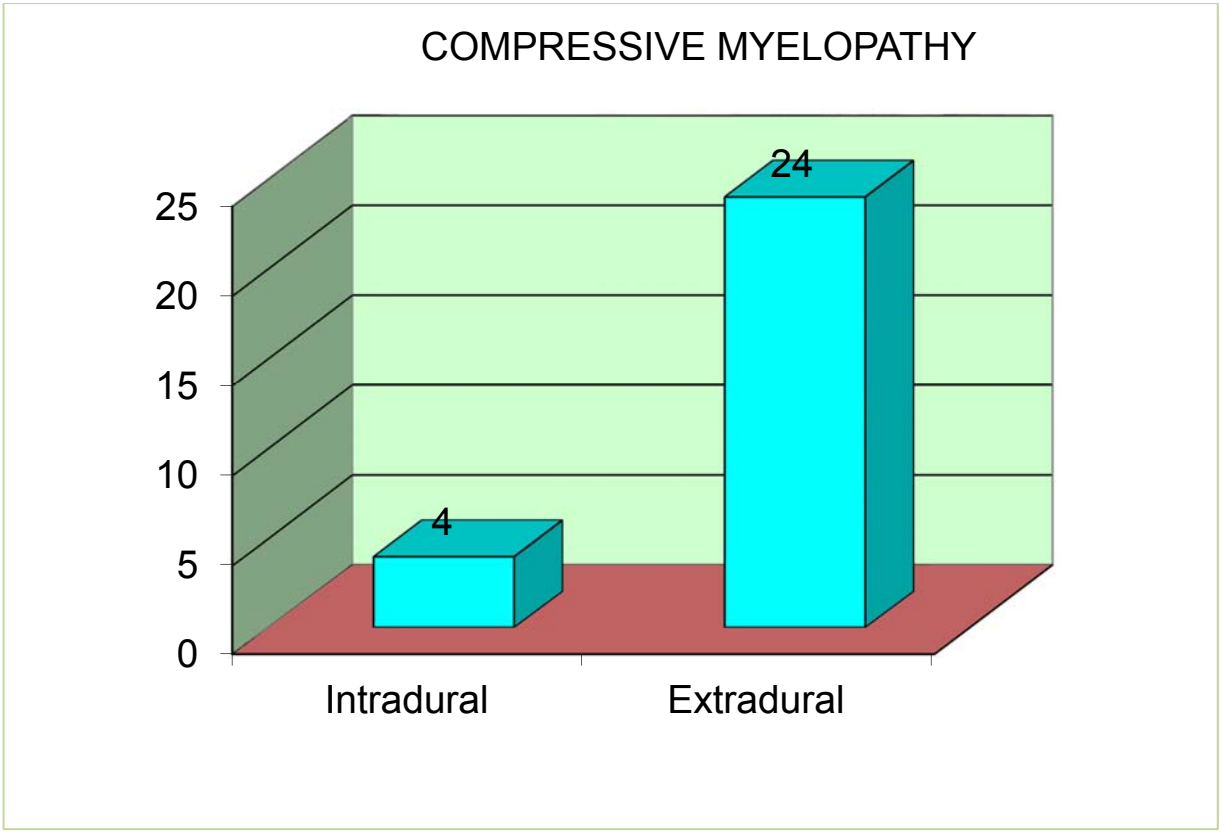


Out of the incomplete myelopathy 28 patients had a compressive cause for myelopathy.

Table 7.1.1: Compressive Myelopathy

Compressive Myelopathy	Cases	
	No	%
Intradural	4	14.3
Extradural	24	85.7
Total	**28**	**100**

Figure 8.1.1: Compressive Myelopathy

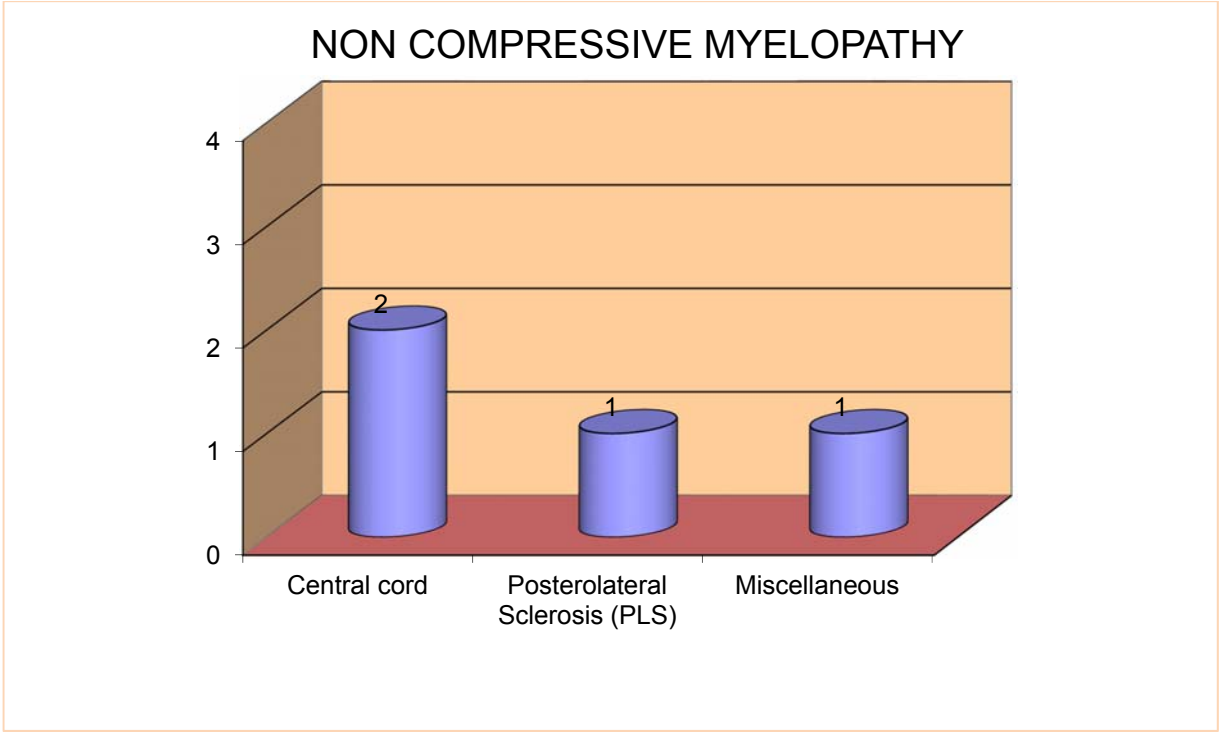


24patients with compressive myelopathy, had an extradural cause for compression

Table 7.1.2: Non Compressive Myelopathy

Non Compressive Myelopathy	Cases	
	No	%
Central cord	2	50
Posterolateral Sclerosis (PLS)	1	25
Miscellaneous	1	25
Total	**4**	**100**

Figure 8.1.2: Non Compressive Myelopathy

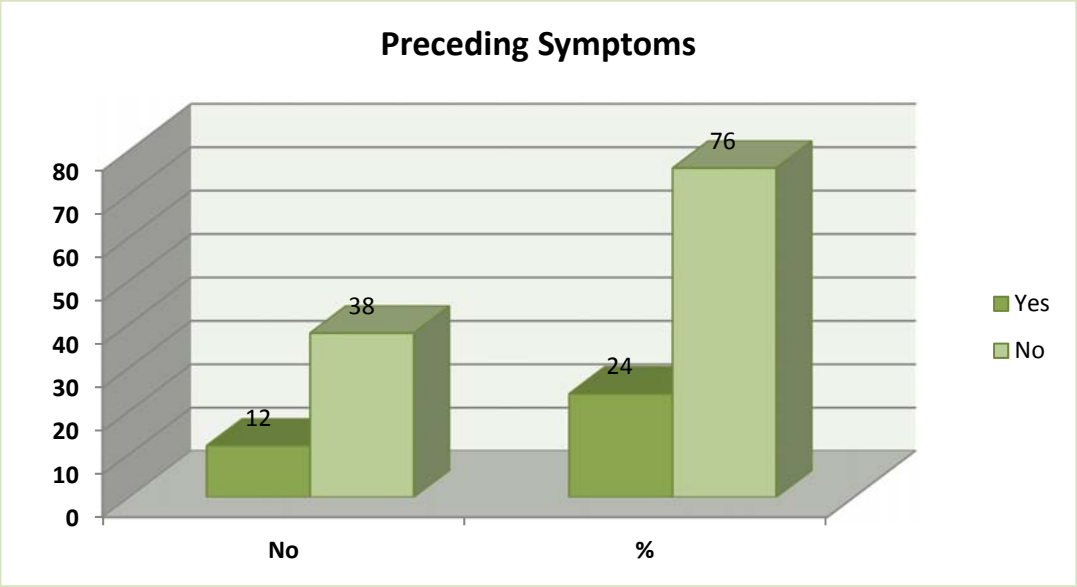


Among the noncompressive causes of incomplete myelopathy, only one case of Sub acute combined degeneration of spinal cord was reported.

Table 8: Preceding Symptoms

Preceding Symptoms	Cases	
	No	**%**
Yes	12	24
No	38	76
Total	**50**	**100**

Figure 9: Preceding Symptoms

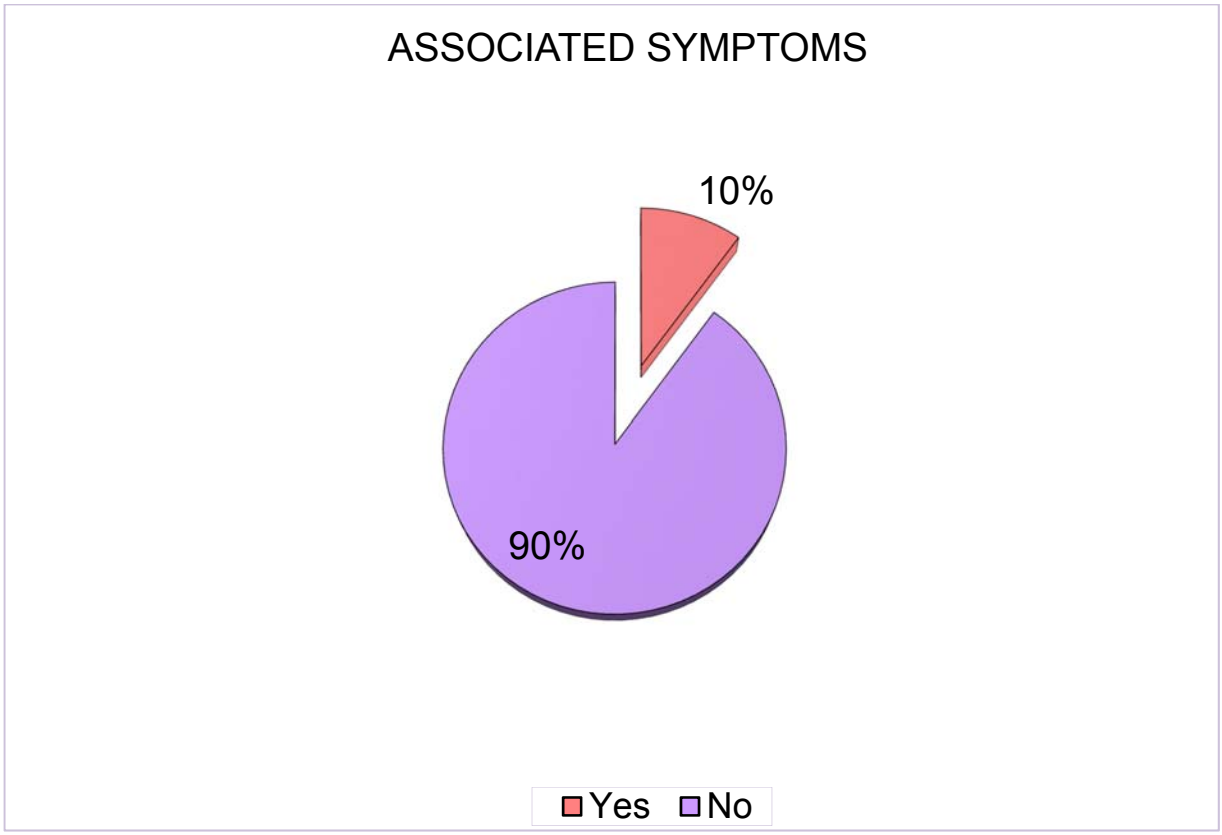


Preceding symptoms were present in 24 % of all cases.

Table 9: Associated Symptoms

Associated Symptoms	Cases	
	No	**%**
Yes	5	10
No	45	90
Total	**50**	**100**

Figure10: Associated Symptoms

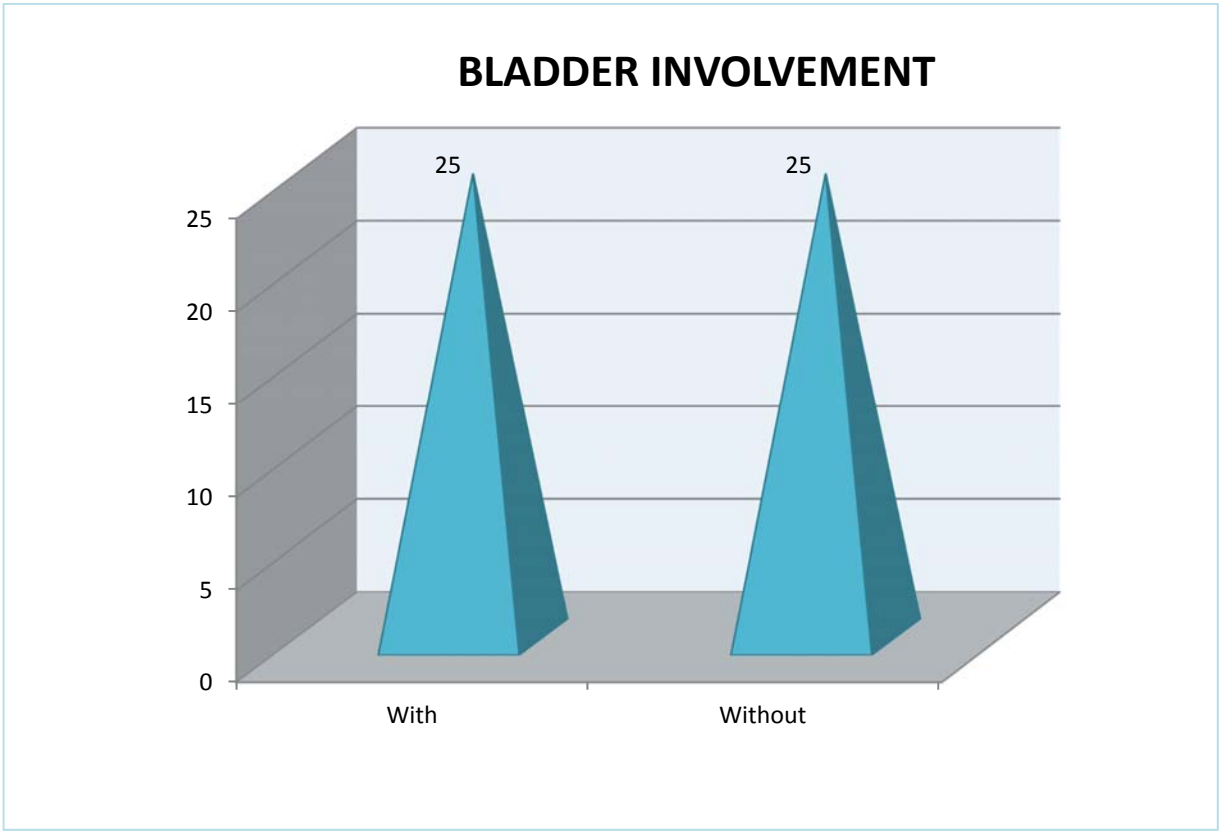


Majority of the patients had no associated symptoms

Table 10: Bladder Involvement

Bladder Involvement	Cases	
	No	%
With	25	50
Without	25	50
Total	**50**	**100**

Figure 11: Bladder Involvement



Half of the patients had involvement of the bladder

Table 11: Radiological Profile of Complete Myelopathy

Complete Myelopathy	Cases	
	No	%
MRI Positive	10	55.6
MRI Negative	8	44.4
Total	**18**	**100**

MRI was positive in 55.6 % of cases who presented with clinical picture of complete myelopathy

Figure 12: Radiological Profile of Complete Myelopathy

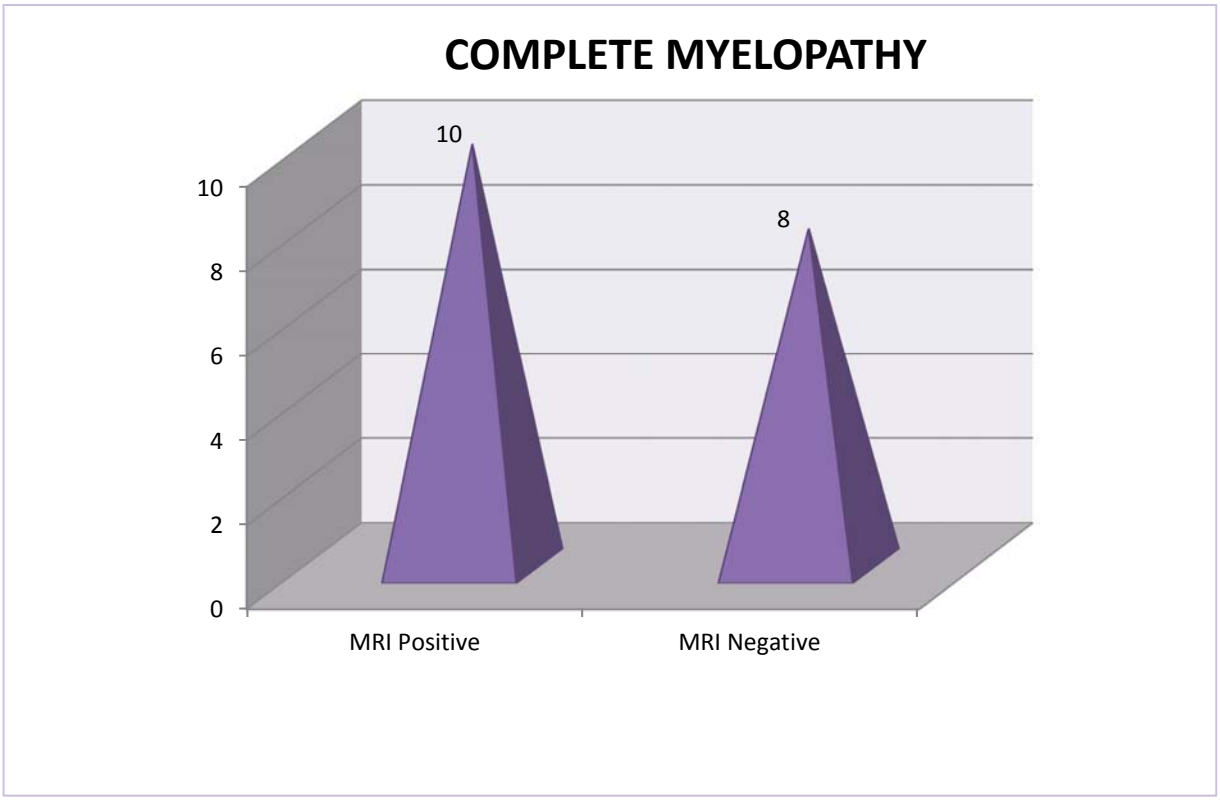


Table 12: Radiological Profile of Incomplete Myelopathy

Incomplete Myelopathy	Cases	
	No	%
MRI positive	31	96.9
MRI negative	1	3.1
Total	**32**	**100**

MRI showed a lesion in all most all cases who presented with the clinical picture of incomplete myelopathy

Figure 13: Radiological profile of Incomplete Myelopathy

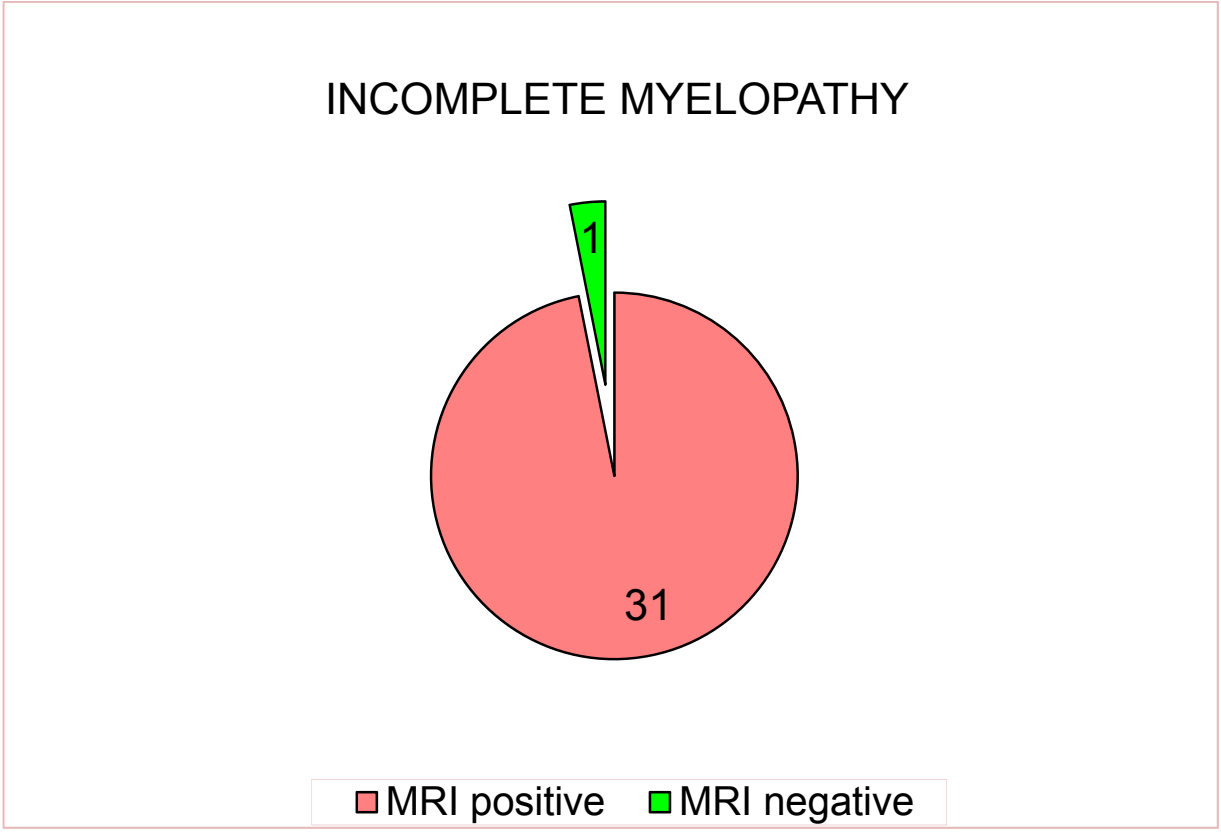


Table 13: Etiological Profile of Non Traumatic Myelopathy

Etiology	Cases	
	No	**%**
Tumours	10	20
Pott's spine	9	18
Disc prolapse	6	12
CV junction anomaly	4	8
Transverse myelitis	8	16
LETM	6	12
SCAD	1	2
Hereditary spastic paraplegia	1	2
ADEM	3	6
Syringomyelia	2	4
Total	**50**	**100**

Most common etiology was tumors followed by Potts spine and Transverse myelitis. Six patients had longitudinally extensive transverse myelitis

Figure 14: Etiological Profile of Non Traumatic Myelopathies

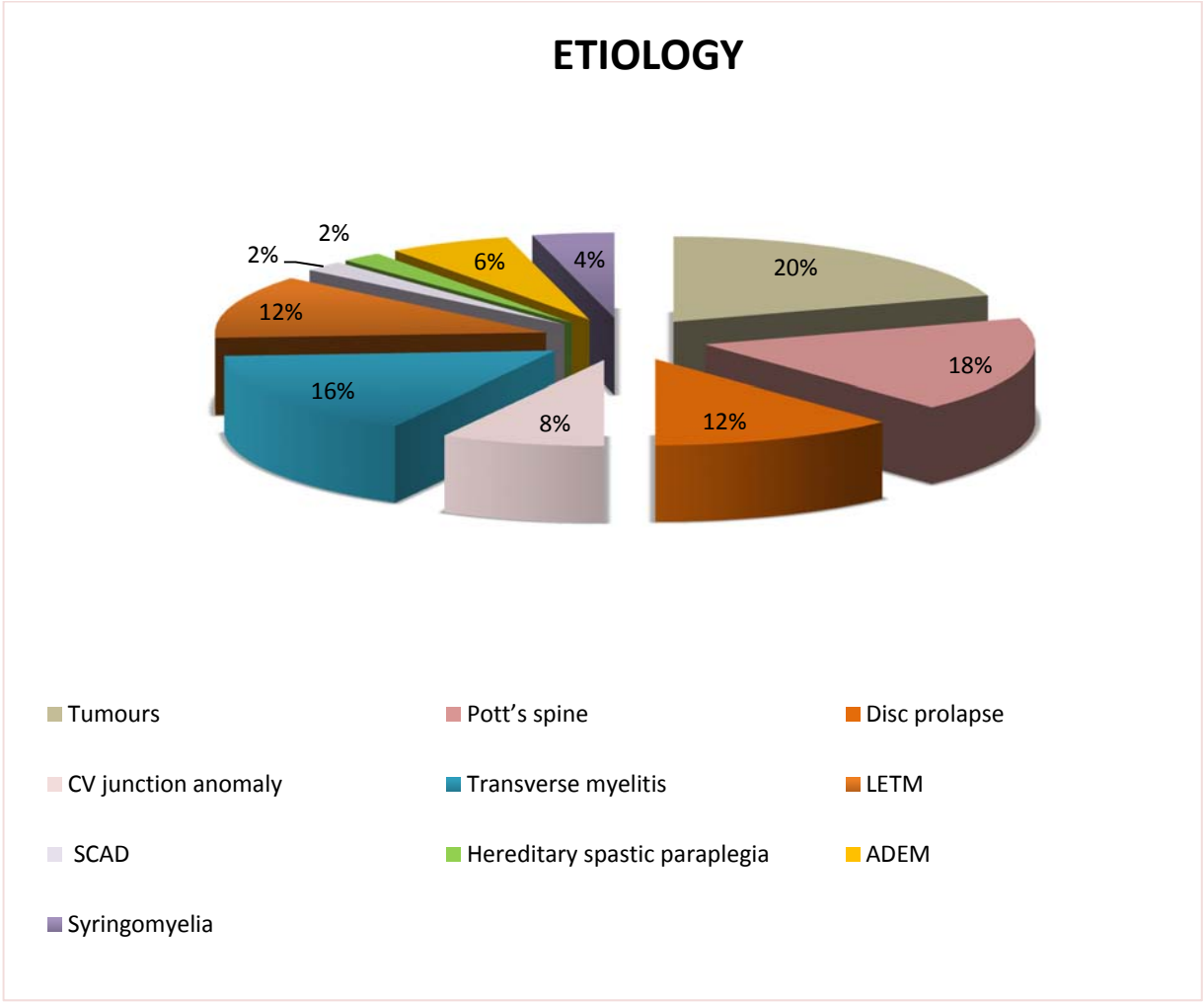
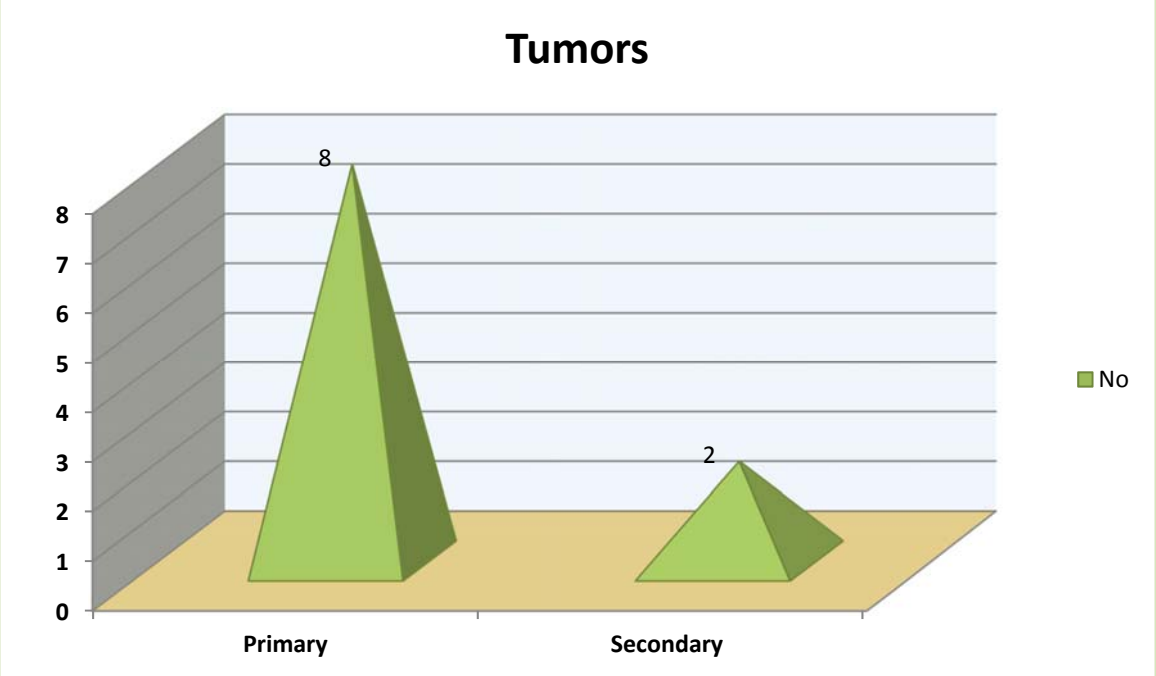


Table 14: Tumors

Tumors	Cases	
	No	%
Primary	8	80
Secondary	2	20
Total	**10**	**100**

Figure 15: Tumors



Primary tumors were more common than secondary tumors

Table 14.1: Primary Tumours

Primary Tumours	Cases	
	No	%
Astrocytoma	1	14.3
Nerve Sheath Tumours	5	71.4
Meningioma	1	14.3
Total	**7**	**100**

Nerve sheath tumors constituted the majority of the primary tumors.

Figure 15.1: Primary Tumours

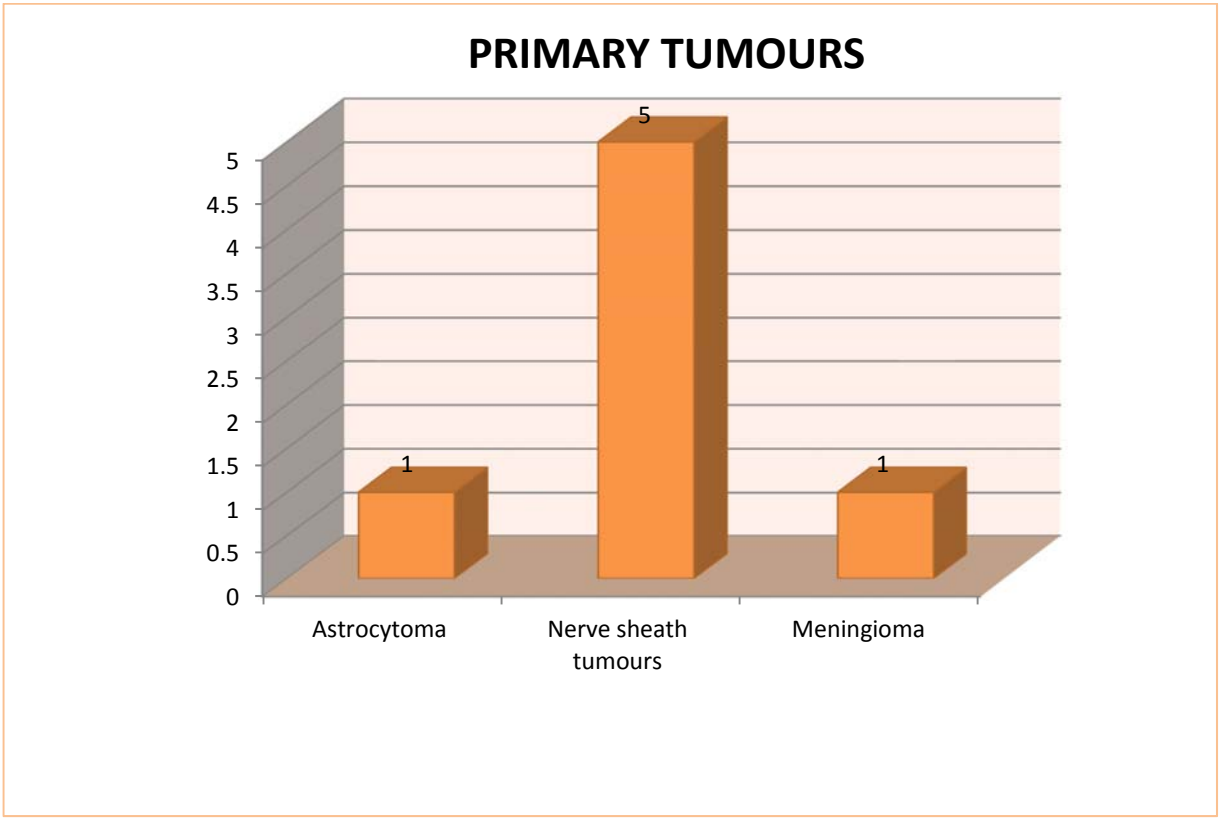
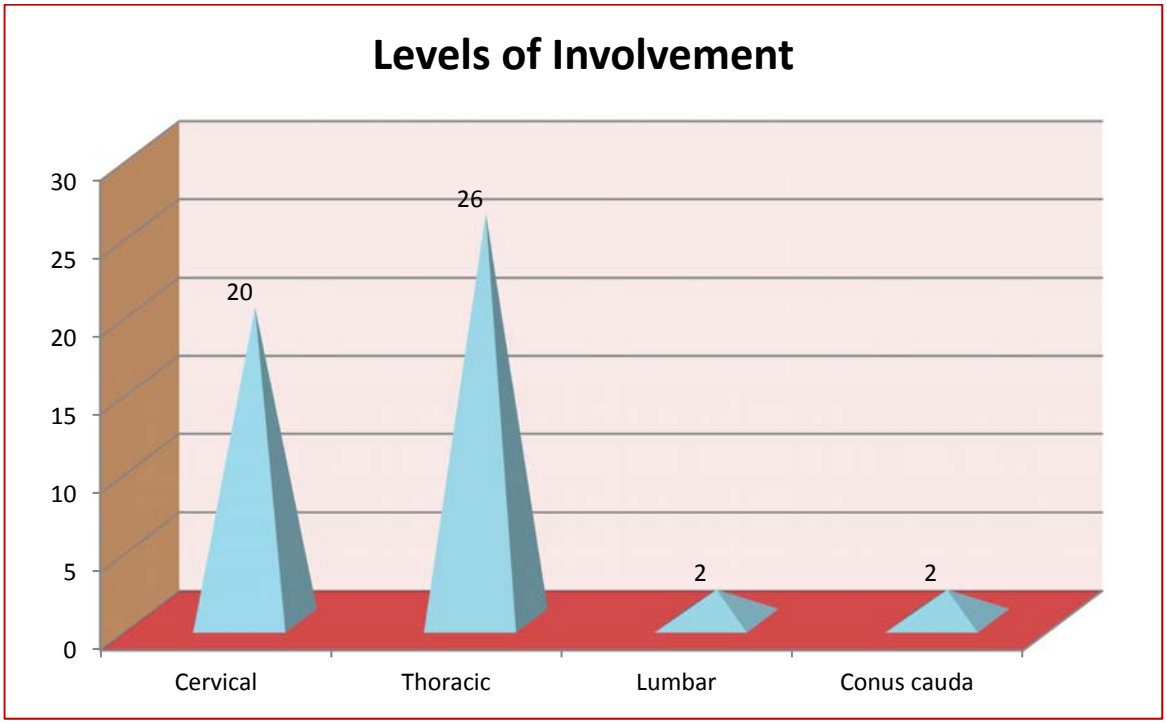


Table 15: Levels of Involvement

Levels of involvement	Cases	
	No	%
Cervical	20	40
Thoracic	26	52
Lumbar	2	4
Conus/ Cauda	2	4
Total	**50**	**100**

Figure 16: Levels of Involvement

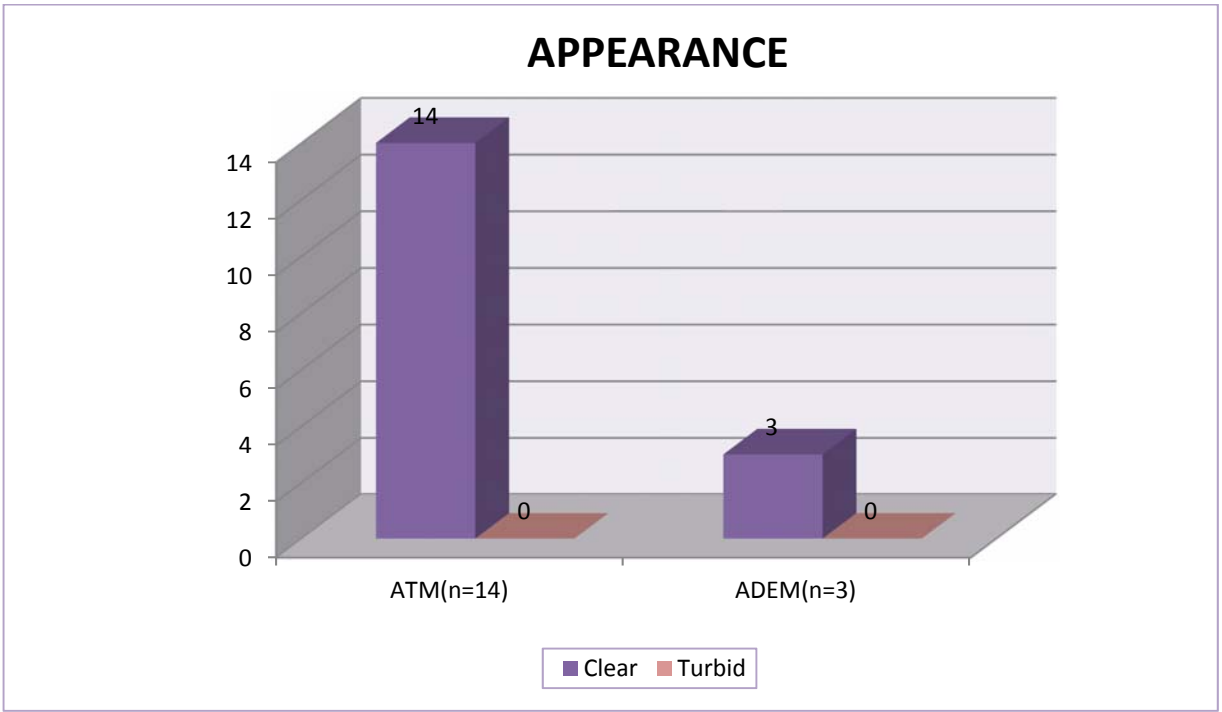


Thoracic spine was the most involved among the varies etiologies studied

Table 16: CSF analysis

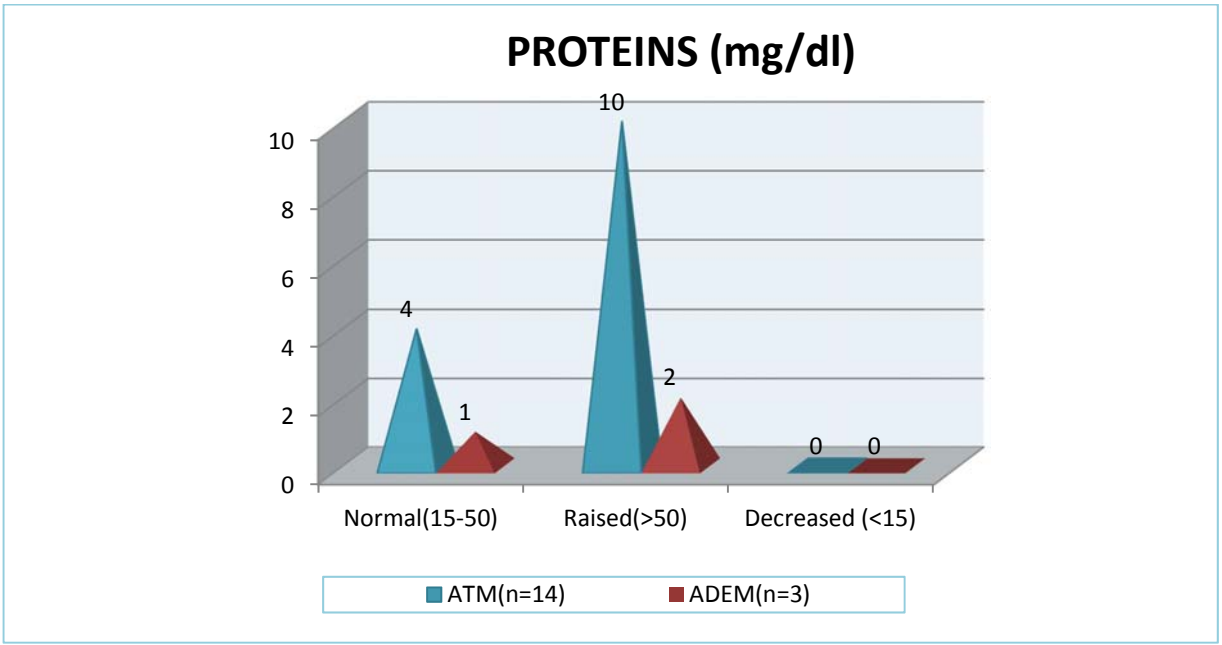
Series CSF		**ATM(n=14)**	**ADEM(n=3)**
Appearance	Clear	14	3
	Turbid	0	0
Proteins(mg/dl)	Normal(15-50)	4	1
	Raised(>50)	10	2
	Decreased (<15)	0	0
Sugar(mg/dl)	Normal(40-70)	10	3
	Increased (>70)	4	0
	Decreased (<40)	0	0
Cells (/uL)	Normal(0-5)	2	1
	Raised(5-10)	12	2

Figure: 17 .1 Appearance of CSF



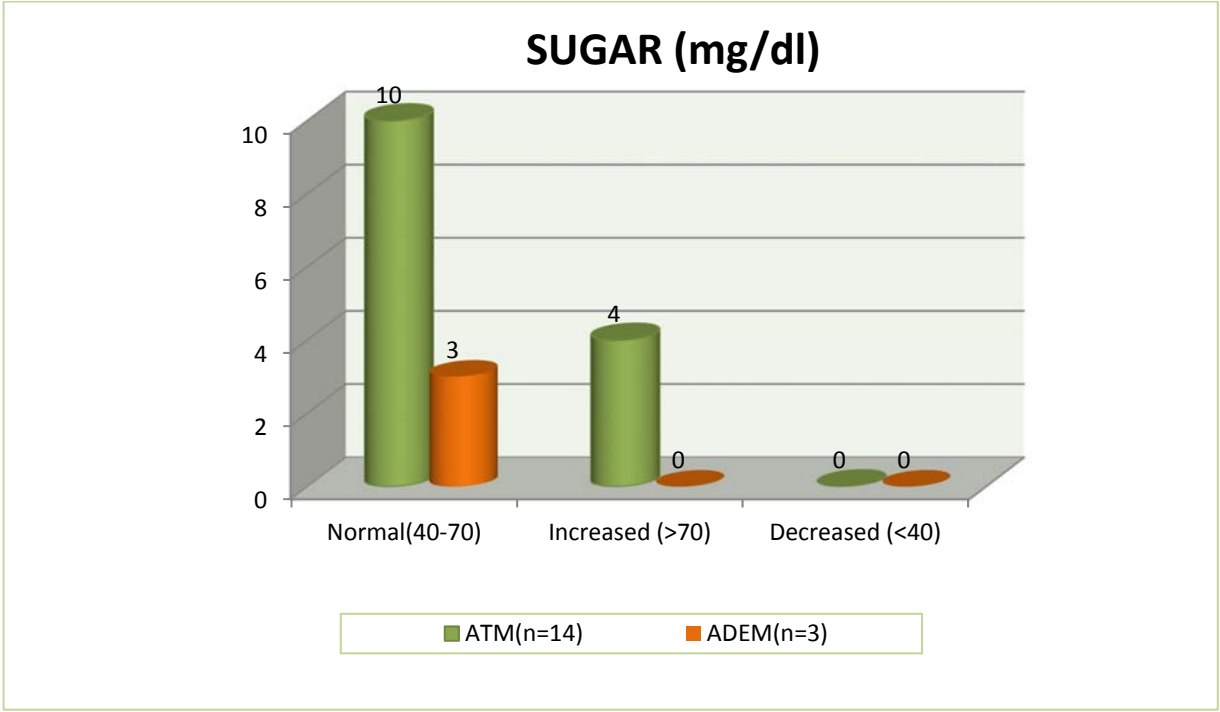
- Appearance of the CSF was normal in all the above cases.

Figure: 17.2 CSF Protein



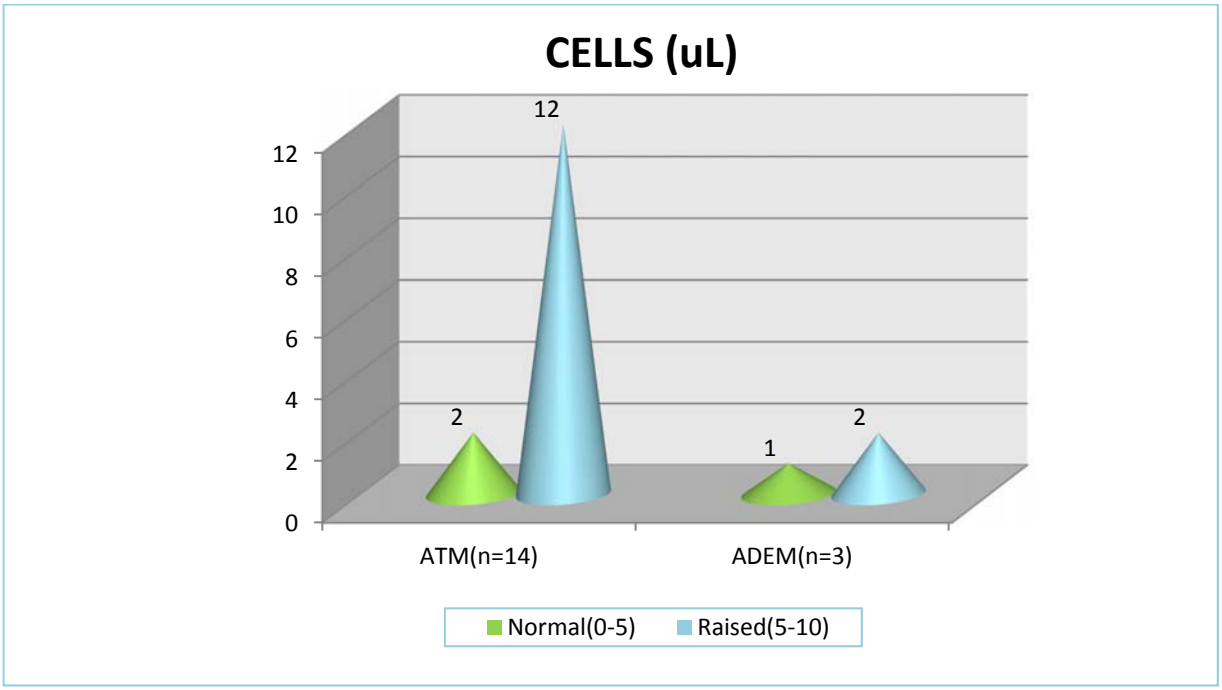
- 70% of the above cases had raised protein levels

Figure 17.3 CSF Glucose Analysis



- 76% of the above cases had normal CSF glucose levels

Figure 17.4 CSF Cytology



- 82% of the above cases had CSF pleocytosis.

DISCUSSION:

Quadriplegia and paraplegia are conditions with considerable morbidity having tremendous social repercussions. It is considered as a disease of great and constant misery to the patient, family and society.

The youngest patient was a 13 year old girl and oldest was a 67 year old lady; mean age group was 43 years. Majority of the population was below 45 years which highlights the fact that the young, bread winning people are affected which causes considerable misery. Both males and females were equally affected.64% of the patients presented with Quadriparesis, rest with paraparesis.26 patients presented with subacute onset of weakness, followed by 13 (26%) with insidious onset followed by 22% with acute onset.18 patients presented with clinical picture of complete myelopathy defined as involvement of descending tracts at the level of lesion, 32 had incomplete myelopathy. Of those with incomplete myelopathy, 28 patients had a compressive cause for the myelopathy and extradural compression accounted for most of them (85%).

24% of the patients had preceding symptoms in the form of fever, rash, arthralgia. Vast majority had no associated symptoms. Bladder involvement was there in half of the patients. MRI was done in all patients, was negative in 8 patients with complete myelopathy and showed lesion in all most all patients with incomplete myelopathy.

The most common cause of compression was tumors. Its incidence in our study is 20%; while in other studies its incidence varies from 21- 30 % of all compression. (Mani *et al*, Mehotra *et al*, Chaudary *et al*).Primary spinal cord tumors accounted the majority, while secondaries comprised 20% of the tumors.

It is estimated that involvement of spine in patients with tuberculosis was less than 1%. Tuberculosis was the second commonest cause of compressive myelopathy in this study and was observed in 18% of patients. In two studies reported in India in 2004 and 2008 tuberculosis was the leading cause of paraplegia accounting for 22%[3]cases and 18%[4] cases respectively. Lower thoracic spine (T_{7-12}) was involved in all the cases. Rest of the studies also showed Pott's spine involved in lower dorsal region. While in a study done by (Chaurasia *et al*) showed the involvement of upper thoracic spine (T_{1-6}) in the majority. Cervical disc accounted for 12% of cases.CV junction anomaly constituted 8% of the study population which included a young girl aged 13 whose MRI showed basilar invagination, others in the study had os odontoidum with spinal cord myelomalacia changes, and of this was an interesting case of Morquio syndrome (Mucopolysaccharidosis IV) with Cervical canal stenoses and thickened longitudinal segments in cervical spine on MRI.

Acute Transverse myelitis (ATM) is a monophasic illness and represents a localized form of post infectious encephalomyelitis. Incidence of ATM causing quadriparesis and paraparesis was 16%.Antecedent events in the form of fever, rash were seen in 40%[83,84] of cases which is consistent with finding in previous studies.

Longitudinally extending transverse myelitis (LETM) typically extends over 3 or more vertebral segments on MRI. In our study, 12% of patients had LETM. Visual Evoked Potentials were done in all those patients. Aquaporin antibodies were positive in two of those patients, and met the criteria for the diagnosis of Neuromyelitis optica (NMO).

Full blown clinical picture of vitamin B12 deficiency consists of macrocytic anemia, atrophic glossitis, peripheral neuropathy and cognitive decline. Of the 50 patients studied only one patient had the typical presentation of subacute combined degeneration of spinal cord. We were not able to do serum methymalonic acid, serum intrinsic factor antibody and folic acid. Syringomyelia constituted about 4 % of cases in the study population. All those patients had past history of tuberculosis supporting the hypothesis that vasculitic thrombosis of vessels supplying spinal cord leading to ischemic myelomalacia causing postinflammatory syringomyelia. There was only one case of hereditary spastic paraplegia admitted during our study period.

Early suspicion, relevant investigations and early treatment are essential to prevent irreversible damage.

There may be many shortcomings in this study. But this study will definitely give us a fair idea of the etiology of non-traumatic spinal cord involvement in the south Tamil Nadu population.

SUMMARY

In our study, it was found,

- Males and females were equally affected
- Age group below 45 years was most commonly affected
- Quadriparesis was the most common presentation
- Majority of patients presented with subacute onset
- Most common clinical type was of incomplete myelopathy
- Tumors and Potts spine were the most common cause of compressive myelopathy
- Acute Transverse myelitis was the most common cause of noncompressive myelopathy

CONCLUSION

Clinical Profile of Non Traumatic myelopathy in South Tamil Nadu is similar to that of reported in other parts of India, with Transverse Myelitis, Tumors, and Spinal Tuberculosis accounting over half the causes.

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PROFORMA

1. Name of the patient
2. Age
3. Sex
4. Occupation
5. Presenting Complaints
   Onset
   Duration
6. Brief History

7. Past History

Similar episodes	TB	DM	HTN	Others

8. Preceding symptoms
9. Associated symptoms
10. Clinical Evaluation:
    Vitals BP PR

    Motor system
    Sensory system
11. Bladder involvement

12. Complete/ Incomplete

13. Level of Lesion

14. Compressive/Non compressive

15. Intradural/Extradural

16. Lab

    CBC
    RFT, LFT
    S.Electrolytes
    HIV
    VDRL
    ANA[a]
    S. B12 levels[a]
    Mantoux

17. Imaging
    X Ray Chest
    X Ray Spine
    MRI Spine
18. CSF analysis
19. Etiologcal Diagnosis

a: in relevant cases

Master Chart

SL No	Name	Age	Sex	CP	Onset	C.type,C/NC	Preceding symptom	Ass. Symptom	Bladder	MRI	Etiology	Level
1	Jeswanathan	48	M	Paraplegia	Acute	Complete	no	no	no	no	TM	lumbar
2	Rajammal	46	F	Quadriplegia	Subacute	Complete	no	no	yes	yes	tumor	cervical
3	Murugan	48	M	Quadriplegia	chronic	Incomplete C-ID	no	no	no	yes	tumor	cervical
4	Annapackiyam	58	F	Paraplegia	Subacute	Incomplete C-ED	Yes	yes	no	yes	tumor	thoracic
5	Muthulakshmi	13	F	Quadriplegia	Subacute	Incomplete C ED	no	no	no	yes	CV jn	cervical
6	Mupidathi 48	48	F	Paraplegia	Acute	Complete	Yes	no	yes	no	TM	thoracic
7	Uikkattan	24	M	Paraplegia	Subacute	Complete	yes	no	yes	yes	TM	thoracic
8	Gurusamy	43	M	Quadriplegia	chronic	Incomplete NC -CC	no	no	yes	yes	Syringomyelia	cervical
9	Sudalai	25	F	quadriplegiac	chronic	Incomplete NC -CC	no	no	yes	yes	Syringomyelia	cervical
10	Gomathy	27	F	Paraplegia	Acute	Complete	Yes	yes	yes	no	ADEM	thoracic
11	Perumal	20	M	Paraplegia	Acute	Complete	yes	no	yes	no	TM	thoracic
12	Mupidathi	50	F	Paraplegia	Subacute	Complete	yes	no	yes	no	ADEM	thoracic
13	Duraipandi	55	M	Paraplegia	chronic	Incomplete C-ED	no	no	yes	yes	tumor	Conus cauda
14	Mariraj	22	M	Paraplegia	Acute	Complete	no	no	yes	yes	LETM	thoracic
15	Palpandiyan	65	M	Paraplegia	Subacute	Complete	yes	no	yes	yes	LETM	thoracic
16	Aruna	52	F	Quadriplegia	chronic	Incomplete NC -Misc	no	no	no	no	HSP	cervical
17	Sankaran	35	M	Paraplegia	Subacute	Incomplete C ID	no	no	yes	yes	tumor	thoracic
18	Ganeshan	35	M	Quadriplegia	Subacute	Incomplete C ED	no	no	no	yes	disc	cervical
19	Samiduram	65	M	Paraplegia	Subacute	Incomplete C-ED	no	no	no	yes	potts spine	thoracic
20	Esakkidurai	65	M	Paraplegia	Subacute	Incomplete C-ED	no	no	no	yes	potts spine	thoracic
21	Valliammal	60	F	quadriplegia	subacute	Incomplete C-ED	no	yes	no	yes	tumor	cervical
22	Arumugam	36	F	quadriplegia	subacute	Incomplete C-ED	no	no	no	yes	disc	cervical
23	Venkatesh	43	M	quadriplegia	subacute	Incomplete C-ED	no	no	no	yes	disc	cervical
24	Ananthi	45	F	paraplegia	subacute	Incomplete C ED	no	no	yes	yes	tumor	conus cauda
25	Balamani	37	F	quadriplegia	chronic	Incomplete C-ED	no	no	yes	yes	disc	cervical
26	Rajaiah	65	M	quadriplegia	subacute	Incomplete C-ID	no	no	no	yes	tumor	cervical
27	SakilaBanu	42	F	paraplegia	acute	Complete	no	no	yes	yes	LETM	thoracic
28	Revathy	33	F	paraplegia	acute	Complete	yes	no	yes	yes	LETM	cervical
29	Petchiammal	42	F	quadriplegia	subacute	Complete	yes	no	no	no	ADEM	cervical
30	Velu	34	M	paraplegia	acute	Complete	no	no	yes	yes	TM	lumbar
31	Kalingam	63	F	paraplegia	chronic	Incomplete C-ID	no	no	yes	yes	tumor	thoracic
32	Shanmugathai	65	F	paraplegia	subacute	Incomplete C-ED	yes	yes	no	yes	potts spine	thoracic
33	Subhulakshmi	52	F	quadriplegia	Subacute	Incomplete C-ED	no	yes	yes	yes	potts spine	thoracic
34	Janaki	27	M	quadriplegia	chronic	Incomplete C-ED	no	no	yes	yes	CV Jn	cervical
35	Uthiraprakasham	18	M	paraplegia	chronic	Incomplete NC-PLS	no	no	no	yes	SCD	thoracic
36	Devaraj	36	M	paraplegia	subacute	Incomplete C-ED	no	no	no	yes	potts spine	thoracic
37	Pandaram	45	M	paraplegia	subacute	Incomplete C-ED	no	no	no	yes	potts spine	thoracic
38	Kittu	57	M	paraplegia	subacute	Incomplete C-ED	no	no	no	yes	disc	cervical
39	Navaneetha Krishna	64	M	paraplegia	subacute	Incomplete C-ED	no	no	no	yes	potts spine	thoracic
40	Mala	21	F	paraplegia	acute	Complete	no	no	yes	yes	LETM	thoracic
41	Gurusamy	52	M	quadriplegia	subacute	Incomplete C-ED	no	no	no	yes	CV Jn	cervical
42	Mupidathi	56	F	quadriplegia	chronic	Incomplete C-ED	no	no	no	yes	disc	cervical
43	Pasukili	40	F	paraplegia	chronic	Complete	no	no	yes	no	TM	Thoracic
44	Subramaniyam	44	M	paraplegia	subacute	Complete	yes	no	yes	yes	TM	Thoracic
45	Velusamy	48	M	paraplegia	subacute	Incomplete C-ED	no	no	no	yes	potts spine	Thoracic
46	Angaleswari	53	F	paraplegia	acute	Complete	yes	no	yes	yes	LETM	Thoracic
47	Leelavathi	40	F	paraplegia	acute	Complete	no	no	yes	no	TM	Thoracic
48	Mohideen	24	M	quadriplegia	subacute	Incomplete C-ED	no	no	no	yes	CV Jn	Cervical
49	Saraswathy	60	F	paraplegia	chronic	Incomplete C-ED	no	no	no	yes	potts spine	Thoracic
50	uma	27	F	paraplegia	chronic	Incomplete C-ID	no	no	no	yes	tumor	Cervical

CP : Clinical Presentation || C.type: Clinical Type || C: Compressive || NC: Non Compressive | ID: Intradural || ED: Extradural || CC: Central Cord

Misc: Miscellaneous || PLS: Posterolateral sclerosis || SCD: Sub Acute Combined Degeneration || TM : Tranverse Myelitis

LETM : Longitudinally Extensive Transverse Myelitis || CV Jn : Cranio vertebral Junction Anomaly