EVALUATION OF T2 HYPERINTENSITIES IN SPINAL CORD LESIONS USING MAGNETIC RESONANCE PROTOCOL - A ONE YEAR OBSERVATIONAL STUDY AT TERTIARY CARE HOSPITAL IN NORTH KARNATAKA.

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ACCEPTANCE LETTER

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To,
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LIST OF ABBREVIATIONS:

ADC	Apparent diffusion coefficient
ADEM	Acute disseminated encephalomyelitis
ATM	Acute transverse myelitis
AVM	Arterio Venous Malformation
BSL	Bright spotty lesions
C, T, L	Cervical, Thoracic, Lumbar
CNS	Central nervous system
DAVF	Dural arterio venous fistula
DCE-MRI	Dynamic contrast-enhanced magnetic resonance imaging
DW-MRI	Diffusion-weighted MRI
HE	Heterogenous enhancement
ISCM	Intramedullary spinal cord metastases
ISS	Intramedullary spinal sarcoidosis
LETM	Longitudinally extensive transverse myelitis
MRI	Magnetic resonance imaging
MS	Multiple Sclerosis
NMO	Neuromyelitis optica
OPLL	Ossification of the posterior longitudinal ligament

SACD	Sub acute combined degeneration
SCI	Spinal cord infarction
STIR	Short time inversion recovery
T1W + C	T1-weighted + contrast
T1WI	T1-weighted images
T2 WI	T2-weighted images
TM	Transverse myelitis

ABSTRACT

Background and objectives:

Myelopathy is the major cause of quadriplegia and disability. Many spinal cord abnormalities are reversible if recognized and treated at an early stage. Plain radiographs have low sensitivity in identification of demyelinating / neoplastic spinal lesions. The role of MRI is to distinguish compressive from non- compressive causes of myelopathy. Spinal cord biopsy is a high-risk procedure with the potential to cause permanent neurological injury. Magnetic resonance imaging is the modality of choice for diagnosis and preoperative assessment of patients with spinal cord abnormalities.

One of the most common finding in imaging is either focal or diffuse hyperintensity on T2 weighted MR images.

Intramedullary T2 hyperintensity pose a serious dilemma in imaging because it has a wide range of causes such as MR artifacts, trauma, primary and secondary tumors, multiple sclerosis, subacute combined degeneration of the spinal cord, transverse myelitis, neurosarcoidosis and syringohydromyelia.

The objective of this study is to approach systematically which incorporates detailed clinical history, acuity of symptoms, distribution of the signal abnormalities, including length of cord involvement and the area that is affected which helps narrowing down the list of differential diagnosis and improve the treatment outcomes.

Methodology:

The current study was a observational study, conducted in the Department of Radio diagnosis at The KLE'S DR. PRABHAKAR KORE Hospital & MRC, BELGAVI. All the patients referred to Department Of Radio-Diagnosis with clinically suspected spinal cord lesions were considered as study population.

A total of 53 eligible subjects were recruited into the study consecutively between January 2019 to December 2019 for a period of 1 year.

Patients with past history of prior surgery of spinal cord, non consenting subjects, patients who have claustrophobia, freshly implanted MRI incompatible prosthesis, patients with aneurysm clips, ferromagnetic implants and pacemakers were excluded from the study. Study was approved by the institutional human ethics committee. Informed written consent was obtained from all the study participants.

MRI spine of all the patients was taken using 1.5-tesla & 3-tesla symphony maestro class MRI with the help of a dedicated spine coil with the patient in supine position, and entire spine will be examined.T1W, T2W, STIR sequences were used in the axial and sagittal planes. T1W + contrast Diffusion-weighted sequences and ADC sequences were used whenever possible. During MRI evaluation, the following parameters were delineated – level of lesion involvement (cervical, thoracic & lumbar), severity of cord compression, ligamentous injury & cord edema / contusion, prevertebral & paravertebral collection and other significant findings if any.

The patients were chosen for study by a process of purposive sampling and data were analyzed by descriptive analysis.

Results:

The mean of age was 44.45 ± 18.69 years. The minimum age was 3 years, and the maximum age was 75 years. Among the study population, 28 (52.83%) cases were males and remaining 25 (47.17%) cases were females. Most common symptom was cervical pain in 14 (26.42%) cases followed by backache in 12 (22.64%) cases. Most commonly involved level of the lesions involved in 53 cases were cervical 33 (62.26%), followed by thoracic 25 (45.22 %), lumbar 3 (5.66%) cases and least common site was cervicomedullary junction 2 (3.77 %) cases. In this study population, most common cause of T2 hyperintensity was cord myelomalacia 15 (28.31 %) cases and demyelinating conditions 15 (28.31 %) cases, congenital abnormalities comprised of 11 (22.64 %) cases and primary intramedullary neoplasms of 3 (5.66 %) cases.

Interpretation and Conclusion:

MRI is definitive modality in assessing spinal soft tissue injuries, especially in evaluation of spinal cord edema and ligaments. Its very sensitive and considered choice of imaging for all spinal tumors. MRI is the most sensitive modality to detect and characterize demyelinating conditions such as transverse myelitis, multiple sclerosis, subacute combined degeneration and neuromyelitis optica. The final diagnosis still relies on biopsy and culture. Till date, MRI is the only modality to directly image the spinal cord.

In my study with the help of MRI I could successfully assess the integrity of spinal cord and characterize the cause of T2 hyper intensities in spinal cord lesions. So in the end I can conclude that MRI is very definitive, sensitive, accurate, though costly but very specific, non invasive and radiation free modality for evaluation of myelopathy.

The study findings can be useful for clinical practitioners dealing with possible cases of T2 hyper intensities in spinal cord lesions in early diagnosis and effective management.

Keywords:

Spine, myelopathy, Intradural neoplasms, Diagnosis, Demyelinating diseases, MRI.

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INTRODUCTION

Myelopathy is nervous system disorder which is related to spinal cord abnormalities which are often a diagnostic challenge, in which there could be compression within it. Focal or generalised hyperintensity on T2W MRI is the most essential finding. In day to day practice radiologists come across these lesions which often creates great confusion. Spinal cord biopsy is a high-risk procedure with potential to cause permanent neurological injury. Hence, there is a need of study to approach systematically that incorporates patient's history, distribution of the signal abnormalities, including length of cord involvement and the area that is affected which helps narrowing down the list of differential diagnosis.

The systematic use of imaging, with knowledge of anatomy & tracts of cord and in combination with clinical features of common spinal cord diseases, is needed for successful outcome. Many spinal cord diseases are reversible if recognized and treated at an early stage thus they are among the most critical of neurologic emergencies. MRI is the modality of choice for diagnosis and preoperative assessment of patients with spinal cord abnormalities.[46]

The efficient use of imaging, guided by a knowledge of the anatomy and in combination with clinical features of common spinal cord diseases, has the potential to save patients from invasive approaches to diagnosis and guide proper treatment.

This study will provide an approach for evaluating intramedullary spinal cord lesions to help narrow down the differential diagnosis.

After a thorough literature search it is found out that there are no such studies in our population, thus making the need for the study quite obvious.

AIMS AND OBJECTIVES

AIM:

To review spinal cord lesions based on T2 hyperintensity.

Objectives:

To evaluate T2 hyperintensities in spinal cord lesions using magnetic resonance protocol

REVIEW OF LITERATURE

ANATOMY OF THE SPINAL CORD

Anatomical knowledge of the spinal cord is essential for detailed evaluation. The spinal cord is the extension from brainstem at the foramen magnum traversing through the vertebral canal upto the conus medullaris at about L1 vertebra in an adult and L2/L3 vertebral level in a child. Inferior to L1 it is continued as film terminale and is inserted at the coccyx. It measures 45 cms in males and 42 cms in females. Its divided into 31 levels, cervical (8), thoracic (12), lumbar (5), sacral (5) and coccygeal (1) where nerve roots emerges. Spinal cord is enveloped by: 'dura mater, arachnoid mater, and pia mater'. The cord is divided into ventrolateral sulcus & dorsolateral sulcus from which ventral and dorsal nerve roots enter and exit the cord.

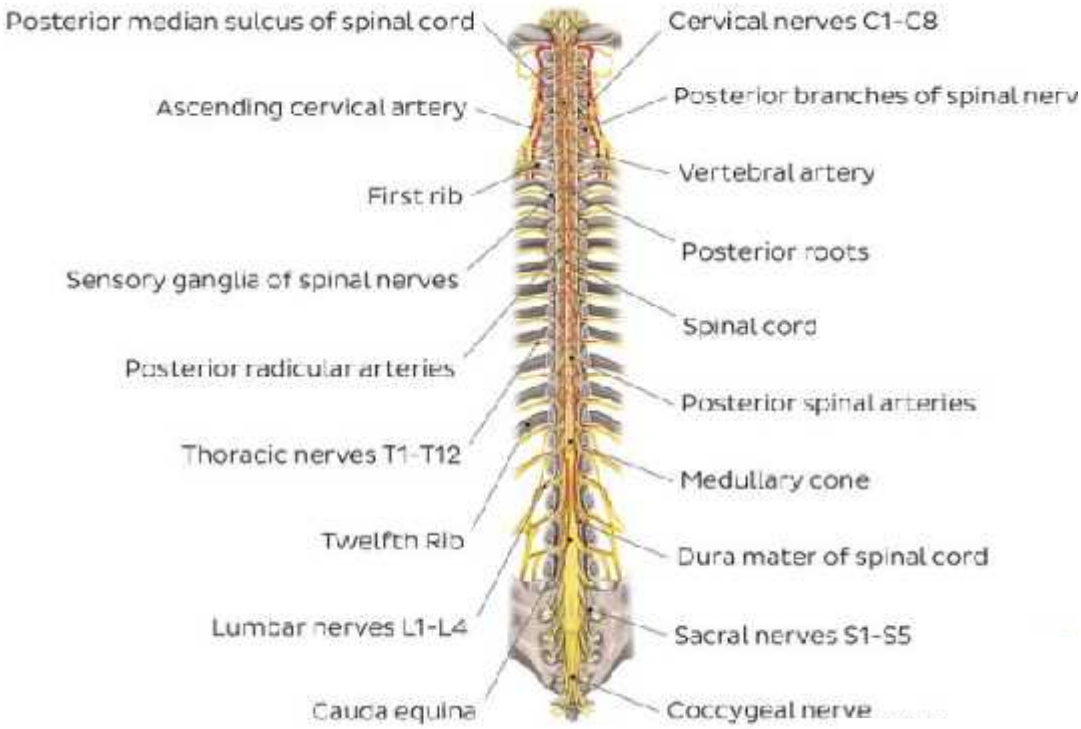


FIGURE 1:- STRUCTURE OF SPINAL CORD: CORONAL VIEW

CROSS-SECTIONAL ANATOMY:

It is made up of grey matter which is the central area and white matter which is the peripheral area.

GREY COLUMN:

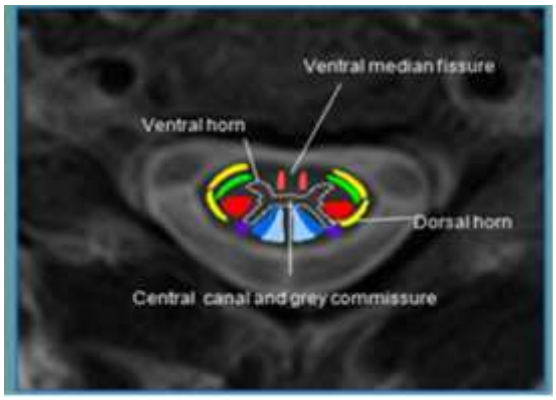


FIGURE 2 :- AXIAL T2W I OF SPINAL CORD

Its is in shape of butterfly made up of cell bodies. In transverse sections its divided into dorsal and ventral horns.

Divided into following columns:

TABLE 1-: COLUMNS OF GREY MATTER

DORSAL (posterior)	Procession of sensory stimuli
INTERMEDIATE (lateral)	Production of preganglionic sympathetic tracts
VENTRAL (anterior)	Cell bodies of alpha motor fibres

WHITE COLUMN:

Consists of myelinated nerve fibres and divided as: Anterior (Ventral) Funiculus, lateral funiculus and posterior (dorsal) funiculus.

On MR imaging axial images provide good morphologic delineation of the posterior elements of the spine, almost equivalent to late-generation CT images without magnification mode. The high signal intensity of the extradural fat pad between the ligamentum flavum and the posterior aspect of the thecal sac is almost always seen in thoracic regions. On coronal images, the normal curvature of the spine prohibits delineation of the spinal cord in a single image plane; scanning of multiple levels is usually required.

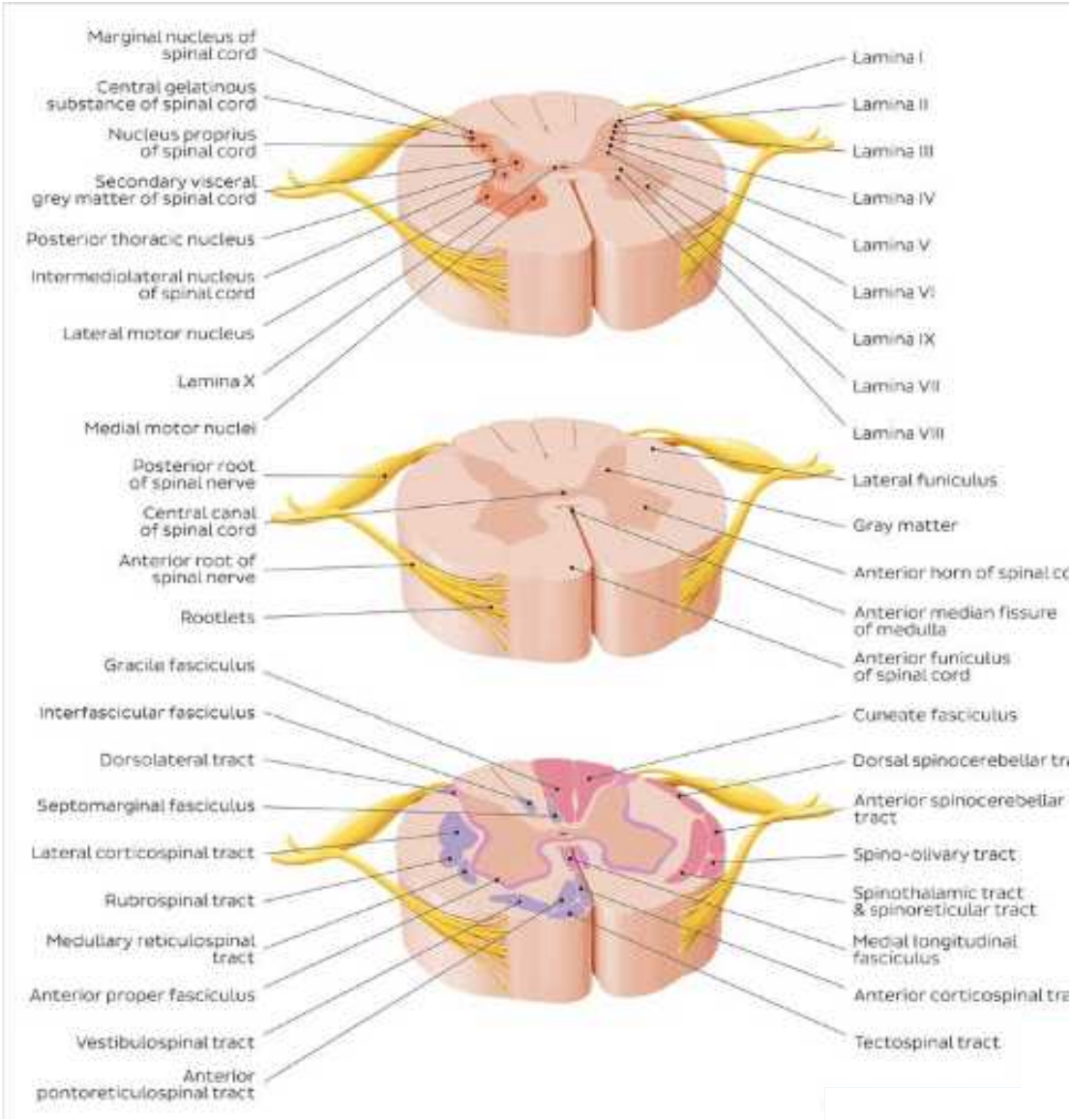


FIGURE 3 :-CROSS-SECTIONAL VIEW OF SPINAL CORD

MAJOR TRACTS OF THE SPINAL CORD:

Intramedullary anatomy comprises of white matter in the form of myelinated ascending (sensory) and descending (motor) tracts which are paired structures.

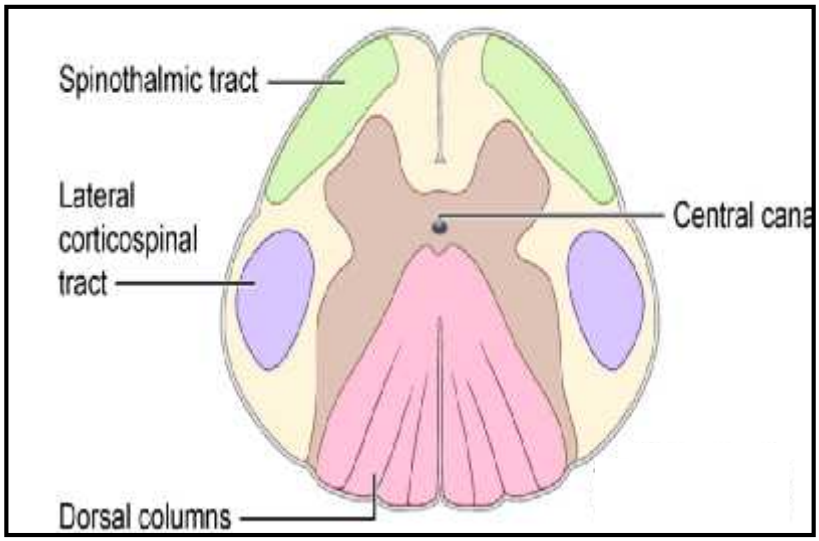


FIGURE 4 :- OVERVIEW OF PYRAMIDAL TRACTS

ASCENDING EFFERENT TRACTS:

Divided into lateral spinothalamic tract, ventral spinothalamic, dorsal column (medial lemniscus & cuneocerebellar. tracts) and spinocerebellar tracts.

TABLE 2-: ASCENDING EFFERENT TRACTS

LATERAL SPINOTHALAMIC	Touch, pain & temperature sensation
VENTRAL SPINOTHALAMIC	Crude touch & pressure
DORSAL COLUMN (MEDIAL LEMNISCUS & CUNEOCEREBELLAR. TRACTS) OTHALAMIC	Light, touch, vibration & proprioception.
SPINOCEREBELLAR	Unconscious stimuli for proprioception in joints & muscles

DESCENDING EFFERENT TRACTS:

Divided into lateral & ventral corticospinal, reticulospinal, rubrospinal, vestibulospinal and tectospinal tracts.

TABLE 3-: DESCENDING EFFERENT TRACTS

LATERAL & VENTRAL CORTICOSPINAL	Voluntary, skilled & motor activities
RETICULOSPINAL	Regulation of voluntary movements
RUBROSPINAL	Promotion of flexor & inhibitions extensors
VESTIBULOSPINAL	Promotes extensor & inhibits flexors
TECTOSPINAL	Provides postural movements

Pathology of the cord can be divided on basis of location:-

Extradural space

Intradural-extramedullary space

Intramedullary space

Our study is based on pathologies within the intramedullary space.

VASCULARIZATION OF SPINAL CORD

Spinal vascular supply consists of bilateral segmental arteries directly arising from aorta. Its dorsal branch supplies dura & nerve root and spinal cord & roots via radiculomedullary arteries.

Vertebral arteries give rise to:

Anterior spinal artery (1)

posterior spinal arteries (2)

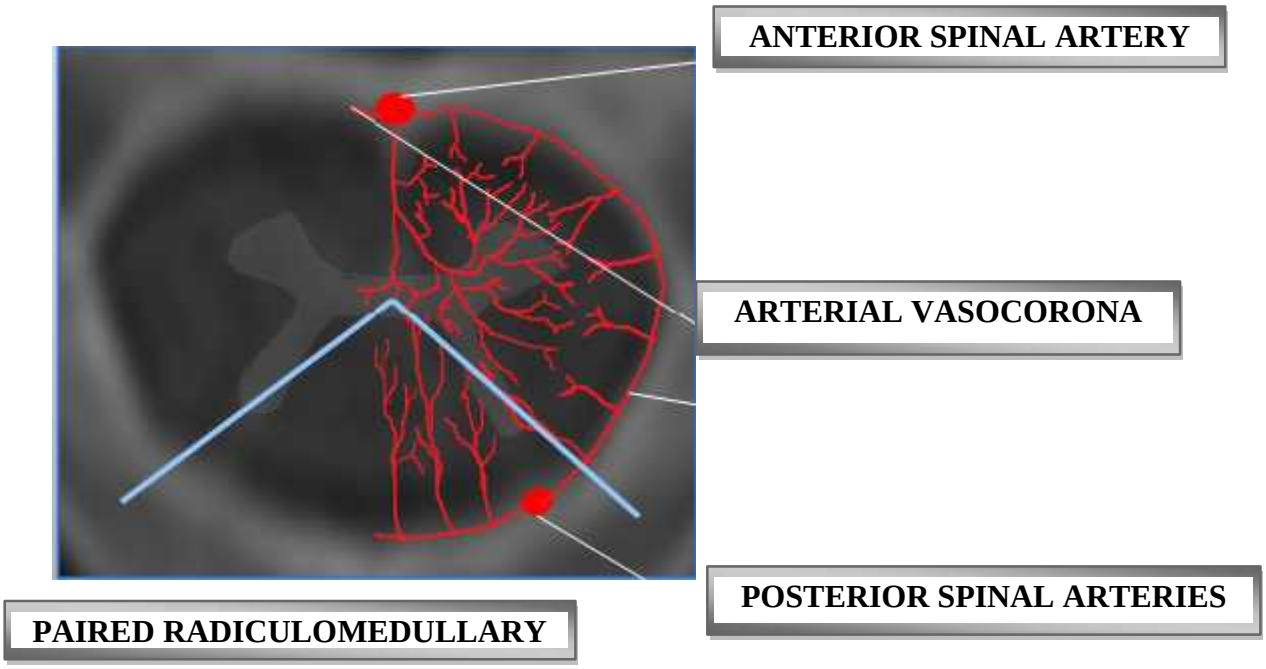
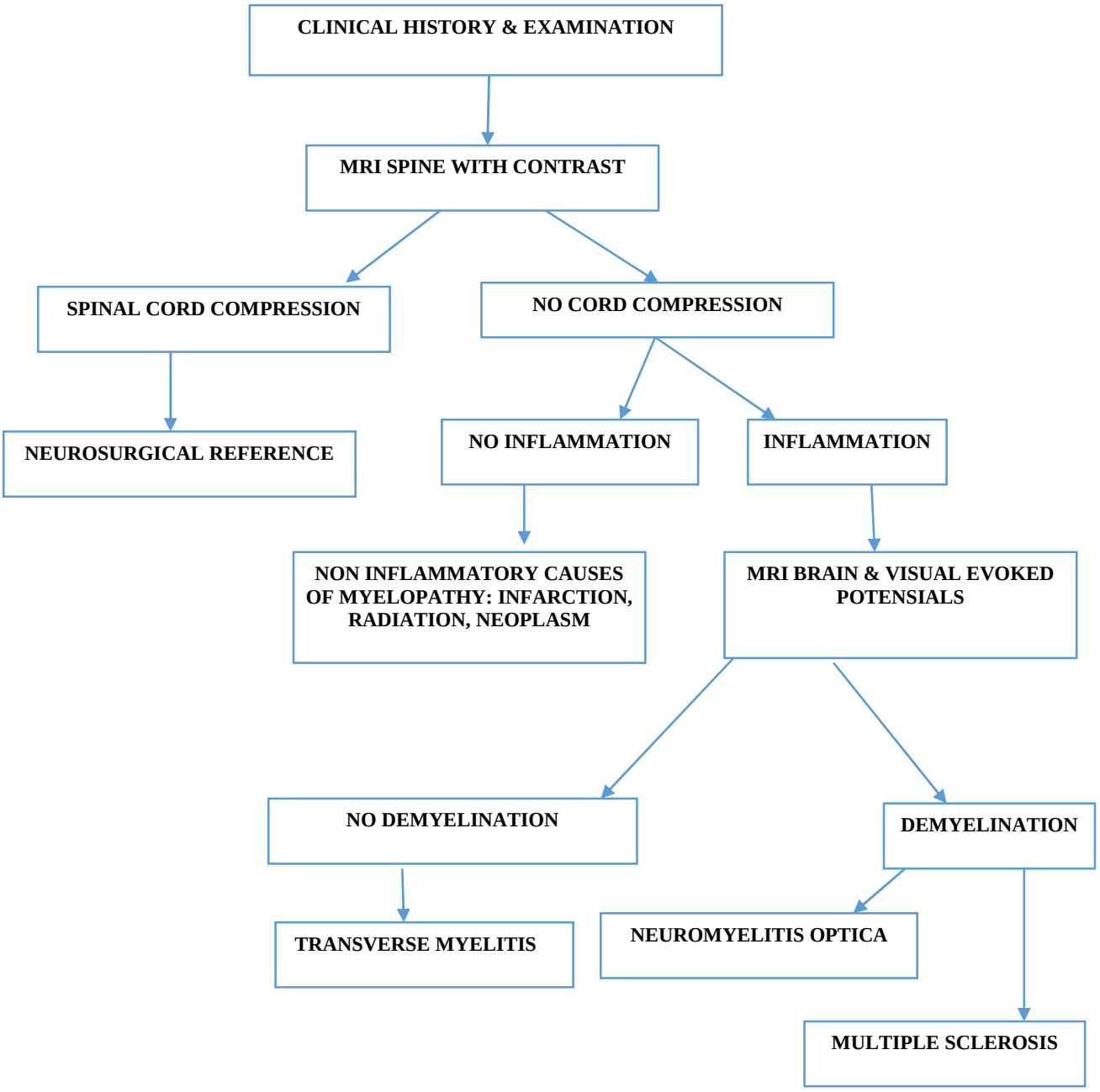


FIGURE 5:- VASCULARIZATION OF SPINAL CORD

ALGORITHM 1:- DIAGNOSTIC APPROACH TO CORD PATHOLOGIES



Differentiating intramedullary pathology is quite challenging but with help of MRI, differential diagnosis could be sorted down by identifying location of lesion, pattern of involvement and type of enhancement.

ANALYZING LESIONS BASED UPON LOCATION:

A): ANTERIOR SPINAL CORD

1. SPINAL CORD INFARCT-:

Many etiologies of cord infarction are present, which are divided according to transverse involvement of the cord in correspondence to the insulted vascular territory. Majority of them are dissection, atherosclerosis, aneurysm, vasculitis and aortic surgery.

On T2 W images, hyper intensities are noted involving grey matter, anterior horn cells and anterior part of cross sectional area of spinal cord. Involvement is usually more than 1 vertebral body segment. It usually involves anterior spinal artery territory.

2. MULTIPLE SCLEROSIS-:

It is a primary demyelinating disease affecting the CNS with both intracranial & spinal involvement. On brain imaging callososeptal, periventricular and perivenular demyelinating lesions are noted. Its clinical presentation is often acute. Its classified into relapsing-remitting, secondary progressive, primary progressive and progressive with relapsing types.

On MRI, lesions appear to be iso to hypointense and the lesions are hyperintense on T2 WI. On contrast study, active lesions show incomplete ring enhancement. On spectroscopy, NAA peaks may be reduced within the plaques. Demyelinating lesions can occur anywhere in the cord. Mechanism of myelopathy is that of inflammation which damages myelin sheath-forming cells. This could be primary like multiple sclerosis, secondary such as acute disseminated encephalomyelitis or transverse myelitis secondary to infection.

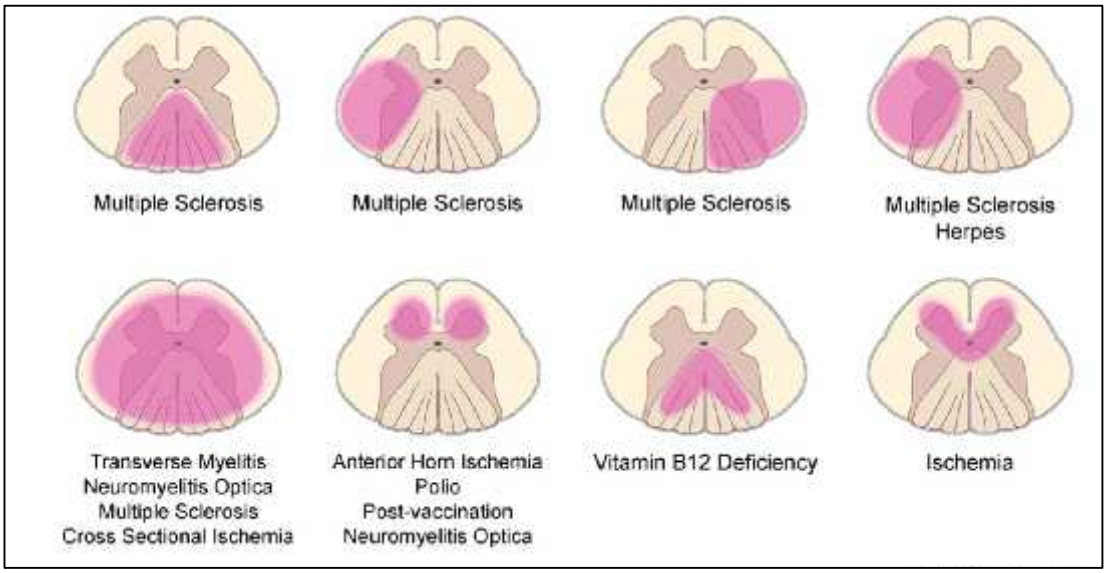


FIGURE 6:- DIFFERENTIATING INTRAMEDULLARY PATHOLOGY LOCATION WITHIN THE SPINAL CORD

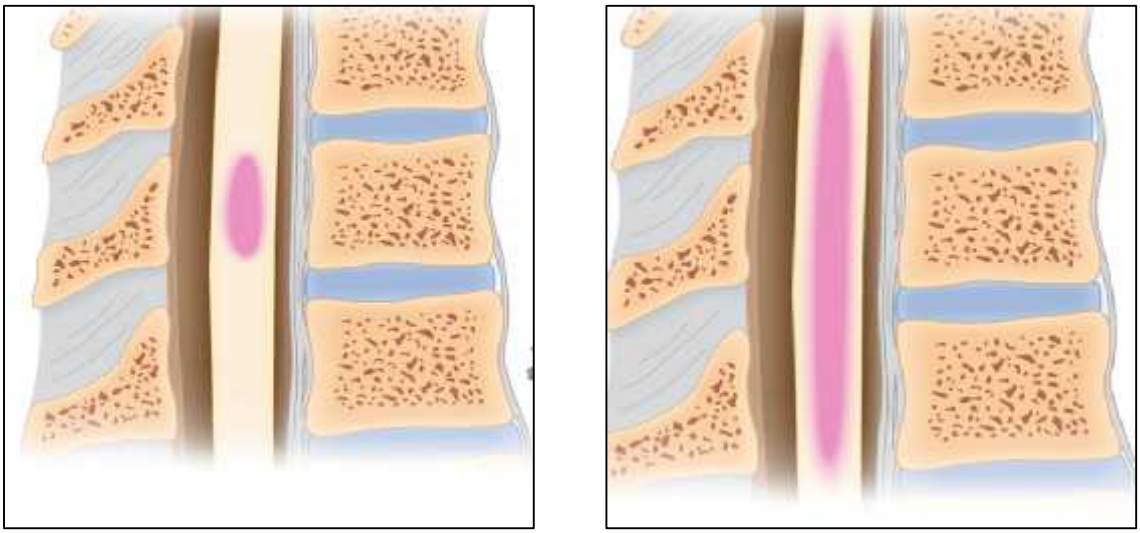


FIGURE 7:- DIFFERENTIATION BASED UPON SEGMENTS SHORT VS LONG

B)- POSTERIOR SPINAL CORD

3. SUBACUTE COMBINED DEGENERATION-:

It is associated with low VIT B12 levels. On imaging, cord expansion with T2W hyperintensity noted involving dorsal columns / lateral columns. Most common location is lower cervical and upper thoracic cord. On contrast mild enhancement is present. However, confirmatory diagnosis is done after laboratory findings.

4. HIV MYELOPATHY-:

Most common clinical presentation is sudden onset of lower limb weakness. Similar findings as SACD but most common location is at the thoracic level. On MRI, symmetrical T2 hyperintensity noted involving white matter tracts associated with atrophy. Contrast imaging shows patchy enhancement.

5. RELAPSING REMITTING MULTIPLE SCLEROSIS:-

Most common location is cervical cord. On MR imaging, T2 W hyper intense lesions are located within dorsolateral aspect of cord. Most of the cases show 2 vertebral segments involvement. Lesions could be solitary / multiple. On contrast study homogenous / ring / nodular enhancement noted. Cord expansion noted in acute stage with lesions showing variable enhancement while on chronic stage atrophy is noted with no enhancement. On brain screening periventricular, subcallosal, brain stem & cerebellar lesions are present in 90 % of cases.

C. CENTRAL SPINAL CORD:-

6. SYRINGOHYDROMYELIA / HYDROMYELIA

Its dilatation of central canal and is associated with chiari I malformation in which peg like herniation of cerebellar tonsil is noted below the foramen magnum.

7. SYRINGOMYELIA-

Its any CSF intensity cavity within the cord.

Both of them shows intramedullary lesions of CSF intensity on T1 W images.

CAUSES OF SPINAL CORD SWELLING / EXPANSION COULD BE DIVIDED INTO:

Acute: Acute myelitis, infection and ischemia.

Chronic: Syrinx

Tumour: intramedullary, metastasis

8. CORD MYELOMALACIA:-

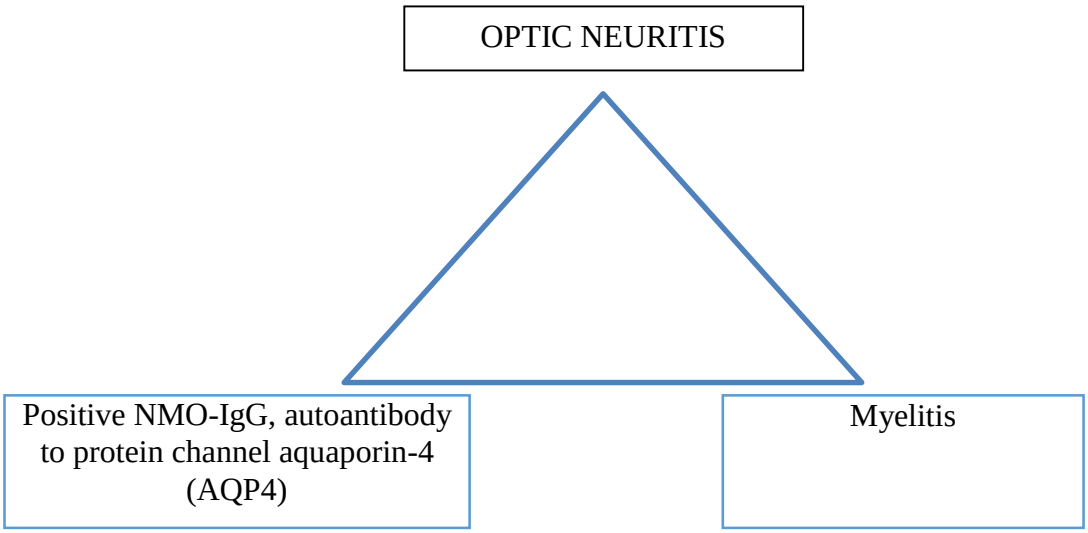
Clinical presentation included sensory deficits. Central canal narrowing with spinal stenosis is noted resulting in focal T2 hyper intensity at that respective disc level. Most common cause is ossification of the posterior longitudinal ligament.

9. TRANSVERSE MYELITIS:-

Its an inflammatory condition with various causes like post radiation, idiopathic and secondary to infection. Patients present with paraesthesia. On MRI, long segment T2 hyper intense cord lesion noted in the centre with bilateral cord involvement. Commonly involves more than 3-4 vertebral segments. Cord expansion is present. Peripheral enhancement is often noted on contrast studies. Most common location is thoracic. Brain screening is often done to rule out ADEM / MS

10. NEUROMYELITIS OPTICA (DEVICS DISEASE)

ALGORITHM 2:- CRITERIA OF DIAGNOSIS FOR NMO:



On MR imaging, shows longitudinally extensive spinal cord lesions extending for 3 vertebral bodies noted. Expansion of cord is present. Brain imaging shows lesions & optic neuritis.

11. ANAPLASTIC ASTROCYTOMA:

Location, length and pattern of enhancement helps in differentiation of all tumours Astrocytoma is an Infiltrating lesion which is hyper intense on T2 W images. long segment is often involved and shows heterogeneous enhancement. Most common location is cervical with cord expansion.

12. MYXOPAPILLARY EPENDYMOMA:

Its a well circumscribed lesion which shows T2 hyper intensity. Typical location is conus and involves 2-4 vertebral segments.On contrast imaging, homogenous enhancement is noted.Rim of hemosiderin deposition at rostral margin referred to as “cap sign” is noted.

D. CENTRAL SPINAL CORD WITH FLOW VOIDS:-

13. ARTERIO VENOUS MALFORMATION:-

Clinical symptoms include spastic paralysis followed by flaccid paralysis. It consists of arteriovenous capillary nidus which is fed by enlarged artery and drained via an enlarged venous plexus .MRI shows long area of T2 hyper intensity. Most common location is within 'cervical or thoracic' cord.

Multiple flow voids noted around cord representing dilated vessels which enhance on contrast images.

14. DURAL ARTERIOVENOUS FISTULA

On imaging, flame shaped area of hyper intensity noted on T2 W images. Dilatation of pial veins noted as flow voids around cord. Cord expansion is often noted. Most common location is conus.

15. HEMANGIOBLASTOMA:

Its the third most frequently seen intramedullary cord tumor. They are infrequent benign tumors. Most common location is in cerebellum in association with Von Hippel Lindau syndrome (VHL). Frequently arises in thoracic cord followed by 'cervical'. Shows long segment cord edema. Flow voids are noted due to feeding arteries and draining veins. On contrast study, heterogeneous enhancement noted in larger lesions.

DISTRIBUTION OF LESIONS BASED ON SEGMENTAL LENGTH:-

Neuromyelitis optica, infarct, tumour and transverse myelitis are characterised under long segment involvement whereas multiple sclerosis under short segment.

TABLE 4-: DISTRIBUTION OF LESIONS BASED ON SEGMENTAL LENGTH

LONG SEGMENT	SHORT SEGMENT
NMO	MS
INFARCT	
TUMOUR	
TM	

TABLE 5-: SUMMARY OF ALL DEMYELINATING LESIONS

DISEASES	LOCATION	LENGTH	ENHANCEMENT
MS	DORSAL	Short	Y
NMO	CENTRAL	Long	PATCHY
ADEM	DIFFUSE	Long	N
TM	>2/3 rd	Short / long	VARIABLE

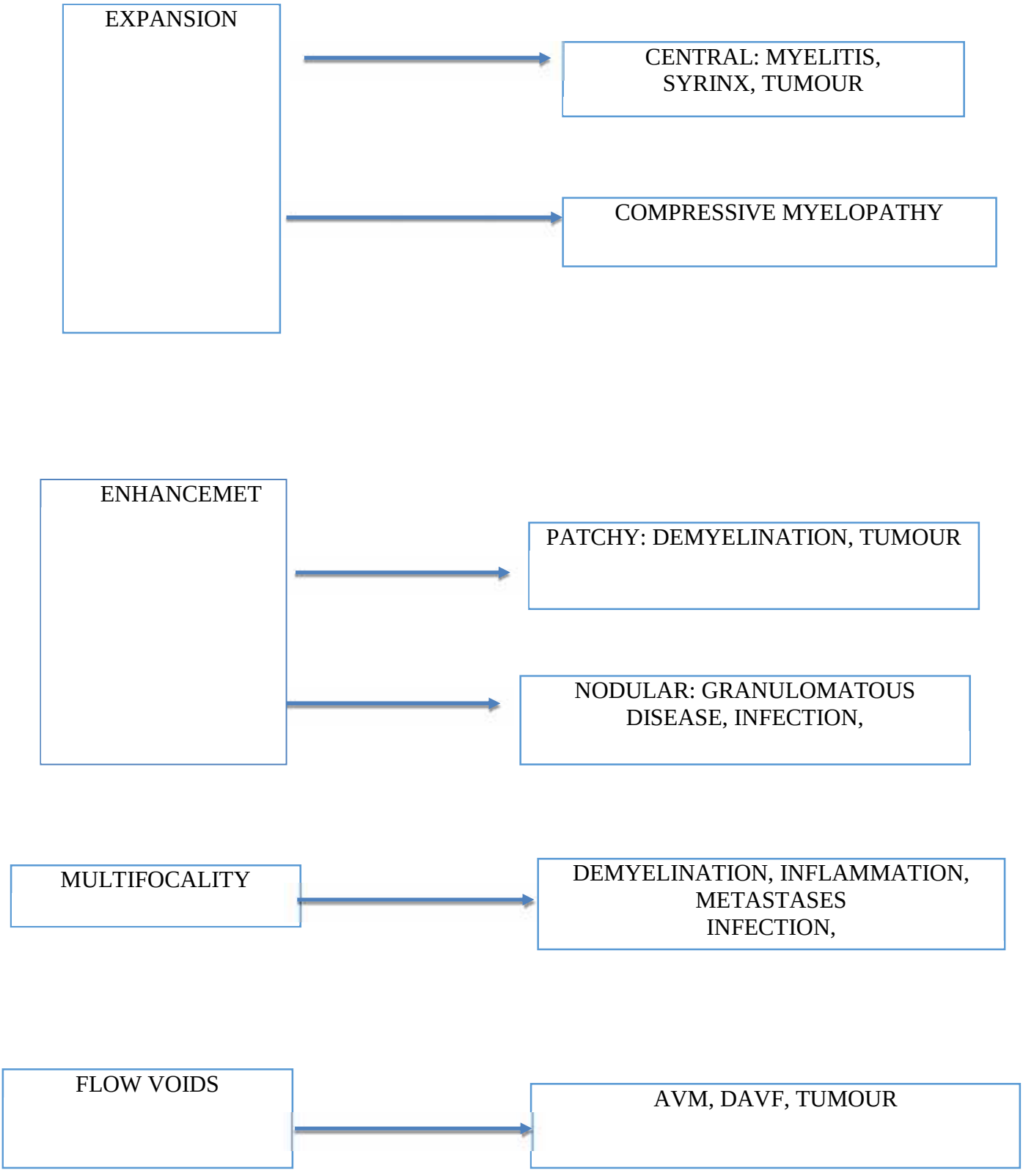
TABLE 6-: SUMMARY OF ALL NEOPLASTIC LESIONS:

LESIONS	LOCATION	VERTEBRAL BODIES	ENHANCEMENT
Astrocytoma	T>C	4-7	HE
Ependymoma	C>T	4	HE
Hemangioblastoma	T>C	SHORT	NODULAR
Metastasis	C>T>L	MULTIPLE	HE

TABLE 7-: SUMMARY OF VASCULAR LESIONS:

LESIONS	LOCATION	LENGTH	ENHANCEMENT
AVM	DORSAL	SHORT	HE
DAF	VARIABLE	LONG	+/-
INFARCTION	ANTERIOR CORD	SHORT	VARIABLE
CAVERNOMA	T>C	LONG	MINIMAL

ALGORITHM 3:- EVALUATION OF LESIONS IN SPINAL CORD



On imaging of the entire spinal cord T2 hyper intensity is at times difficult to give an accurate diagnosis. Hence, there is a need of study to approach systematically that incorporates distribution of the signal abnormalities, including length of cord involvement and the area that is affected which helps narrowing down the list of differential diagnosis.[1]

A study was conducted by Bou-Haider P. et al. [2,3] on differential diagnosis of T2 hyperintense spinal cord lesions in which the aim was to review common conditions which cause high signal intensity on T2 weighted images and outline a systematic approach to diagnosis with reference to pertinent features.

They explained in their study that the causes of increased signal intensities on T2 weighted images can be broadly classified as: trauma, neoplastic conditions, primary demyelination, ischemic, infective, granulomatous and cavitary lesions of the spinal cord. Their study concluded that characterization of abnormal areas in T2 W images in combination with patients symptoms lead to a unique diagnosis.

Jane Watts.et al. [4] conducted a study in which they mentioned many conditions of cord lesions resulting in similar imaging findings on MRI, and hence it is essential to understand the anatomy of cord, imaging features of lesions and presentation of clinical symptoms in detail in order to diagnose correctly.

Pekcevik, Y.et al. [5] conducted a study to investigate the spinal MRI findings of longitudinally extensive transverse myelitis which helps differentiating neuromyelitis optica from MS. They conducted studies on 94 patients with LETM. Lesions in NMO patients involved more than half of the cords cross-sectional area, centrally and peripherally located in the cord. The presence of 'T1 dark' signal and BSL was higher in NMO patients. Differentiating LETM from NMO is very important to start early immunosuppressant therapy.

Wingerchuk DM.et al. [6] conducted a study to evaluate the various spectrum of neuromyelitis optica (NMO), including characteristics of the optic neuritis (ON) and myelitis, neuroimaging, CSF, and serologic studies, and to evaluate the long-term course.

JL Kitley.et al. [7] described that longitudinal myelitis means inflammation of cord resulting in T2 hyper intensity on MRI. Early identification is important in order to provide proper treatment. Clinico-laboratory correlation helps in distinguishing inflammatory versus non-inflammatory causes.

K H Taber.et al. [8] reported that MRI imaging of the spine is of great importance inspite of many artifacts which would come across such as chemical shift, motion artifacts arising from respiration & swallowing and because of nonuniform magnetic field.

Use of motion compensation and fat saturation helps in reducing artifacts. The technician should use correct surface coil, FOV and sequences in order to minimize the risk of artifacts.

Anthony Bozzo et al. [9] stated the perfect use of MRI in managing patients with cord injury in which sagittal T2W image was of great importance. There are seen increase vertebral artery injuries in cases of spinal cord injuries at the cervical vertebrae level.

T Kameyama et al. [10] conducted studies on T2 hyper intense spinal cord lesions in cases of compressive myelopathy. They noted that this particular finding was related to severity of clinical symptoms and degree of cord compression. The background varied from focal edema to myelomalacia. Most often medulla spinalis was compressed at several levels but high signal intensity was found at a single level where compression was highest.

Martin J. Fine, MD et al. [11] reported MR imaging characteristics of spinal intramedullary ependymomas.

Naohiro Tachibana et al.[12] conducted studies on cord lesions in cases of compressive myelopathy which presented as snake-eye appearance on T2W images. In their studies the term myelomalacia was described as ill defined area of T2 hyper intense and T1 hypo intense signal changes.

Kenzo Uchida et al. [13] conducted a study on changes noted in spinal cord signal intensities on MRI in patients with compressive causes in which they quantified signal intensity and correlated intramedullary signal changes on MRI T1- and T2-weighted images with clinical outcome and prognosis.

Kim G.et al. [14] conducted a study on multiple sclerosis in which they compared the spinal cord volume on T1 &T2 W based images in healthy subjects. Their sample size was 31. For T1 cord area the mean difference was less as compared to T2 cord area. They concluded by saying that T2-weighted images are excellent in measuring cord atrophy in multiple sclerosis patients.

A study was conducted to determine the MR appearance of spinal cord multiple sclerosis (MS) plaques in patients presenting with myelopathy. They concluded that spinal cord demyelinating plaques presented as well defined foci of increased T2 signal that asymmetrically involve the spinal cord parenchyma. Knowledge of their usual appearance prevent unnecessary biopsy. MR imaging of brain confirm the imaging suggestion of spinal cord demyelinating disease. [15]

A study was conducted in Kerala, India. They have done case series in which 1 patient had symptoms with dorsal cord involvement with paraparesis, vitamin B12 level was found to be low, MRI T2 hyperintensities involved “lateral & posterior” columns and another patient presented with paresthesias. Vitamin B12 level was

found to be normal whereas MRI showed posterior column involvement and. In third patient, had no neurologic complaints were noted. Serum B12 level was low. MRI findings revealed bilateral paired areas of T2 hyper intensity noted as an "inverted V" in the dorsal columns. [16]

A study was conducted on approach to a case of myeloneuropathy in which they encountered conditions which are diagnostic challenges. Giving the correct diagnosis of myeloneuropathy is of atmost importance because myelopathies lead to great confusion. Once myeloneuropathy is identified, all the patients should undergo clinical tests. The biochemical results and pattern of involvement aids in diagnosing correctly. [17]

M Vorgerd et al. [18] conducted a study on progressive myeloneuropathy in association with vitamin E deficiency in which they described patients clinical symptoms. On laboratory investigations he had severe vitamin E deficiency. MRI of the cervical spine showed T2 hyperintensities in posterior columns.

A study was conducted which stated that early treatment in myelopathy due to vitamin B12 deficiency results in reversal. Since it has a variety of clinical symptoms early diagnosis and treatment are vital. They described a case with sensory symptoms with low serum vitamin B12 levels. On MRI, myelopathy was diagnosed. Without further delay he was treated with cyanocobalamin injections. On repeat MRI, resolution of findings noted. [19]

K. Yamada et al. [20] conducted studies on sub acute degeneration of spinal cord in which MRI revealed lesions in posterior and lateral columns, involving the cortico-spinal tracts. They reported a case with history of tingling in bilateral hands. MRI showed symmetrical areas of T2 signal abnormality involving the dorsal

columns of the cervical cord. On treatment with vitamin B12 supplements gradual improvement noted. Repeat MRI was done which showed decrease in abnormal signals.

S G Srikanth et al.[21] conducted studies on MRI in subacute combined degeneration of spinal cord.

Michel E. Mawad et al. [22] conducted a study in which they concluded that MRI is modality of choice for diagnosing spinal cord infarction.

J L Sherman et al. [23] conducted a study on MR appearance of syringomyelia. They studied 58 patients in which 24 of them were communicating syringomyelia. 16 patients had posttraumatic syringomyelia, 9 patients associated tumors and nine of them had idiopathic syringomyelia. Imaging findings included T2 hyperintensity, CSF flow-void sign in syrinx cavity and finally cord enlargement. Most common location was cervicothoracic junction. The theories of syrinx development and propagation were reviewed.

L M Tartaglino et al. [24] conducted studies on Idiopathic acute transverse myelitis: MR imaging findings in which they analysed MRI findings in idiopathic acute transverse myelitis (IATM) in relation to pathologic findings and MR findings in Guillain-Barré syndrome and ischemia. They concluded that MR findings were atmost similar in both the conditions which suggest a possible relationship.

Stefan Weidauer et al. [25] analyzed ischemic spinal cord lesions with the help of imaging and clinical symptoms. Total of 16 patients were taken. MRI was performed. Pencil-like hyperintensities and cord enlargement were noted on sagittal T2W images. Contrary to the presumed spinal cord watershed at the lower cervical and upper thoracic level, and despite numerous central arteries in the cervical cord, their data suggested a high ischemic vulnerability of the cervical spinal cord.

A study conducted on imaging of spinal cord ependymomas in which 60 subjects were considered. Length of the lesions was measured from 1 to 10 vertebral segments. All of them presented with hyperintense signals on T2W MR images. 75% of them had homogenous contrast enhancement. In 90% of them had rostral cysts which were T1 hypointense and hyperintense on T2-weighted images. Most common location of ependymomas was cervical cord. [26]

J L Kitley et al. [27] conducted studies on differential diagnosis of longitudinally extensive transverse myelitis which is inflammation of the spinal cord resulting in T2 hyperintensity on MRI extending over 3 or more vertebrae. Identification in very early stage with the help of imaging & laboratory findings while sorting out the underlying cause is essential in initiating correct therapy. Its main stay on treatment depends upon whether the condition is inflammatory or non-inflammatory.

Gina M. Lowe et al. [28] stated that MRI has significant impact on the diagnosis of cord tumors by providing excellent localization of tumor and by defining its characteristics. They have conducted studies on MRI of spinal cord tumors in which they stated that MRI has significantly changed the detection and diagnosis of intramedullary spinal cord tumors. With its multiplanar capabilities and high contrast resolution, it allows identification and characterization of the lesion in a noninvasive fashion. The addition of intravenous contrast allows further delineation of the mass, separating tumor from edema and cysts. In addition there are newer pulse sequences such as magnetic resonance spectroscopy (MRS) which may someday play a role in diagnosis, particularly in excluding other etiologies for abnormal MRI signal such as demyelination, inflammation, or infarction.

A study was conducted on intramedullary spinal cord metastases: diagnosis

and treatment. They reported 284 patients with ISCM out of which 32 had been treated surgically. Survival rate increased in patients who underwent surgery rather then who got treated conservatively. ISCM occur rarely but their frequency is increased due to increase use of MRI. [29]

R I Grossman et.al. [30] assessed spinal cord damage in MS using MRI. Techniques used in assessing damage to the cord included: hyper intensities on T2W images, enhancement of cord and atrophy. Latest methods such as magnetization transfer, diffusion and proton spectroscopy aids in specific diagnosis of MS.

Most common cause of SACD is low vitamin B12 levels. On imaging, posterior and lateral columns of spinal cord are noted with high signals on T2W images. After treatment clinical improvement was noted with normal imaging on repeat MRI.

S S Junger et al. [31] conducted studies on intramedullary spinal sarcoidosis: clinical and magnetic resonance imaging characteristics in which they presented retrospective series of the clinical and MRI findings in 16 patients with ISS. MRI findings were “leptomeningeal enhancement”, cord expansion and cord atrophy.

Studies were conducted on axonal damage in the spinal cord of MS patients which shows largely independent of T2 MRI lesions To determine the degree of axonal damage in relationship to signal abnormalities on T2-weighted high-resolution MRI in spinal cord tissue of patients with MS. the major finding is that widespread axonal damage and loss occurs in the spinal cord, largely independent of the findings on T2-weighted MRI. Acute inflammation can affect the cord signal on T2-weighted images, but all cord lesions were histologically old lesions without acute inflammation so this will not have influenced the correlation. Whereas abnormal signal on T2-weighted MR closely reflects the extent of demyelination,2 axonal pathology apparently occurs relatively independent of demyelination, as has also been

suggested in a recent histopathologic study.9 In this study axonal loss and increase of axonal diameter in both totally demyelinated areas as well as in focally demyelinated areas and NACT of MS patients was found. Three other studies showed axonal loss up to 80 to 85% in MS plaques in the spinal cord10, 11 and increased axonal diameter in lesions. [32]

L M Tartaglino et.al. [33] conducted studies on multiple sclerosis in the spinal cord: MR appearance and correlation with clinical parameters in which their purpose was to determine the peculiar MR features of that affected the spinal cord.

Florian Roser et al. [34] studied cases on defining the line between hydromyelia and syringomyelia which is possible based on electrophysiological and magnetic resonance imaging studies. Hydromyelia patients do not share same clinical & radiological features with syringomyelia patients. Hence, these conditions must be separated with an underlying disorder.

Studies were conducted on The MR appearance of syringomyelia. Total of 58 patients with cord cavities were included in this study. On T2- and T1-weighted images characteristics of each syrinx and cerebellar tonsils were analyzed. Imaging features in some patients included T2 hyper intense areas, CSF flow-void sign (CFVS), eccentric cavities, beaded cavities and cord enlargement. T1-weighted images were helpful in identifying anatomy of syrinx & cerebellar tonsils. Most common location was cervicothoracic junction. 5 to 9 vertebral segments was the average involvement. [35]

Daniel D. Do-Dai et al. [36] conducted studies on imaging of the spinal Cord tumours. Its very important to differentiate between benign and neoplastic process. In this article they described benign etiologies such as syrinx, contusion, abscess, infarction, myelitis, multiple sclerosis and arteriovenous malformation whereas

neoplastic conditions such as dermoid tumor, astrocytoma, ependymoma, hemangioblastoma, lymphoma and metastases. MRI features as well their correlation with clinical context was also described.

A recent study proposed criteria for idiopathic ATM leading to a relatively homogenous group in terms of clinical and MRI data but even if severe initial symptoms are correlated with a poor prognosis, the outcome remains unpredictable. There is therefore a need for serum or CSF markers to identify patients with a high risk of poor outcome before therapeutic trials in idiopathic ATM can be performed. Several studies have looked for CSF markers 14-3-3 protein, neuron specific enolase, and others) in acute myelopathies, but they did not focus on idiopathic ATM, and the results were contradictory.In the current study, they found that severe initial symptoms suggesting spinal shock were highly predictive of a poor outcome, in accordance with previous studies on ATM which did not distinguish between the various etiologies [37].

B Hemmer et al. [38] conducted studies on SACD in which MRI was done before and after the treatment. Most common symptoms were in association with posterior column.

On T2W I hyper intense lesions were detected in posterior column. After treating with cobalamin all patients showed good recovery. They concluded that vitamin B12 deficiency should be considered in differentiating all spinal cord disorders.

Studies were conducted on spinal arteriovenous malformation. Study was conducted on 10 patients with AVMs using MRI. Findings such as myelomalacia, thrombosis and wall thickening of draining vein--were demonstrated & confirmed. Flow-sensitive sequences were helpful in the depiction of AVM as hyperintense and

differenting nidus from old hematoma. MRI showed various cord abnormalities that no other imaging could detect. [39]

K B Baker et al. [40] conducted studies on MR imaging of spinal hemangioblastoma Its the most common primary neoplasm of cerebellum in adults, but its presentation in spine is quite rare .Preoperative imaging limits to some extent of surgery . Variety of appearances on imaging have been described like its association with cyst or syrinx, diffuse cord enlargement and minimal spinal cord reaction. Hemangioblastoma of spine presents with T1 hypo intensity and T2 hyperintensity. On contrast imaging intense enhancement was noted. Administration of contrast is vital as small lesions are often missed whereas large lesions are visualized with no contrast material.

Kieran J. Murphy et al. [41] conducted studies on spinal cord infection: Myelitis and abscess formation. In their study they tried to differentiate abscess from other cord infections based upon imaging. Study subjects were 6 with mean age of 38 years. On imaging cord hyper intensity on T2W I and on contrast images marginal enhancement with central hypo intensity was noted. After the start of treatment T2 signals reduced & on contrast ring enhancement was noted. Finally, they concluded that characteristic sequence of imaging findings aids in the differentiation of cord infection from other intramedullary lesions.

Christine Goh et al. [42] reviewed neuroimaging in acute transverse myelitis. Being an acute condition its pathogenicity is divided into 4 categories. On imaging, pattern of enhancement on contrast images and the area of cord involvement helps to understand the underlying causes. Hence, its important to analyse the subgroups which would aid in treatment.

A Campi et al. [43] conducted studies on acute transverse myelopathy. They intended to evaluate MRI in helping out the causes & frequency of lesions. They tried to find out the specific types of intracranial lesions detected at the onset of disease. In about 30 patients they conducted both spinal and brain imaging. Contrast study was done only in 10 patients. MR findings reported abnormal in 14 patients. They concluded that MR contribution was only see in 40% of the total cases.

Kerslake RW et al (1991) [44] reported that spinal trauma is a common and devastating event with important long term sequelae for both the individual and society. It is in patients with incomplete neurological deficits that MR imaging in the acute phase offers the most potential practical benefit.

Kulkami et al (1987) [45] described three patterns of cord damage in acutely injured patients corresponding to:

1. Central haemorrhage which increased with time
2. Central petechial haemorrhage resolving with time
3. Edema and contusion only

Cervical spinal cord hemorrhage has a negative effect on lower extremity motor function.

Pattern I (MC) intraspinal hemorrhage.

Pattern II -Spinal cord edema

Pattern III- Combination of intraspinal hemorrhage and edema.

They concluded that patients with low intensity on T2WI had a poor prognosis. Many factors affects prognosis. The degree of cord compression appears to be the most important factor. Some patients with severe cord compression showed normal intensity on TI and T2WI had a good prognosis

Most patients who showed abnormal intensity on T2WI did not recover later.

Hyper intensity itself on T2WI seems non -specific.

The intensity on T2WI is considered to be a useful sign for clinical follow up. Hyper intensity disappears in patients with good recovery, while hyper intensity was unchanged in patients with poor prognosis.

Sarah M et al.[46] conducted studies on systematic approach to differentiate intramedullary spinal cord lesions. They stated spinal cord abnormalities are diagnostic challenges for radiologists. Hence, detailed focus of lesion location, cord length and segment involvement is essential as well as enhancement pattern to narrow down differential diagnosis.

MATERIALS AND METHODS

Study site: This study was conducted in the Department of Radio diagnosis at The KLE'S DR. PRABHAKAR KORE Hospital & MRC, BELGAVI.

Study population: All the patients referred to Department Of Radio-Diagnosis with clinically suspected spinal cord lesions at the KLE'S DR. PRABHAKAR Kore Hospital & MRC, BELGAVI were considered as the study population.

Study design: The current study was a observational study

Sample size: Prevalence rate of T2 hyperintensities in spinal cord lesions in Indian population in most of the studies done is very less, hence the sample size would be very large which is quite difficult to attain in our hospital. Therefore all diagnosed cases of spinal cord lesions over a period of one year at KLE'S DR Prabhakar Kore Hospital & Medical Research Centre, BELAGAVI would be taken as the sample size. Average number of patients within the last two years span with spinal cord lesions showing T2 hyperintesities, referred to the department of radio-diagnosis, was found to be around 25-35 each year.

My intended study was carried out on 53 patients visiting the OPD / IPD referred to MRI scan to the department of radio diagnosis at KLE'S DR Prabhakar Kore Hospital & Medical Research Centre, BELAGAVI for a period of 12 month duration.

Sampling method: Universal sampling.

Study duration: The data collection for the study was done between January 2019 to December 2019 for a period of 1 year.

Inclusion Criteria:

All the patients with clinical findings such as pain, motor deficits, sensory deficits, abnormal reflex and urinary dysfunction who undergo MRI and are found to have T2 hyperintense spinal cord lesions.

Exclusion criteria:

- Non consenting subjects
- Patients with past history of prior surgery of spinal cord.
- Patients who had claustrophobia, freshly implanted MRI incompatible prosthesis. Patients with aneurysm clips, ferromagnetic implants and pacemakers.

Ethical considerations: Study was approved by the institutional human ethics committee. Informed written consent was obtained from all the study participants, and only those participants willing to sign the informed consent were included in the study. The risks and benefits involved in the study and voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Methodology:

Patients with spinal cord abnormalities were referred to the department of radio diagnosis were included in the study. The clinical and demographical data was collected from requisitions forms which were referred to the department of radio diagnosis for patient's MRI scan of the spine, and the MRI findings were recorded on the preformatted forms. Data collection includes age, sex, history, clinical examination, biochemical findings and x-ray spine. MRI spine of all the patients was taken using 1.5 Tesla MRI scanner (Magnetom Avanto TIM, 18 channel; Siemens, Erlangen, Germany) and 3.0 Tesla MRI scanner (Magnetom Spectra, 32 channel; Siemens, Erlangen, Germany) with the help of a dedicated spine coil with the patient in supine position, and entire spine will be examined.T1W, T2W, STIR sequences were used in the axial and sagittal planes. Post contrast T1W, Diffusion-weighted sequences and ADC sequences were used whenever possible.

Following parameters were delineated - vertebral body involvement, irregularity of end plates, marrow edema, prevertebral collection, paravertebral collection, spinal cord compression, epidural involvement, subligamentous spread and other significant findings if any.

Statistical methods:

Descriptive analysis: Descriptive analysis was carried out by the mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram and pie diagram.

RESULTS

A total of 53 people were included in the analysis

T**able 8: Descriptive analysis of Age (Years) in the study population (N=53)**

Age groups	No of patients	% of patients
<=20yrs	7	13.21
21-30yrs	8	15.09
31-40yrs	7	13.21
41-50yrs	9	16.98
51-60yrs	12	22.64
>=61yrs	10	18.87
Total	53	100.00
Mean age	44.45	
SD age	17.61	

In this study population, the mean of age was44.45 years. (Table8)

Among the study population, 7 (13.21%) cases were aged less than 20 years, 7(13.21%) cases were aged 31 to 40 years, 9 (16.98%) cases were aged between 41 to 50 years, 12 (22.64%) cases were aged between 51 to 60 years, 10(18.87%) cases were aged more than 61 years and above. (Table 8&graph 1)

Graph 1: Pie chart for age wise distribution of patients

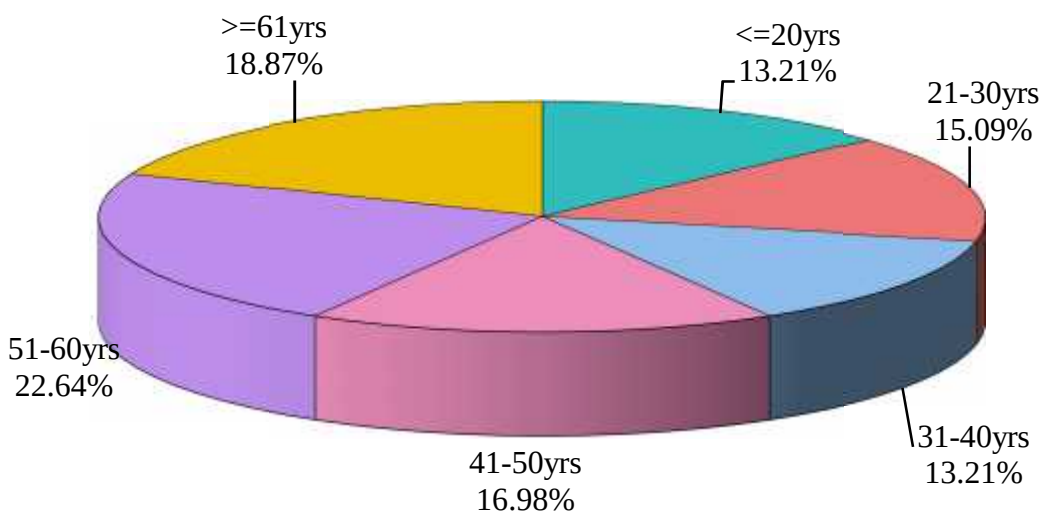


Table 9: Descriptive analysis of gender in the study population (N=53)

Gender	No of patients	% of patients
Male	28	52.83
Female	25	47.17
Total	53	100.00

Among the study population, 28(52.83%) cases were males and remaining 25(47.17%) cases were females. (Table 9&graph 2)

Graph 2: Pie chart for gender wise distribution of patients

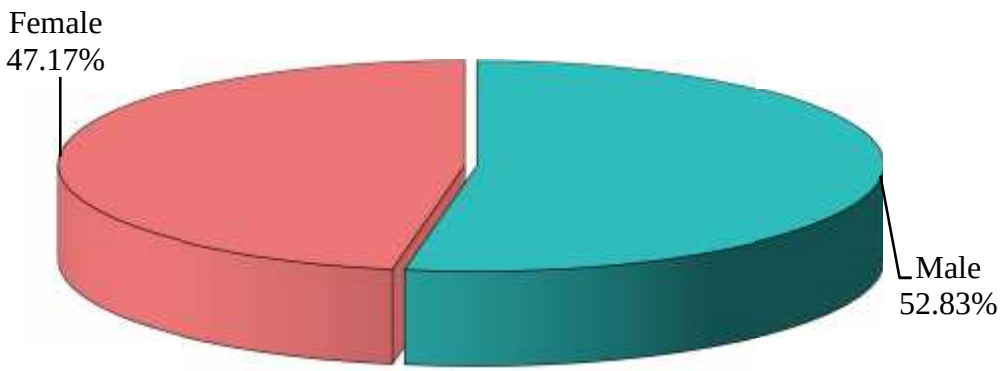
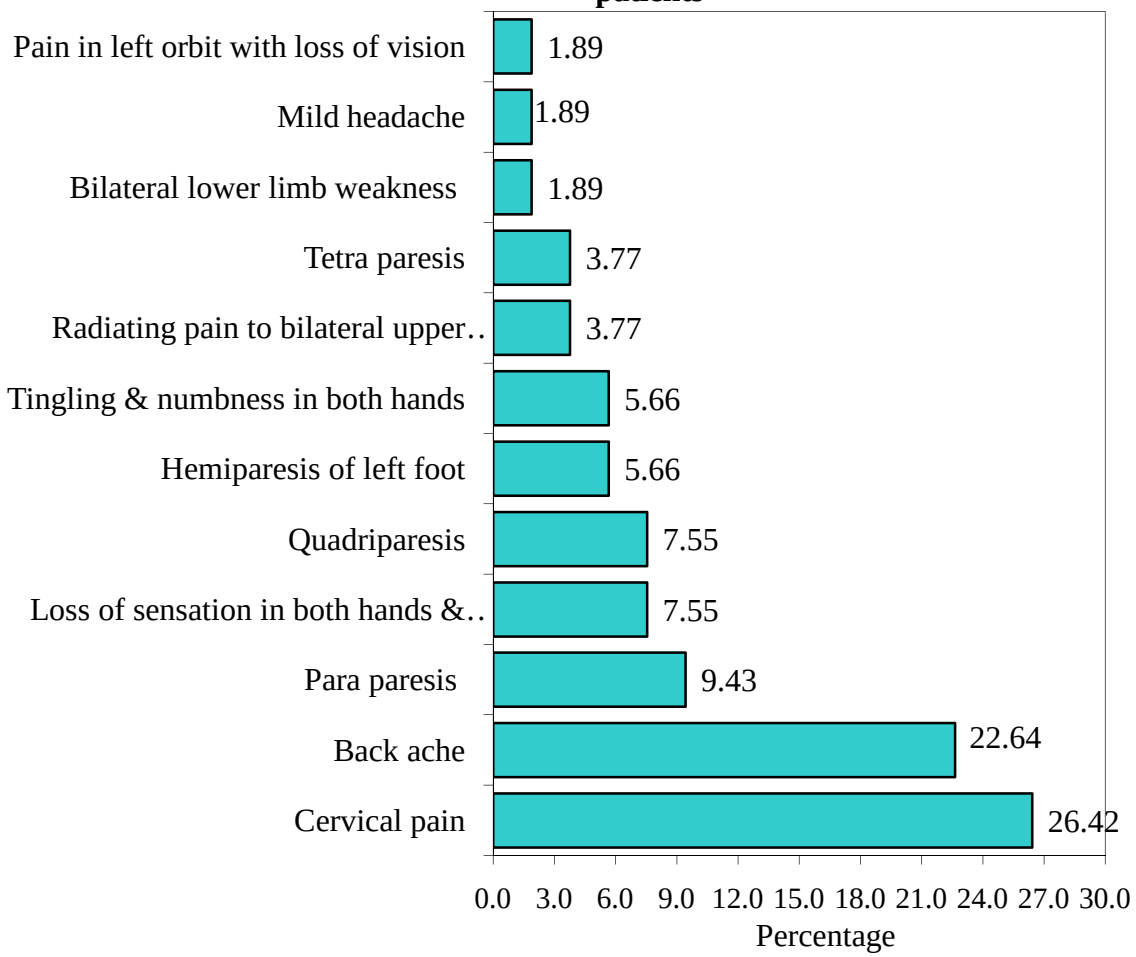


Table 10: Descriptive analysis of clinical symptoms in the study population (N=53)

Clinical symptoms	No of patients	% of patients
Back ache	12	22.64
Bilateral lower limb weakness	1	1.89
Cervical pain	14	26.42
Hemiparesis of left foot	3	5.66
Loss of sensation in both hands & feet	4	7.55
Mild headache	1	1.89
Pain in left orbit with loss of vision	1	1.89
Para paresis	5	9.43
Quadriparesis	4	7.55
Radiating pain to bilateral upper limbs	2	3.77
Tetra paresis	2	3.77
Tingling & numbness in both hands	3	5.66
No complaints	1	1.89
Total	53	100.00

Graph 3: Bar graph for clinical symptoms wise distribution of patients



Among the study population,the most common symptom was cervical pain in 14 (26.42%) casesfollowed by backache in 12(22.64%) cases, para paresis in 5 (9.43%) cases, quadriparesis in 4 (7.55%) cases, Tingling & numbness in both handsin 3 (5.66%), Pain in left orbit with loss of vision in 1 (1.89 %) casesand mild headache in only 1(1.89%) case. (Table 10&graph 3)

Table 11: Descriptive analysis ofduration of symptoms in study population. (N=53)

DURATION	No of patients	% of patients
ACUTE	12	22.64
SUBACUTE	13	24.52
CHRONIC	28	52.83
Total	53	100.00

Among the study population, most frequent duration of symptoms are chronic 28 (52.83%), followed by subacute 13 (24.52%) and acute 12 (22.64%) cases. (Table 11&graph 4)

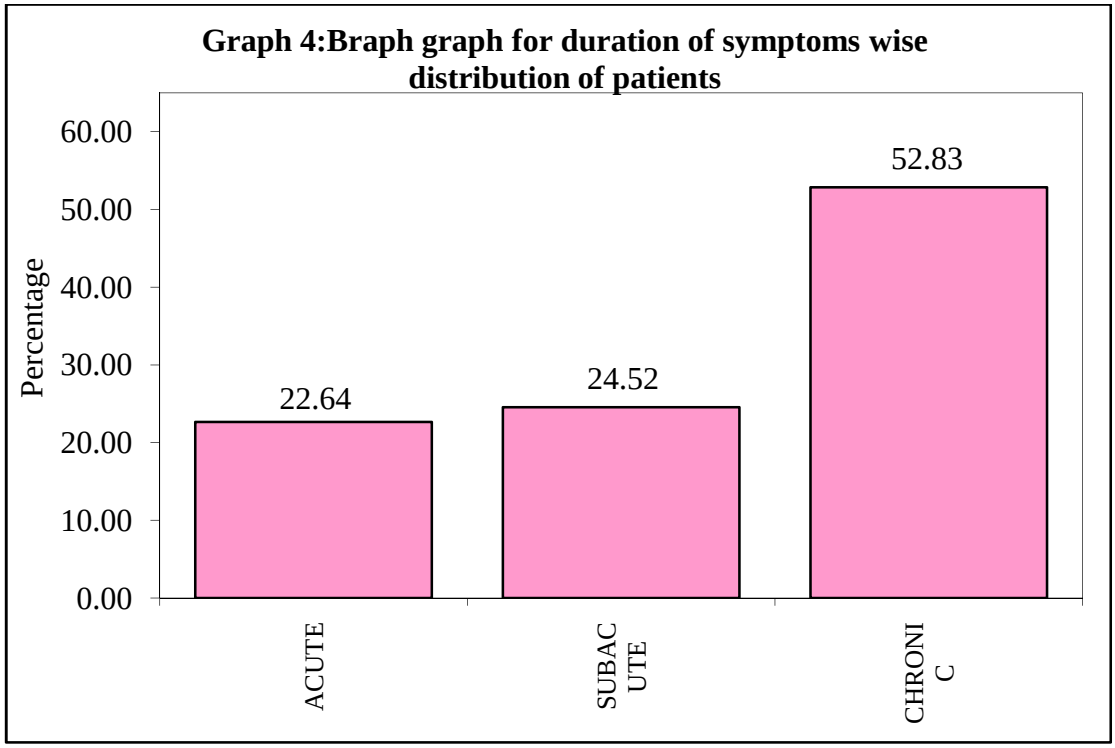


Table 12: Descriptive analysis of radiographs wise distribution of patients in the study population (N=53)

Findings of radiographs	No of patients	% of patients
Normal	12	22.64
Abnormal	24	45.28
Not done	17	32.08
Total	53	100.00

Among the study population, 12 (22.64%) cases showed normal findings and 24 (45.28%) cases showed abnormal findings. In 17 (32.08%) patientsX rays were not taken. (Table 12& graph 5)

Graph 5: Pie chart for findings of radiographs wise distribution of patients

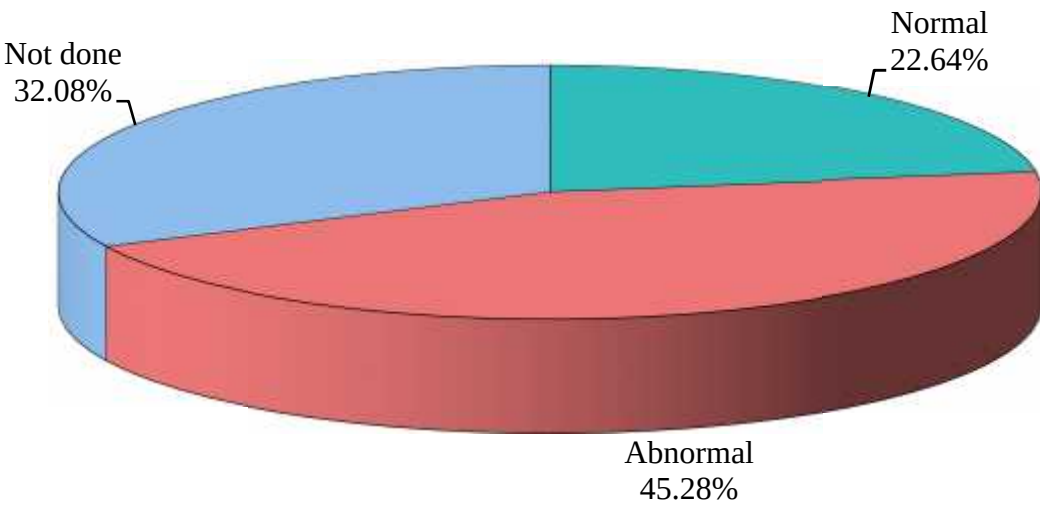


Table 13: Descriptive analysis of lesion wise distribution of patientsin the study population (N=53)

Levels of lesion	No of patients	% of patients
Cervical	33	62.26
Thoracic	11	20.75
Lower Thoracic	11	20.75
Lumbar	3	5.66
Cervicomedullary junction	1	1.89
Upper Thoracic	2	3.77

Among the study population,most commonly involved level of lesions was cervical in 33 (62.26%) cases, followed by thoracic in 11 (20.75%), lumbar in 3 (5.66)and cervicomedullary junction in only 1 (1.89) case. (Table 13& graph 5).

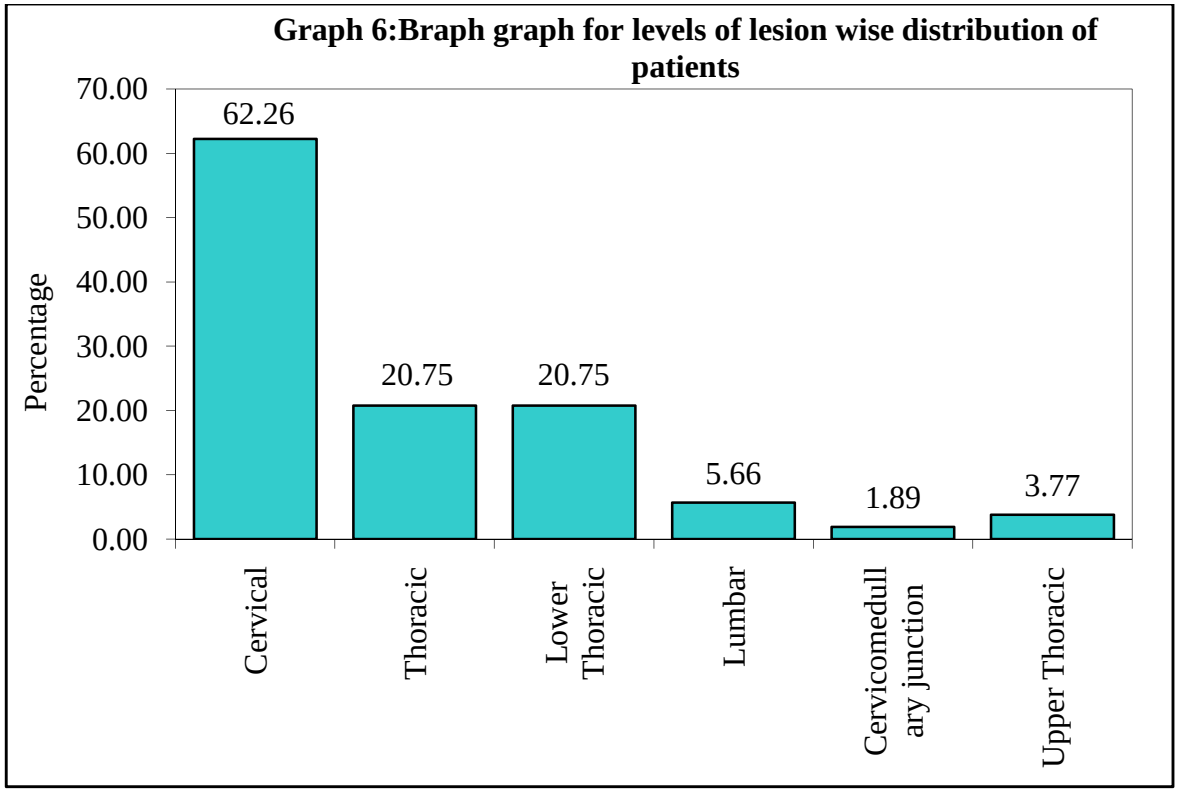


Table 14: Descriptive analysis ofLocation of lesions in spinal cord in study population. (N=53)

LOCATION	No of patients	% of patients
ANTERIOR	2	3.77
CENTRAL	49	92.45
POSTERIOR	2	3.77
Total	53	100.00

Among the study population, most common location is central 49 (92.45 %), anterior 2 (3.77%) and posterior 2 (3.77 %) cases. (Table 14&graph 7)

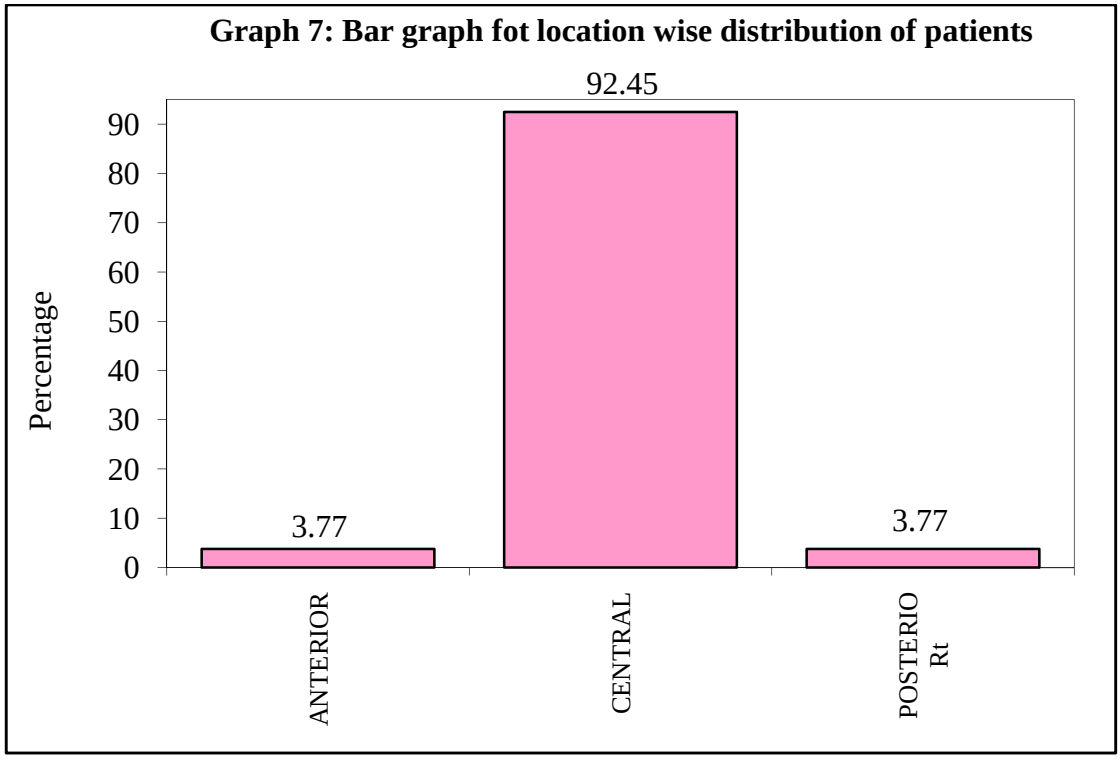


Table 15: Descriptive analysis of causes of SC compression in the study population (N=53)

Cause of SC compression	No of patients	% of patients
Cord edema	1	1.89
Disc Bulge	5	9.43
Disc Protrusion	2	3.77
Extra Medullary Lesion	1	1.89
Extra Dural Soft Tissue	1	1.89
Lesion	1	1.89
Osteophytic Disc Complex	2	3.77
Wedge Compression Fracture	1	1.89

Among the study population, commoncause of spinal cord compression was disc bulge 5(9.43 %) followed by disc protrusion 2(3.77%) cases. (Table 15&graph 8

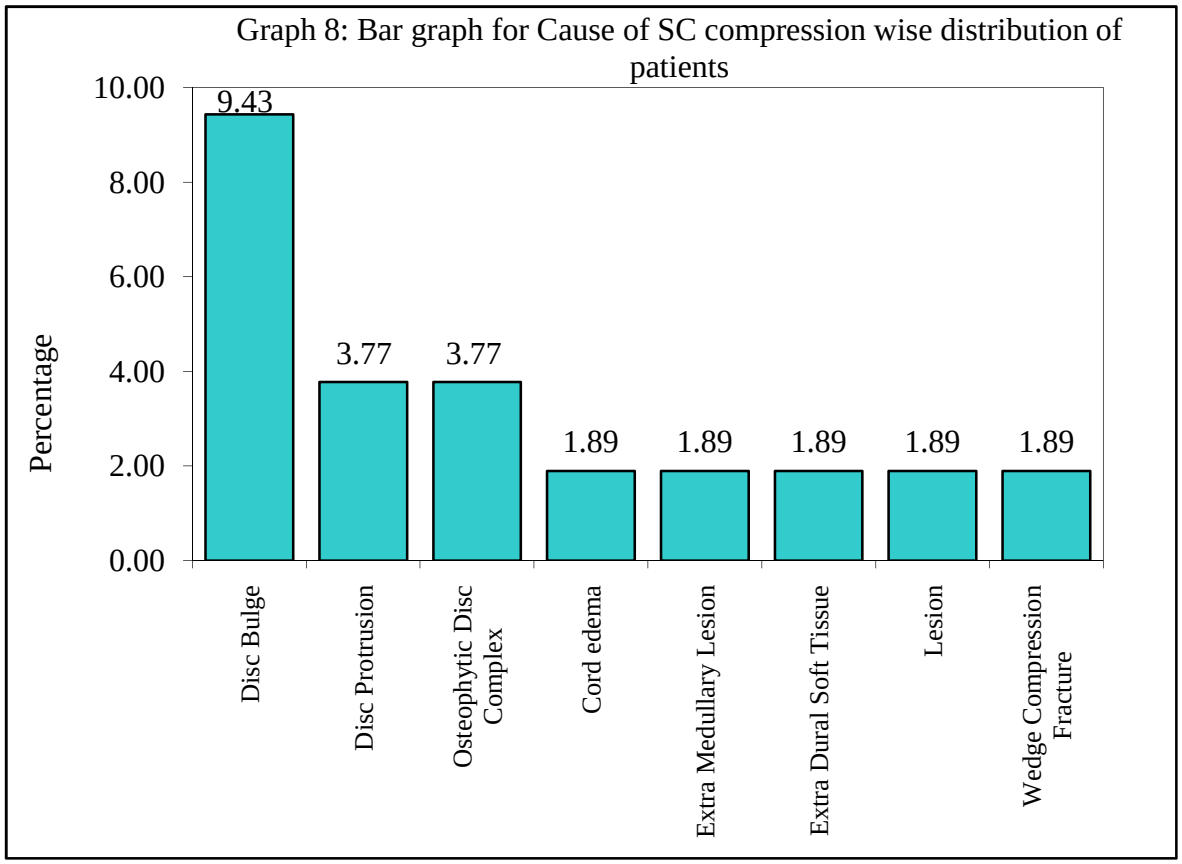


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Table 16: Descriptive analysis of post contrast in study population. (N=53)

Post contrast	No of patients	% of patients
Homogenous enhancement	3	5.660
Non enhancement	11	20.755
Peripheral enhancement	3	5.660
Not done	36	67.925
Total	53	100.00

Among the study population, homogenous enhancement was seen in 3(5.66%), Peripheral enhancementin 3(5.66%), no enhancement in11 (20.75) and contrast studies not done in 36 (67.92). (Table 16 &graph 9)

Graph 9: Pie chart for post contrast wise distribution

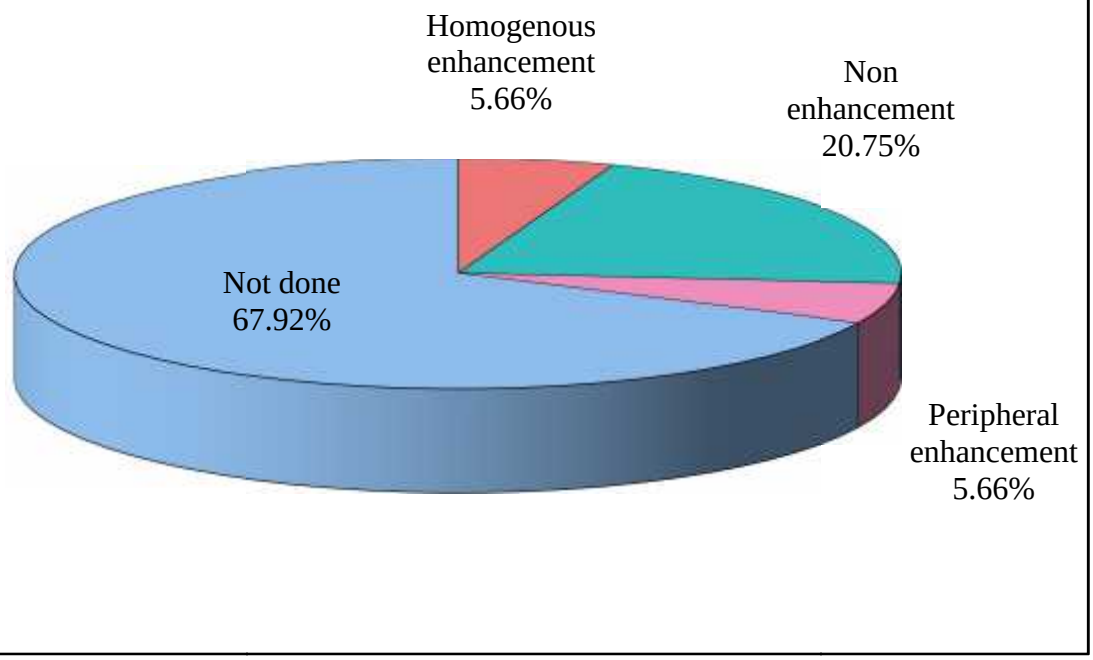


Table 17: Descriptive analysis of Cord changes in study population. (N=53)

Cord changes	No of patients	% of patients
Negative	4	7.55
Positive	49	92.45
Total	53	100.00

Among the study population, positive cord changes noted in 3(5.66%), Peripheral enhancementin 49 (92.45%) and no cord changesin4 (7.55). (Table 17&graph 10)

Graph 10: Pie chart for cord changes wise distribution of patients

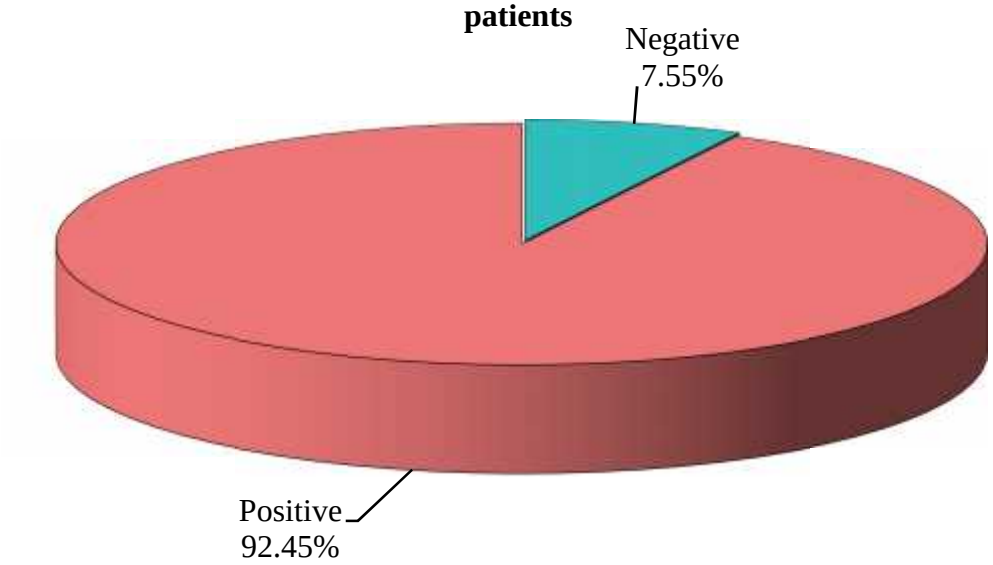


Table 18: Descriptive analysis of Ligamentous disruption in study population. (N=53)

Ligamentous disruption	No of patients	% of patients
Negative	36	67.92
Positive	17	32.08
Total	53	100.00

Among the study population, ligamentous disruption noted in 17 (32.08%) and no ligamentous disruption in 36 (67.92%) cases. (Table 18&graph 11).

Graph 11: Pie chart for ligamentous disruption wise distribution of patients

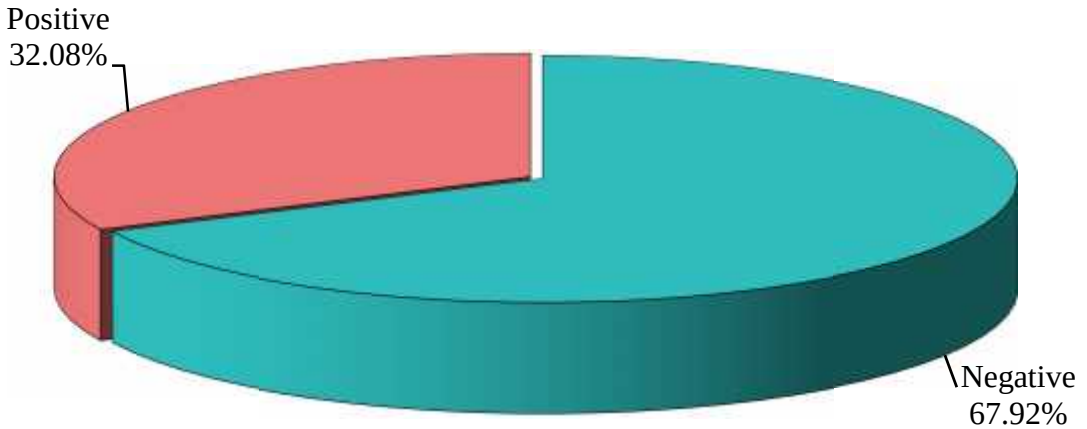


Table 19: Descriptive analysis of posterior elements in study population. (N=53)

Posterior elements	No of patients	% of patients
Abnormal	27	50.94
Normal	26	49.06
Total	53	100.00

Among the study population, 27 (50.94%) cases had abnormal findings and 26 (49.06%) cases had normal findings. (Table 19&graph 12)

Graph 12: Pie chart for posterior elements wise distribution of patients

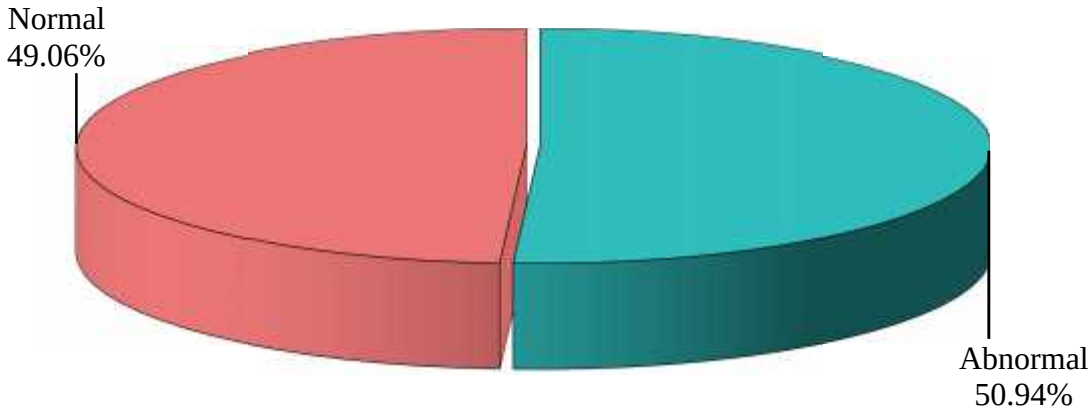


Table 20: Descriptive analysis of pre & para vertebral collection in study population. (N=53)

Pre & para vertebral collection	No of patients	% of patients
No	46	86.79
Yes	7	13.21
Total	53	100.00

Among the study population, Pre & para vertebral collection seen in 46 (86.79%) and no collection in 7 (13.21%) cases.(Table 20&graph 13)

Graph 13: Pie chart for pre & para vertebral collection wise distribution of patients

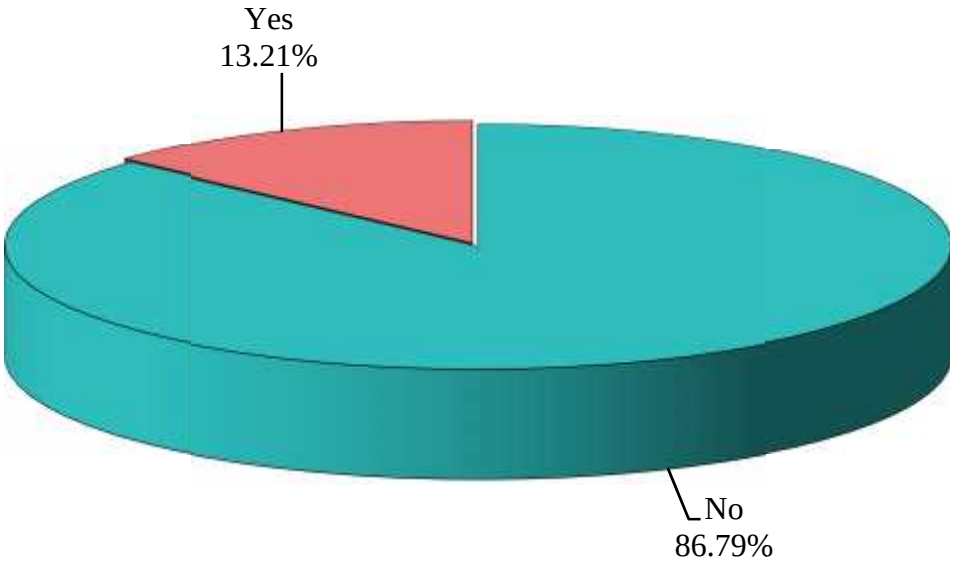


Table 21: Descriptive analysis of MR diagnosis in study population. (N=53)

MR diagnosis	No of patients	% of patients
Astrocytoma	1	1.89
Central Cord Syndrome	2	3.77
Cord Edema	2	3.77
Cord Myelomalacia	15	28.30
Ependymoma	1	1.89
Demyelinating disease	1	1.89
Multiple sclerosis	1	1.89
Metastases	1	1.89
Transverse Myelitis	10	18.87
Neoplasm	2	3.77
Secondary myelitis	1	1.89
Sub acute combined degeneration	2	3.77
Spinal cord infarct	1	1.89
Spinal epidermoid cyst	1	1.89
Syrinx	11	20.75
Syrinx with Cord Myelomalacia	1	1.89
Total	53	100.00

Among the study population, most common MR diagnosis is cord Myelomalacia 16 (30.18%), followed by syrinx 11 (20.75 %), transverse Myelitis 10 (18.87%), neoplasm, sub acute combined degeneration, cord edema&central cord syndrome comprised of 2 (3.77 %) of cases and demyelinating disease, ependymoma and astrocytoma comprised of 1 (1.89 %) of cases. (Table 21&graph 14)

Graph14: Bar graph for MR diagnosis wise distribution of patients

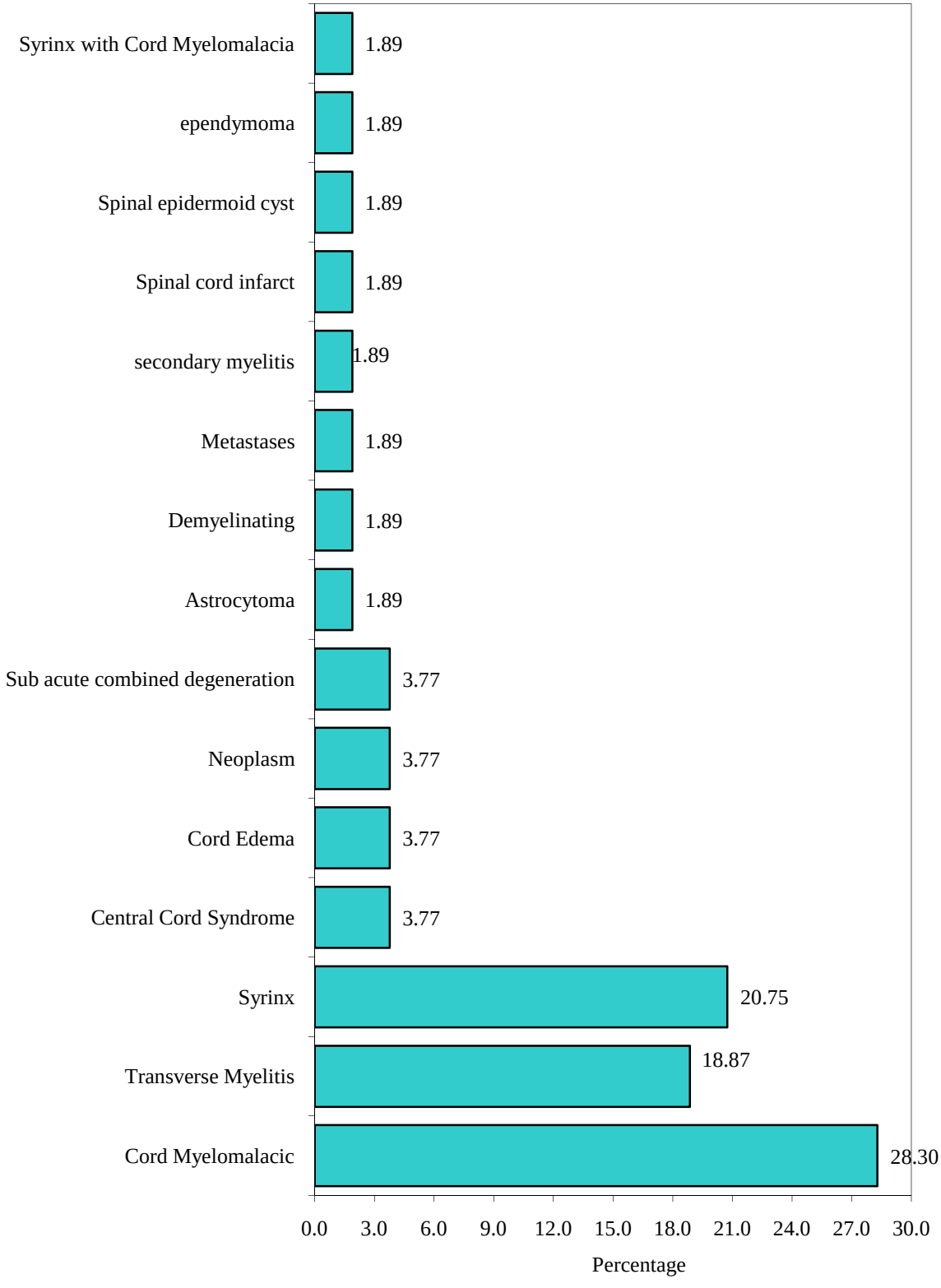


Table 22: Descriptive analysis of follow upin study population. (N=53)

Follow up	No of patients	% of patients
No	11	20.75
Yes	37	69.81
Expired	5	9.43
Total	53	100.00

Among the study population, follow up cases were 37 (69.81 %), not followed up are 11 (20.75 %) and expired 5 (9.43 %).(Table 22&graph 15)

Graph 15: Pie chart for Follow up wise distribution of patients

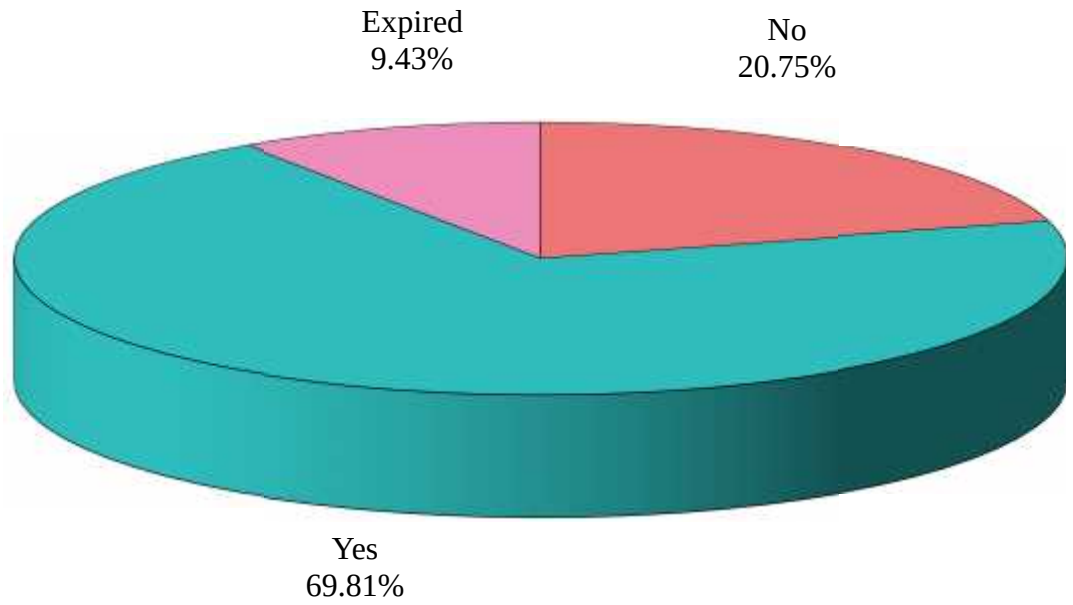


Table 23: Descriptive analysis of Remarks in study population. (N=53)

Remarks	No of patients	% of patients
Acute disseminated encephalomyelitis	1	1.89
Correct diagnosis	31	58.49
Equivocal	6	11.32
Multiple sclerosis	2	3.77
Spinal cord infarct	1	1.89
Spinal cord astrocytoma& lymphoma	3	5.66

Among the study population, Correct diagnosis in 31 (58.49 %) ,equivocal 6 (11.32%), multiple sclerosis12 (3.77 %),SCA & lymphoma3 (5.66 %) , Spinal cord infarct1 (1.89 %) and ADEM in 1 (1.89 %) cases.(Table 23&graph 16)

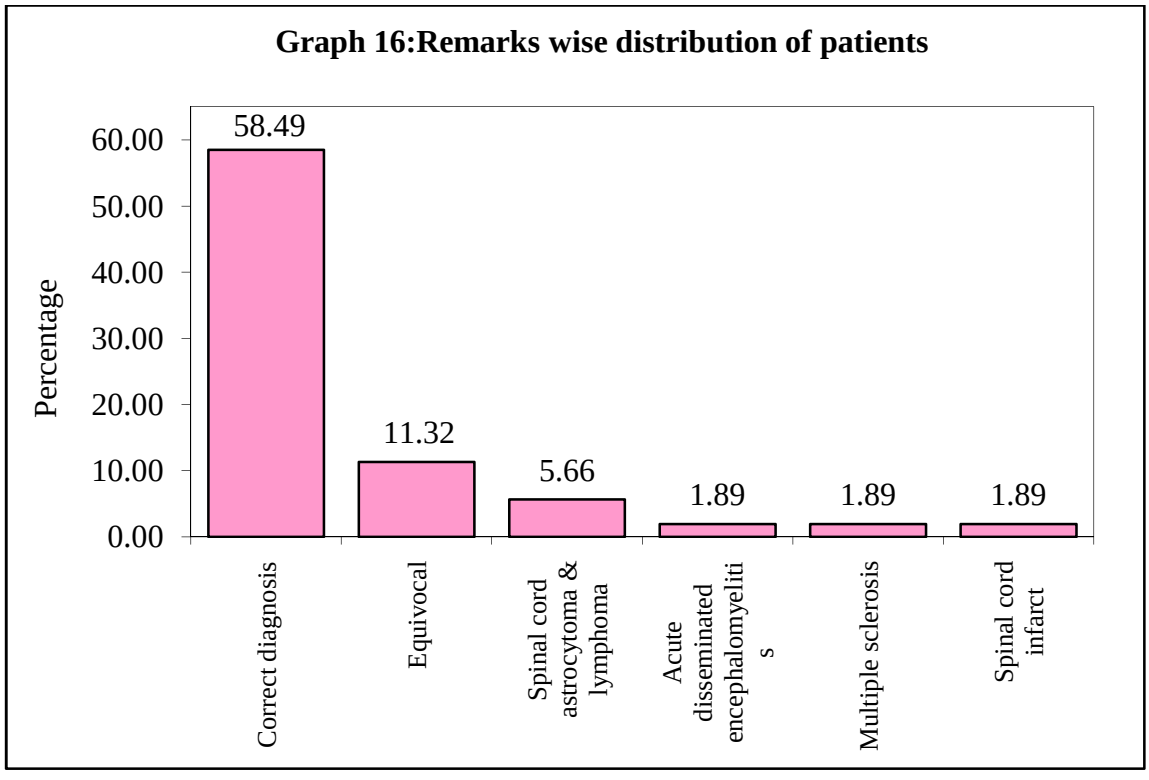
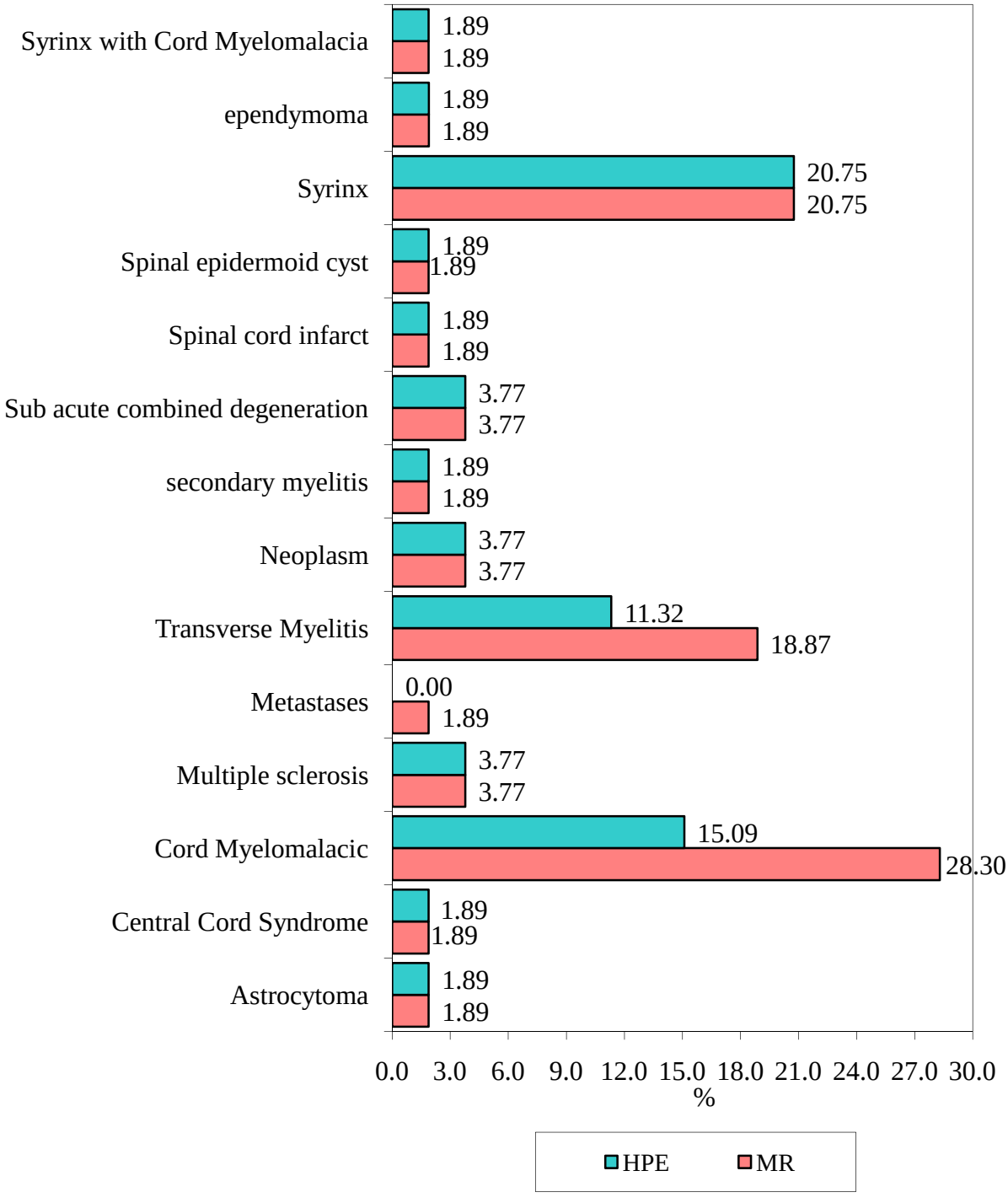


Table 24: Descriptive analysis of all the causes of T2 hyper intensities in spinal cord lesions in study population. (N=53)

MR diagnosis	MR	%	HPE	%
Astrocytoma	1	1.89	1	1.89
Central Cord Syndrome	1	1.89	1	1.89
Cord Myelomalacia	15	28.30	9	15.09
Ependymoma	1	1.89	1	1.89
Multiple sclerosis	2	3.77	2	3.77
Metastases	1	1.89	0	0.00
Transverse Myelitis	10	18.87	6	11.32
Neoplasm	2	3.77	2	3.77
Secondary myelitis	1	1.89	1	1.89
Sub acute combined degeneration	2	3.77	2	3.77
Spinal cord infarct	1	1.89	1	1.89
Spinal epidermoid cyst	1	1.89	1	1.89
Syrinx	11	20.75	11	20.75
Syrinx with Cord Myelomalacia	1	1.89	1	1.89

Among the study population, most common cause is Cord Myelomalacia16 (30.18 %) followed by syrinx 11(20.75%), transverse Myelitis11 (18.87 %), SACD &multiple sclerosis2 (3.77 %) cases , astrocytoma, metastases,ependymoma, spinal cord infarct and secondary myelitis comprised of 1 (1.89 %) cases.(Table 24&graph 17)

Graph 17: Bar graph for summary of all the causes of T2 hyper intensities in spinal cord lesions



DISCUSSION

As compared to Myelography and CT, MRI has a greater sensitivity and specificity in direct visualisation of spinal cord in cases for trauma, neoplasm and degenerative disorder. Its zero risk of radiation makes it the choice of imaging modality for various spinal cord pathologies.

On T2-weighted MR images (T2 WI) common MRI finding in myelopathy is either focal or diffuse cord hyperintensity which can lead to confusion in spinal imaging because of the presence of its numerous causes, which are often difficult in differentiating. The clinical symptoms of myelopathy are often vague in presentation. By just examination of the symptoms its often difficult in pin- pointing the level of pathologic abnormality.

Detailed clinical history, acuity of symptoms (acute versus insidious onset), distribution of the signal abnormalities, including length of cord involvement, specific tract involvement, and the region of the spinal cord that is affected, are very useful in making the diagnosis.

In our study of 53 patients of myelopathy we found various causes of T2 hyper intensities. Among these are compressive (15) , congenital (12), metabolic (2), inflammatory (11) and vascular (1).

Age and Gender:

In the current study, the mean age of the participants was 44.45 ± 18.69 years. It differed across the various study. Current study comprised of 28 (52.83%) cases males and remaining 25 (47.17%) cases females. The gender differed across various

studies. This is in comparison with the study done by Yeliz Pekcevik, et al.[5] where the mean age in the study was 44.5 years.

Clinical symptoms:

In the current study, the most common symptom was cervical pain in 14 (26.42%) cases followed by backache in 12 (22.64%) cases, paraparesis in 5 (9.43%) cases, quadriparesis in 4 (7.55%) cases, hemiparesis of foot in 3 (5.66%) cases and headache & loss of vision in only 1 (1.89%) cases respectively. This was in accordance to the study done by Jane Watts et al [3], in which most common clinical symptoms consisted of motor and sensory impairment. In syrinx pain & thermal sensory impairment was most commonly observed. In ATM rapid onset of bilateral motor & sensory dysfunction was observed. SACD comprised of paraparesis or tetraparesis.

Spine x-ray

In the current study, 24 (45.28%) cases showed abnormal findings on spine X-ray, and 12 (22.64%) cases showed normal findings. 17 (32.08%) patients X rays were not taken.

This was in accordance to the study done by Naohiro Tachibana et.al.[12]Levels of lesion involved

In our study the level of lesions involved in 53 cases were cervical 33 (62.26%), thoracic 25 (45.22 %), lumbar 3 (5.66%) and cervicomedullary junction 2 (3.77 %).this is comparable to the study conducted by kerslake et al. [44]

MRI demonstrated spinal cord abnormalities such as cord compression and abnormal Signal intensities within the spinal cord. Spinal cord compression was observed in 13 cases.

In the current study most frequent duration of symptoms were chronic 28 (52.83%) followed by sub acute 13 (24.52 %) cases.

In our study out of 53 cases most common location of lesions in spinal cord was central 49 (92.45%) followed by posterior 2 (3.77%) and anterior 2 (3.77%) cases. This is comparable to the study conducted by Sarah M .[46]

In our study out of 53 cases 15 (28.31 %) were of cord myelomalacia in which we found various causes of cord compression. Among these Causes of spinal cord compression included disc bulge 5 (9.43%), disc protrusion 2 (3.77%), extra dural soft tissue 1 (1.89%), osteophytic disc complex 2(3.77 %) and wedge compression fracture 1 (1.89%).

T Kameyamaet al.[10]conducted a study on compressive myelopathy which showed T2 hyperintensities. They described that severity of clinical symptoms were proportional to degree of cord compression. In most cases T2 hyper intensity was found more where the level of compression was maximum.

Similarly in our study the frequency of T2 hyper intensity was proportional to clinical severity & degree of spinal cord compression.

MRI depicted not only spinal cord changes in our patients but also relationship of ligamentous disruption in which positive 17 (32.08 %) & negative 36 (67.92 %) and posterior elements involvement was in 27 (50.94) and pre & pair vertebral collection was in 7 (13.21 %).

Advantage of MRI in demonstrating all the changes is shown in studies done by kulkarni et al.[45]

In our study we had 15 cases (28.31 %) of demyelinating conditions out of which acute transverse myelitis 10 (18.87 %), Multiple sclerosis 2 (3.77 %), ADEM 1 (1.89 %) and Sub acute combined degeneration 2 (3.77 %) cases.

Out of 10 cases of acute transverse myelitis (ATM) clinical symptoms included paraparesis in 4, quadriparesis in 3, tetra paresis in 2 and loss of vision was seen in 1 case. MR imaging demonstrates T2 hyper intense spinal cord lesions in almost all the cases. Classic lesions extended over more than two segments with involvement of more than two-thirds of the cross-sectional area of the cord. In our study most common site of involvement included cervical cord 33 (62.26%) followed by thoracic 24 (45.27%), lumbar 3 (5.66%) and cervicomedullary junction 1 (1.89%). Out of 53 cases on contrast studies homogenous enhancement in 3 (5.66 %) cases, peripheral enhancement in 3 (5.66 %) cases, non enhancement in 11 (20.75 %) and in 36 (67.92 %) did not undergo contrast studies.

This has also been shown by studies done by Christine goh et al.[42]

R Bakshi et.al.[47]did comparative study on ATM vs MS in which they concluded that in both the groups cord T2 hyper intensity was present with cord expansion. Most common location was cervical in both of them. However in post contrast studies ATM showed abnormal enhancement unlike of MS. MR brain imaging helped in differentiating both the groups as the former presented with findings than the later.

2 cases of subacute combined degeneration were reported in our study in which clinical manifestations included loss of sensation in both hands & feet. Vitamin B 12 level was found to be low (70 pg/ml) and (143 pg/mL) respectively. Initially the patient was subjected to MR brain imaging with contrast which revealed no diagnostic pathology. On further imaging of the spine T2 hyperintensities were noted involving posterior column of the cord which on axial images, bilateral paired areas of T2 hyper intensity are seen as an "inverted V" shape in the dorsal column tracts extending from cervico-medullay junction till C2vertebrae.

This is sustained by a study conducted in Government Medical College, Kerala, India.[16]

V K Katsaros et al.[49]explained that deficiency in vitamin B 12 can result in SACD. They have demonstrated the importance of MRI in detecting lesions in the spinal cord and following up while administrating vit B 12. In their study they included screening of not only the spine but also the brainstem and cerebellum in which the lesions differed.

In our study we had 2 cases of multiple sclerosis in which one of them presented with paresthesia and urinary incontinence. On spine imaging there is short T2 hyperintensities for an approximate length of 1.1 cms involving dorsal aspect of the cervical spinal cord with mild diffusion restriction on DWI sequence. No cord atrophy / edemawas noted.

MRI brain imaging with IV gadolinium was done in which T2 & flair hyper intense lesions noted in left periventricular region with diffusion restriction. The lesions showed open ring incomplete enhancement suggestive of tumefactive demyelinating plaques. On follow up the patient was treated with methylprednisolone

and good response was noted while the other one had expired. MR findings of this demyelinating plaques were reported by several authors.

Christopher R. Tench, et.al.[48] explained. In detail the role of MRI in diagnosing MS. They told that imaging of spine is useful in conditions were brain imaging is normal incases of early MS. They clearly explained the importance of brain imaging in differentiating between NMO, ADEM, MS & TM.

Out of 15 cases of demyelinating conditions we had only 1 case of ADEM in which the patient presented with dysarthria & loss of consciousness. He had past 3 weeks history of upper respiratory tract infection. The patient was referred for MR screening of brain. On imaging T2 & FLAIR hyper intensities noted involving pons, bilateral basal ganglia and fronto-parietal regions. The patient further underwent spine screening in which long segment T2 hyperintensities noted involving the entire circumference of the spinal cord (periaqueductal grey matter) with cord expansion for an approximate length of 11.8 cms. On further more investigations anti-myelin oligodendrocyte glycoprotein (MOG) antibodies came positive hence, supporting the diagnosis. On follow up imaging after 2 months T2 hyper intensity of the spinal cord had resolved.

In our study out of 53 cases we had 11 (22.64 %) of congenital abnormalities in which syrinx 7 (13.21 %) and syringohdromyelia 4 (7.54 %). The peculiar findings of each syrinx and presence of cerebellar tonsils were analyzed on T2- weighted images. On imaging, T2 hyper intense lesion, CSF flow-void sign (CFVS) in the syrinx cavity. Most common level of lesion was cervical cord (8 cases).

This is comparable to the study conducted by J L Sherman et al.[23] on MR appearance of syringomyelia.

Our study consisted of 4 (7.55%) cases of primary intramedullary neoplasms, out of which 2 cases were astrocytoma, 1 lymphoma and 1 ependymoma. Of the 3 cases of neoplasm MR diagnosed 2 cases as astrocytoma. In 1 case MR could not differentiate between intramedullary metastases and lymphoma. Clinical manifestations in both the cases of astrocytoma in our study showed backache with both limb weakness. On MR imaging one of the case showed T1 isointense & T2 hyper intense lesion with expansion of cord with well defined margins. On IV Gd-DTPA heterogenous enhancement noted.

In the other case with MR diagnosis as neoplasm patient presented with paresthesia and urinary bladder incontinence. He was subjected to entire spine screening in which T2 hyper intensity noted involving cervico-medullary junction till the C3 vertebrae on further imaging of the brain with contrast heterogenously enhancing lesion noted involving the medulla oblongata extending posteriorly upto the cerebellum.On follow up the patient was operated which on histopathology has turned out to be ependymoma. After 8 months of repeat imaging recurrence of the lesion noted.

A study was conducted by Bo Sun et.al. [26] in which imaging features of spinal ependymoma were depicted. They summarized that most common location of these tumors was upper cord & most of them were cellular type. Papillary type occurred most commonly in conus medullaris. They concluded that imaging features such as homogenous enhancement and well defined margins helped them to differentiate them from other tumours.

We had 1 case of lymphoma which MRI has diagnosed as intramedullary metastases / lymphoma as differential diagnosis. It. showed isointensity on T1 WI

&hyper intensity on T2 WI which on IV Gd-DTPA showed intense homogenous enhancement extending for about 3 Vertebral levels in the lumbar region.

Out of 53 cases Our study included 1 case (1.89 %) of spinal cord infarct . Patient presented with acute & progressive sensorimotor deficit associated with backpain. On spinal imaging abnormal T2 cord hyper intensity noted involving grey matter sparing the white matter in the cervical, dorsal & lumbar region of spinal cord. Cord expansion was also noted at that level. On DWI sequence diffusion restriction noted suggesting spinal cord infarction. MR brain screening was also subjected to the patient which appeared normal. These signal changes are in consistent with studies done previously by Jane Watts et al.[4]

CONCLUSION

1. This is aobservational study aimed to evaluate the role of MRI in myelopathy in correlation with clinical context conducted in the department of radio diagnosis of a tertiary care teaching hospital. 53 patients were chosen by purposive sampling.

2. The diagnostic utility of regular imaging modalities like X-ray is limited in this population. But advanced imaging modalities like MRI may be of high utility. In our study, myelopathy was spread across various age groups and gender. The most common clinical symptoms in patients were cervical pain followed by backache, paraparesis, quadriparesis and hemiparesis of foot.

3. Patients with suspected myelopathy were evaluated with MRI to charaterize the imaging features of the lesion.

4. Most common causes of T2 hyper intensities in our study was demyelinating conditions in which most common was transverse myelitis followed by cord multiple sclerosis.

5. Only one case ofacute disseminated encephalomyelitis was observed in our case study.

6. Apart from demyelinating conditions most common cause was cord myelomalacia.

7. 2 cases of astrocytoma & 1 case of ependymoma were reported in our study.

LIMITATIONS:

1. The major limitation was the limited sample size, due to which the detailed evaluation and further characterization of some of the cases was not possible.

2. The role of confounding in key features like the age of the individual, gender distribution could not be evaluated again due to limited sample size.

3. Contrast imaging was not a criteria taken into consideration which would increase the diagnostic precision.

RECOMMENDATIONS:

1. It is non invasive method with no risk of exposure of the patient to contrast agents or radiation exposure to diagnose spinal cord abnormalities.

2. There is a need to conduct more studies with a larger sample size to evaluate the diagnostic utility of the imaging modality in different subgroups of the population.

3. The findings of the study must be generalized with caution, considering the differences in the demographic features of the population, the quality of the equipment used and the experience & skill of the radiologists.

SUMMARY

A prospective observational study was conducted in the department of Radio-diagnosis of a tertiary care teaching hospital to assess the diagnostic ability of MR imaging in evaluation of spinal cord abnormalities.

MRI turned out as modality of choice in evaluation of demyelinating conditions, cord infarctions, & tumours. Eventually, final diagnosis is based on tissue pathology.

In this study I could draw an approach to evaluate the cause of T2 hyper intensities in spinal cord lesions.

The study has highlighted the features of myelopathy on MRI evaluation in a tertiary care hospital. The study findings can be useful for clinical practitioners dealing with possible cases of spinal myelopathy by providing organized approach in identification of cord lesions and in effective management.

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ANNEXURE I - INFORMED CONSENT

TITLE OF THE STUDY: EVALUATION OF T2 HYPERINTESITIES IN SPINAL CORD LESIONS USING MAGNETIC RESONANCE PROTOCOL. - A ONE YEAR OBSERVATIONAL STUDY TERTIARY CARE HOSPITAL IN NORTH KARNATAKA"

PRINCIPAL INVESTIGATOR: REG. NO. BS0118005

INTRODUCTION AND PURPOSE:

A wide range of spinal cord lesions show hyperintense spinal cord signal on T2 weighted image. The purpose of this article is to review them and provide a systematic approach to help narrow down the list of differential diagnosis.

PROCEDURE:

I request you to kindly participate in the study titled **EVALUATION OF T2 HYPERINTESITIES IN SPINAL CORD LESIONS USING MAGNETIC RESONANCE PROTOCOL - A ONE YEAR OBSERVATIONAL STUDY TERTIARY CARE HOSPITAL IN NORTH KARNATAKA"** We request you to participate in this study as you are eligible to be included. During the study you will be asked questions regarding your present and past medical history and you will be required to answer to the best of your knowledge.

If you agree to participate in the study please furnish the details pertaining to the study.

The scanning equipment to be used is 1.5 tesla MRI machine manufactured by Siemens and/or 3 Tesla machine manufactured by GE with the help of a dedicated body coil with the patient in a supine position.

BENEFITS:

- Minimally invasive
- No radiation exposure, therefore even the pregnant women can be included.
- Results will help the radiologists in better and early diagnosis of spinal cord lesions of the patients and hence allow for a better treatment plan.

RISKS:

- No side effects related to research study. Side effects related to anesthesia and sedation (if required) are there which will be taken care of by referring doctor.

ALTERNATIVES:

- If patient is not willing to take part in the study, his / her treatment or any other further investigations the patient wants to undergo, in future, in KLE will not be affected by his / her decision.

VOLUNTARY PARTICIPATION/WITHDRAWAL:

- Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. My decision will not change the present or future health care or other services that I receive. The study doctor or the sponsor may stop my participation in this study. I will tell of any important new findings that may change my willingness to continue to take part. If I choose not to take part in the study I will receive the standard treatment for patients with my condition.

COSTS:

No additional cost other than MRI which is advised by referring consultant for diagnostic purpose.

COMPENSATION:

In the event that I become injured as a result of taking part in this study, treatment will be offered to me, No reimbursement, compensation or free medical care is given.

CONFIDENTIALITY:

All information collected about me during the course of the study will be kept confidential to the extent permitted by the law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

QUESTION:

If any enquiries in the future or in case of research related injury illness, you may contact following person.

- **REG. NO. BS0118005**, Department of radio-diagnosis, KLE'S Dr. Prabhakar Kore hospital and MRC, Belagavi or by phone: _____

- **Dr_____**, professor, department of radio-diagnosis, KLE'S Dr. Prabhakar Kore hospital and MRC, Belagavi or by phone: _____, Ext. _____.

- If you have any queries about the rights as a study subject, you may call **Dr. Dr. ROOPA M BELLAD**, Professor, Department of pediatrics, Chairman of JNMC Institutional Ethical Committee of Human Subjects Research, phone no: 0831-2473777, Ext. 1529 at JNMC Belagavi.

CONSENT TO PARTICIPATE IN RESEARCH STUDY:

I voluntarily agree to take part in this study by signing on the line below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicated that I have read this entire consent form or it has been read to me, and has been explained to me in my vernacular language and had all my questions answered. I will be given a copy of this consent form.

Signature /Left Thumb print of the Participant or legally authorized representative.

Participant's Name :……

Signature/ Left Thumb impression. : ……..

Name of the legally authorized representative:……

Signature/ Left Thumb impression. :……

Witness's Name : ……..

Signature/ Left Thumb impress :……...

Investigators name and Signature : ……

Date and Place : ……

ANNEXURE II –ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : domc@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ \7 **Date: 24/11/2018**

To,

REG.NO. BS0118005

PG student in Radio-Diagnosis,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"EVALUATION OF T2 HYPERINTENSITIES IN SPINAL CORD LESIONS USING MAGNETIC RESONANCE PROTOCOL IN CORRELATION WITH CLINICAL CONTEXT – A ONE YEAR OBSERVATIONAL STUDY AT TERTIARY CARE HOSPITAL IN NORTH KARNATAKA",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE III -PROFORMA

DATA COLLECTION INSTRUMENT

EVALUATION OF T2 HYPERINTESITIES IN SPINAL CORD LESIONS USING MAGNETIC RESONANCE PROTOCOL. - A ONE YEAR OBSERVATIONAL STUDY TERTIARY CARE HOSPITAL IN NORTH KARNATAKA"

PATIENT DATA

SL.NO.		DATE	
PATIENT NAME		MRI SCAN NO.	
AGE		SEX	

HISTORY

SYMPTOMS	
FEVER WEIGHT LOSS	YES/NO YES/NO
BACK PAIN RADIATING PAIN TO BOTH LIMBS WEAKNESS NUMBNESS	YES/NO YES/NO YES/NO
DURATION OF SYMPTOMS	

H/O TRAUMA	YES/NO

INVESTIGATIONS

CXR	
X RAY SPINE	
CT SPINE	
MRI BRAIN	

MRI FINDINGS

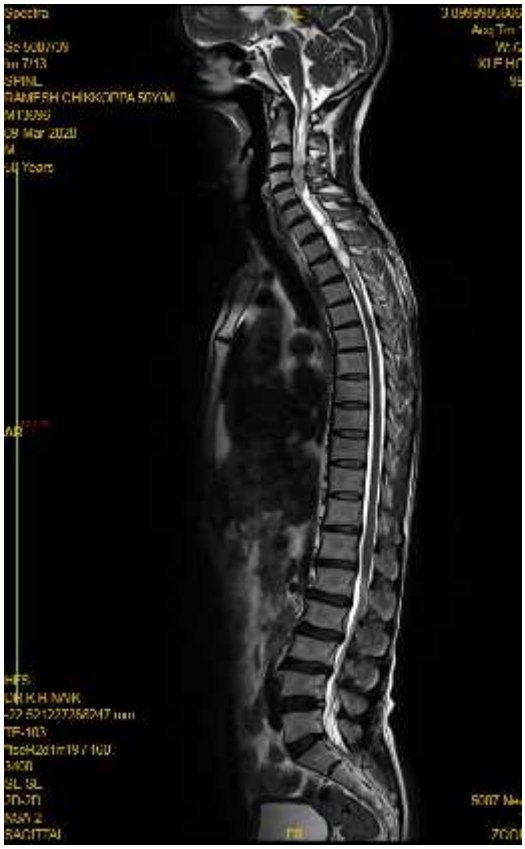
LEVEL OF LESION	
LOCATION OF LESION IN SPINAL CORD	
SPINAL CORD COMPRESSION	
PREVERTEBRAL COLLECTION	
PARAVERTEBRAL COLLECTION	
CORD CHANGES	
LIGAMENTOUS DISRUPTION	
OTHER SIGNIFICANT FINDINGS, IF ANY	

T1W	
T2W	
STIR	
T1W + CONTRAST	

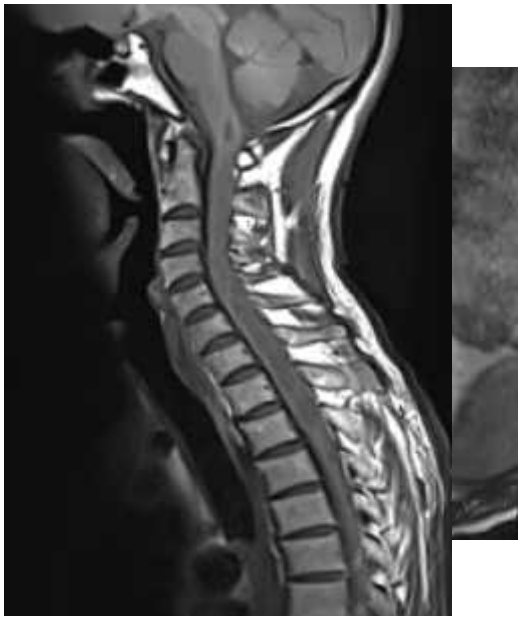
ANNEXURE IV –FIGURES

Figure 8 :Photographs and Description of case ofependymoma:

Case 1: 56 years old male case of ependymoma underwent MRI entire spine which showed well defined intramedullary T2 hyperintense & T1 isointense lesion, approximately measuring 2.3 x 0.9 cms in the cervical spinal cord, extending from lower border of C3 vertebral body to lower border of C5 vertebral body, resulting in mild expansion of the cord at that level. On DWI sequence areas of diffusion restriction noted.On contrast study, the lesion shows heterogeneous enhancement.There is cord expansion with marked cystic dilatation of central canal extending from cervical to dorsal vertebrae (C1 to D3 vertebral level) for an approximate length of 13.0 cms.

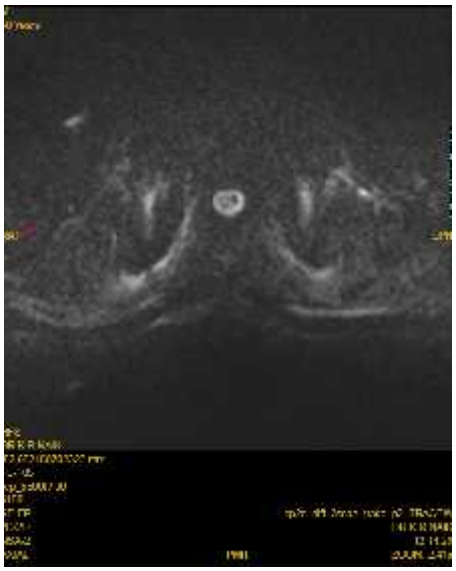


1a: T2W saggital



1b: T2W AXIAL

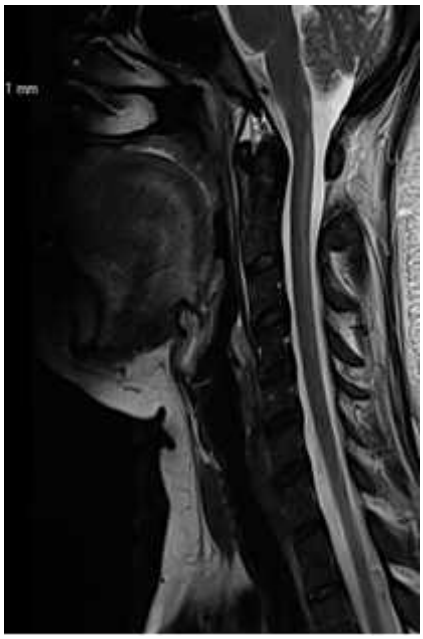
1c: T1W SAGITTAL



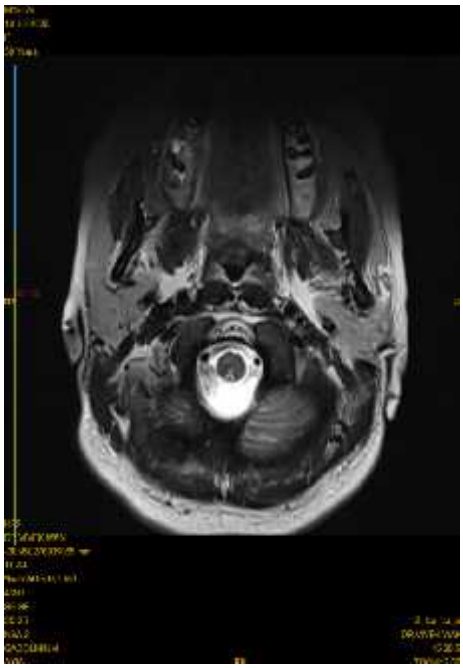
1d: DWI

Figure 9:Photographs and Description of case of subacute combined degeneration:

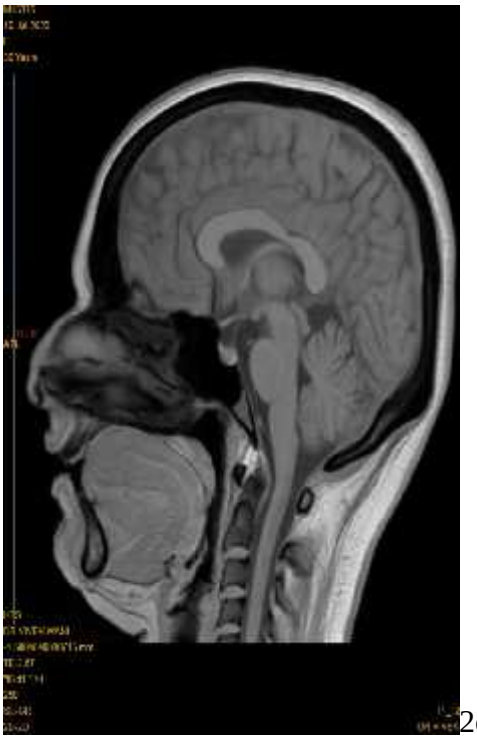
Case 2 : 30 year old female case of subacute combined degeneration underwent MRI brain and spine which revealed inverted V shaped T2 hyperintensity in the dorsal column tracts of cervical spinal cord extending from the level of cervicomedullary junction to C6 veretebra. No significant diagnostic pathology was identified on brain imaging.



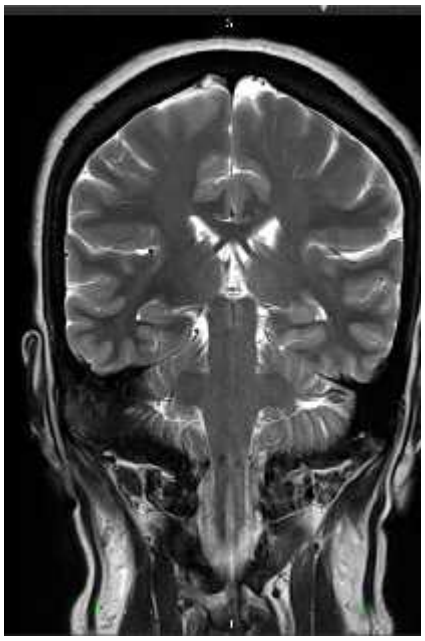
2a: T2W SAGGITAL



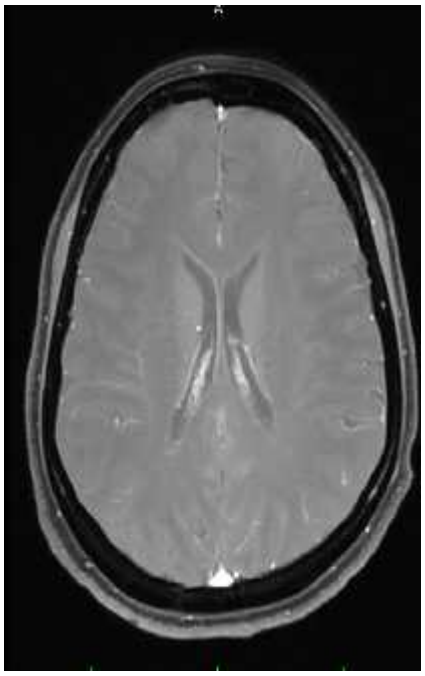
2b: T2W AXIAL



2c : T1 W SAGGITAL



2d: T2 W coronal



2e: T1 W + C AXIAL brain

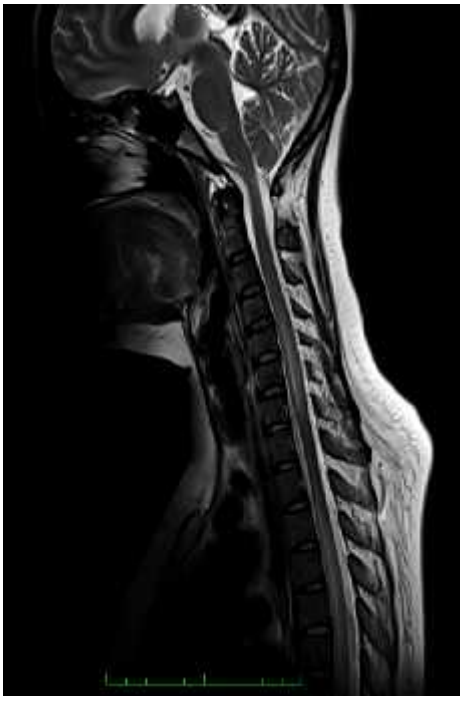
Figure 10:Photographs and Description of case of spinal cord infarction:

CASE3:

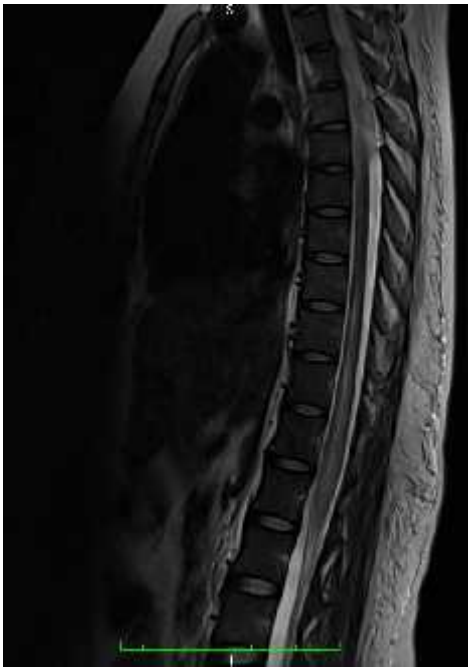
24 year old female case of spinal cord infarction underwent whole spine imaging which revealed abnormal T2 cord hyperintensity involving grey matter sparing the white matter in the cervical spinal cord extending from the level of C5 to C7 vertebrae for an approximate length of 4.0 cms. Cord expansion noted at that level. On DWI sequence diffusion restriction noted.

Similar long segment intramedullary T2 cord hyperintensity noted in the thoracic spinal cord extending from the level of D2 to D6 vertebrae for an approximate length of 8.0 cms.

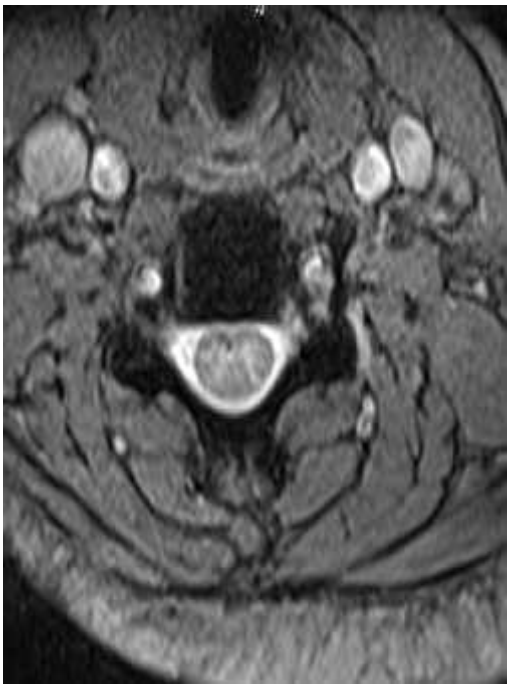
Long segment intramedullary T2 cord hyperintensity also noted in the dorso-lumbar spinal cord extending from the level of D9 to L1 vertebrae for an approximate length of 10.5 cms.On contrast study, no abnormal enhancement noted in the spinal cord.



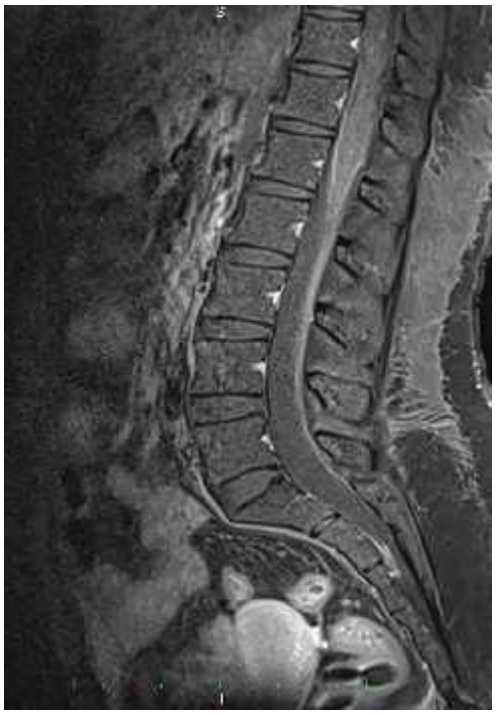
3a: T2WSAGITTAL



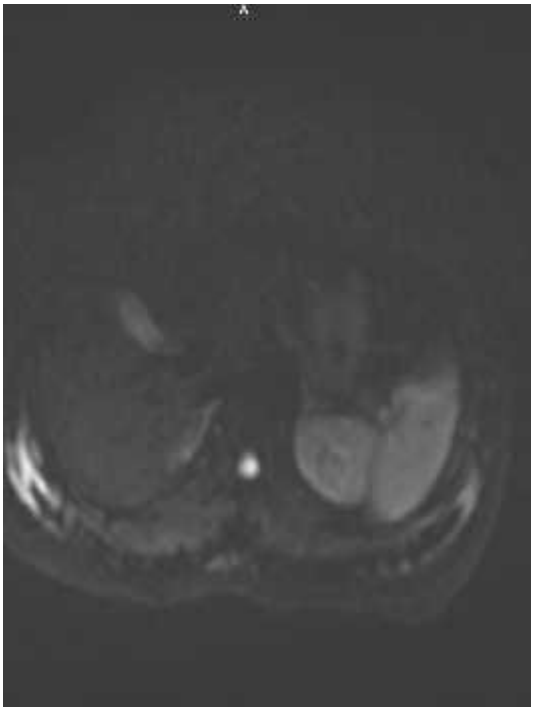
3b: T2W SAGITTAL



3c : T2W AXIAL

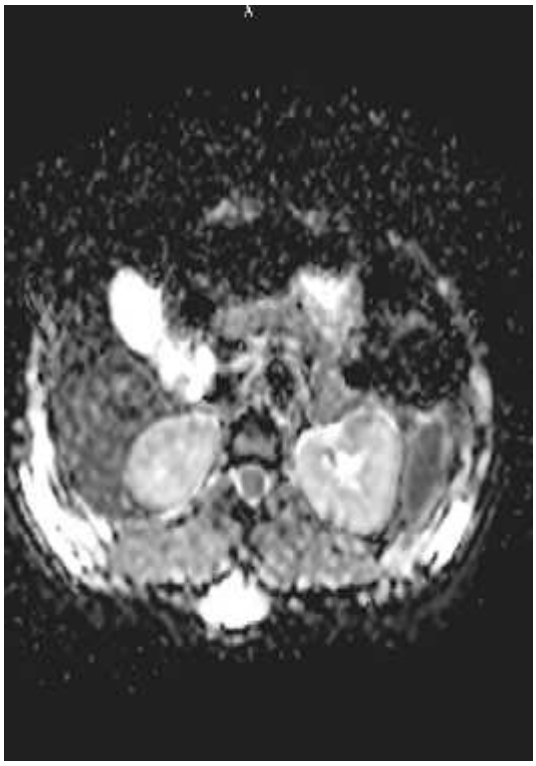


3d : T1W +C SAGITTAL



3f: DWI AXIAL

3e : T1W +C SAGITTAL



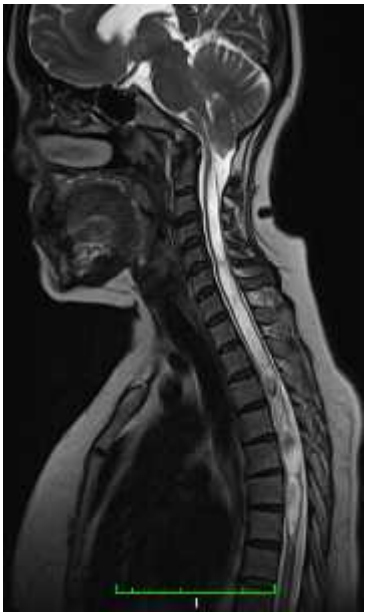
3g : ADC

Figure 11 :Photographs and Description of case of syringohydromyelia:

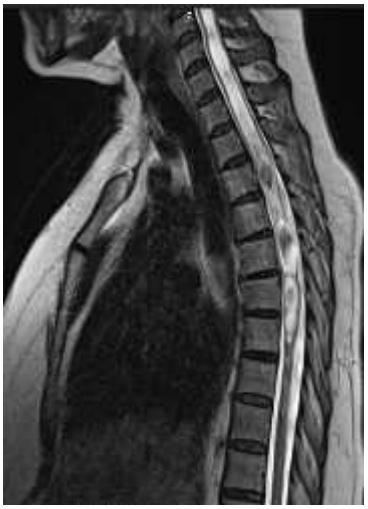
CASE4:-

A 54 year old female case of syringohydromyelia with cervical pain underwent MR spine screening which showed well defined expansile T1 hypointense, T2 predominantly hyperintense intramedullary lesion extending from medulla onblanganta to the level of D9 veretbra, approximately measuring 1.1 x 1.2 x 25.0 cms.On DWI no obvious restriction noted.

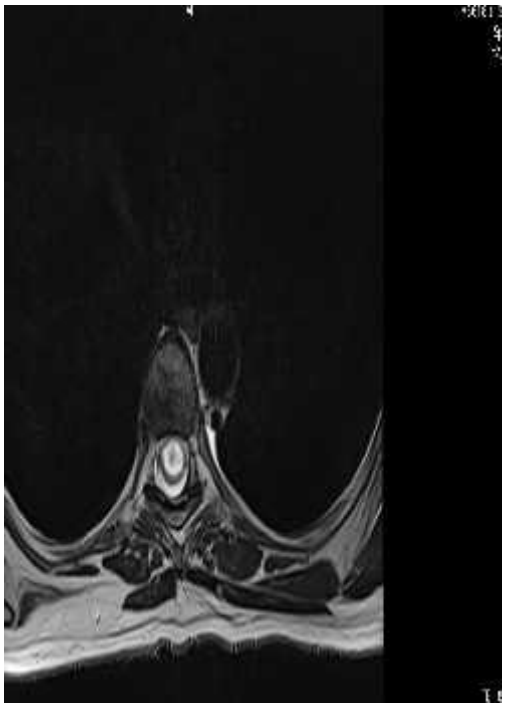
Few well defined hyperintense areas within the lesion are noted due to CSF flow.



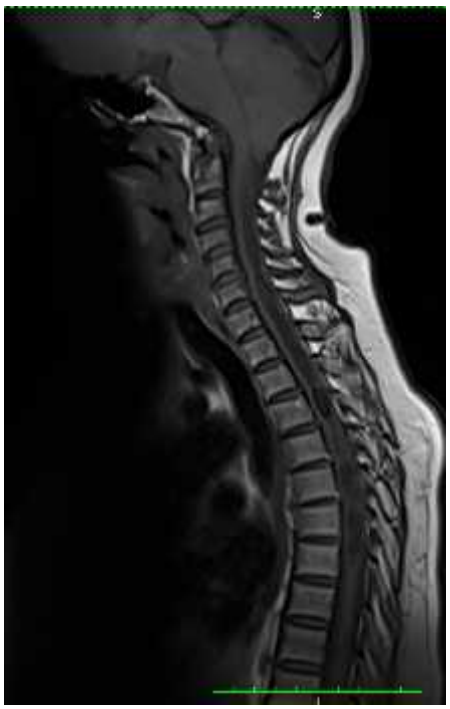
4a : T2W SAGITTAL



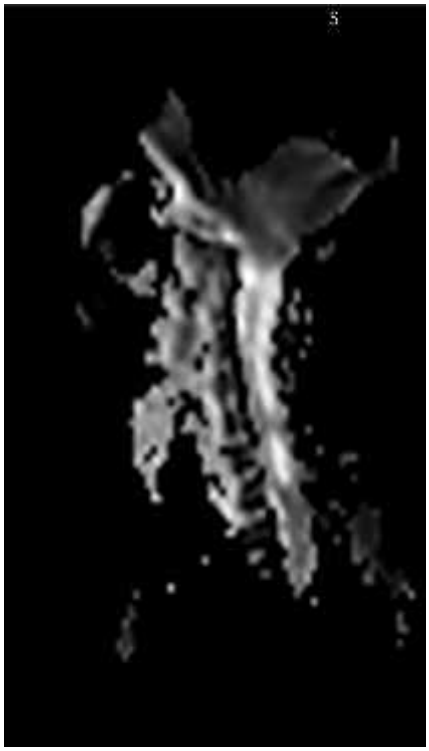
4b : T2W SAGITTAL



4c : T2W AXIAL



4d :T1 W SAGGITAL

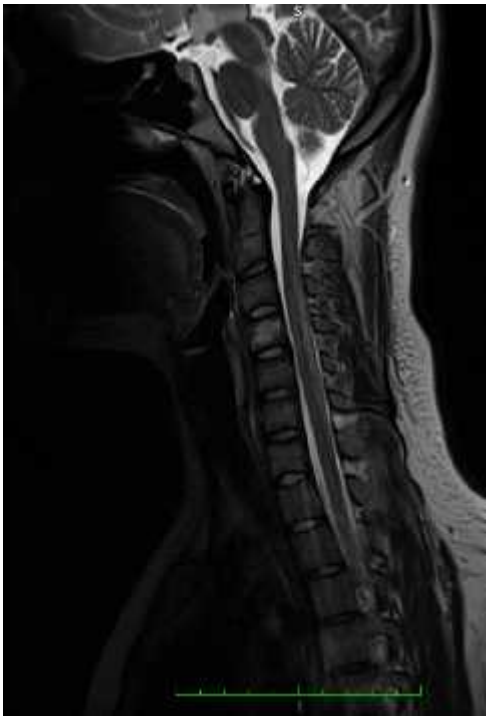


4e : ADC

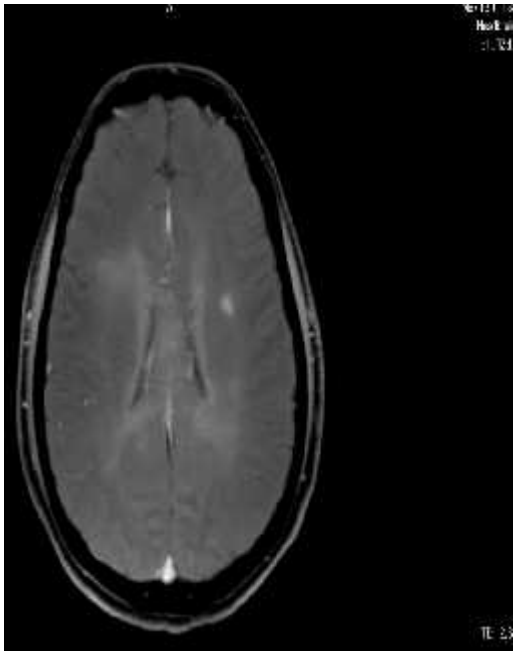
Figure 12 :Photographs and Description of case of multiple sclerosis:

Case 5:-

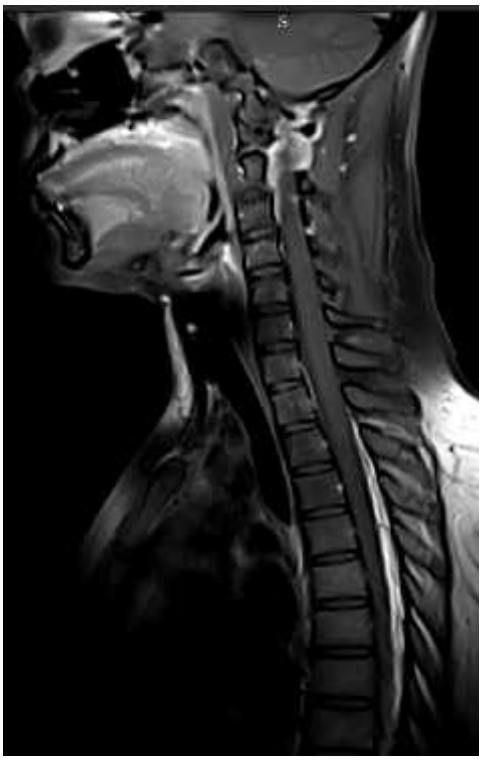
A 22 years old female case of multiple sclerosis underwent MRI brain and spine contrast which revealed open ring incomplete enhancement, T2 & FLAIR hyper intense lesions in bilateral periventricular regions, peritrigonal and right frontal regions which shows restriction on diffusion weighted sequences suggesting tumefactive demyelinating plaques. On spine imaging few T2 hyperintense lesions noted in the cervical spinal cord extending from C3 to C4 vertebral levels.
On contrast study the lesions show mild nodular enhancement suggestive of demyelinating disorder due to multiple sclerosis-active demyelination



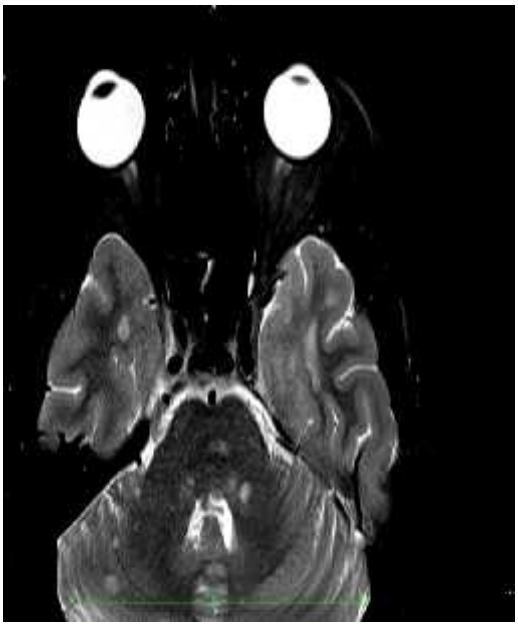
5a : T2W SAGGITAL



5b : T1W + C AXIAL BRAIN



5c :T1W + C SAGGITAL



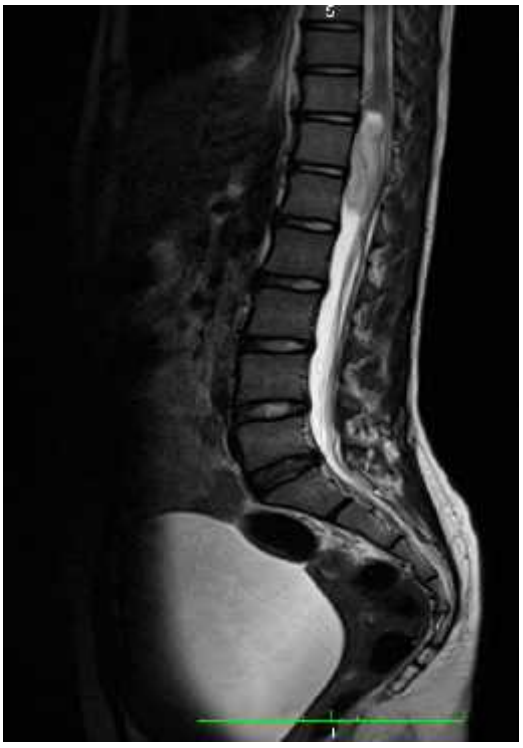
5d : T2W AXIAL BRAIN

Figure 13:Photographs and Description of case of astrocytoma:

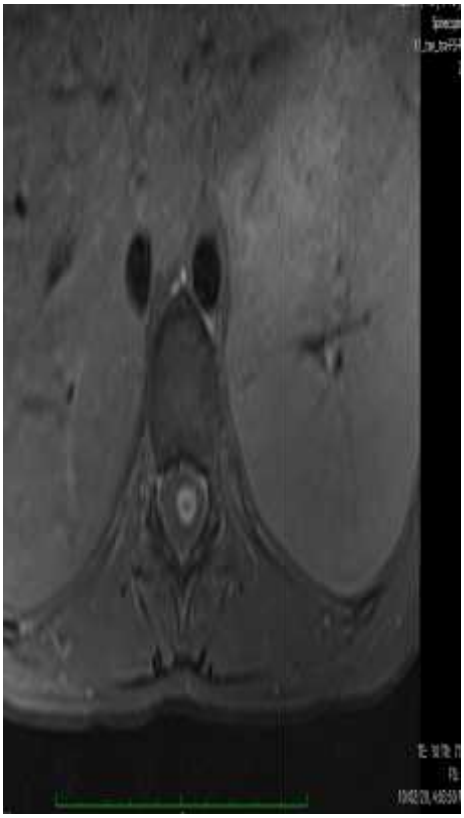
Case 6:-

15 year old female case of astrocytoma underwent MRI spine with contrast which reveled heterogeneously enhancing T1 hypointense and T2 hyperintense mass lesion in the region of conus medullaris at the level of D11 & D12 vertebrae which shows areas of diffusion on DWI sequence, approximately measuring 1.1(AP) x 1.9(ML) x 2.5(CC) cms causing cord expansion and mild adjacent cord edema likely to be neoplastic etiology.

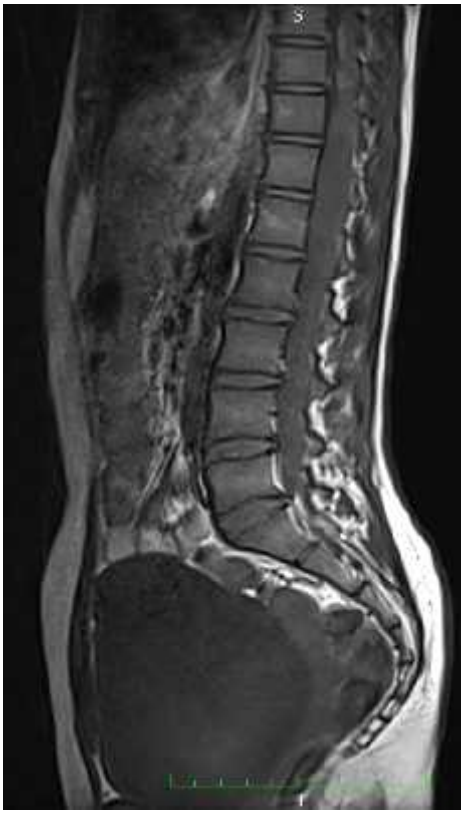
Anotherwell defined peripherally enhancing T1 hypointense and T2 hyperintense lesion in the conus medullaris (6 mm proximal to the above mentioned lesion), at the level of D10-D11 intervertebral disc approximately measuring 5.9 (AP) x 7.8(ML) x 8.3(CC) mm causing expansion of the cord likely to be satellite lesion.



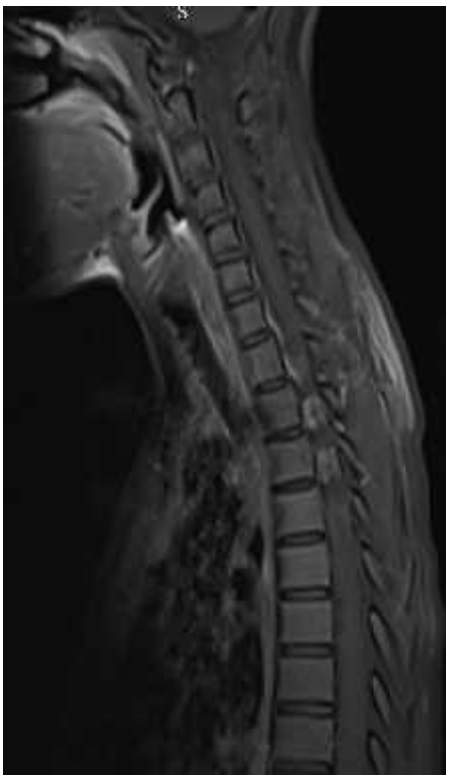
6a : T2 W SAGGITAL



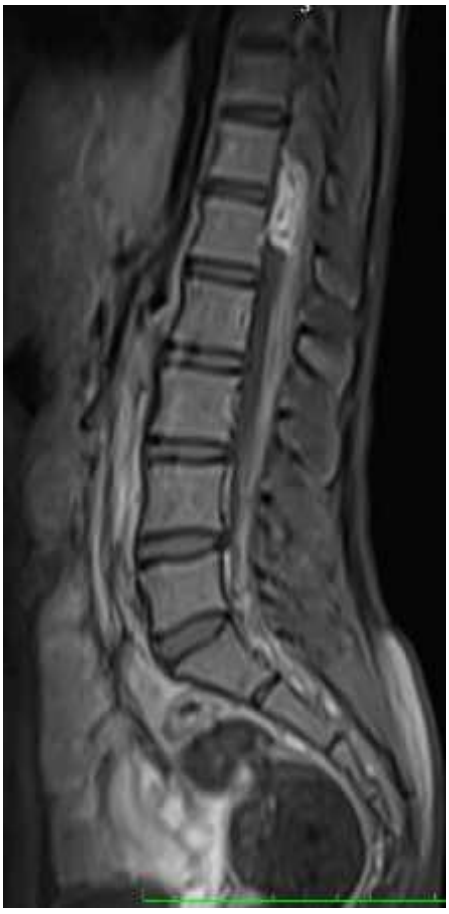
6b : T2 W AXIAL



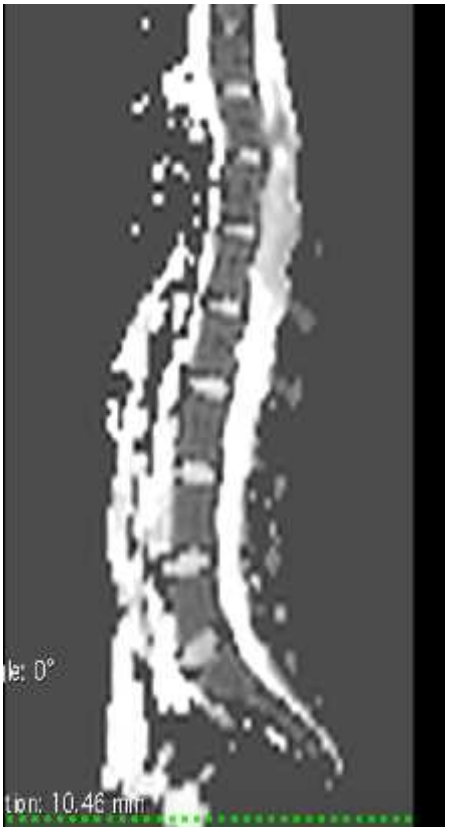
6c:T1 SAGGITAL



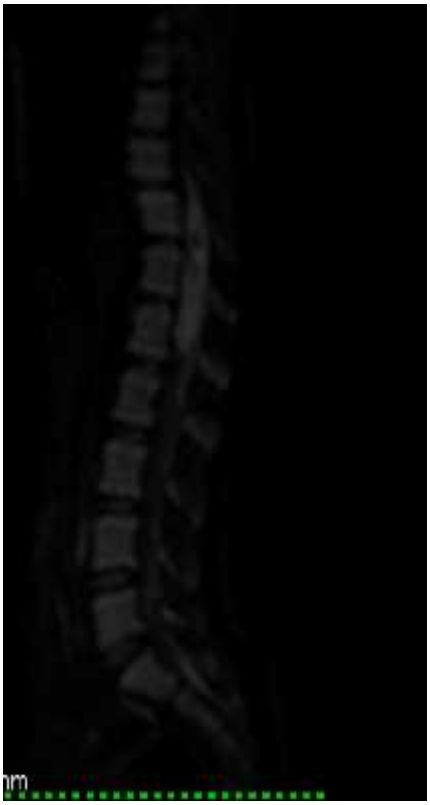
6d : T1 W + C SAGGITAL



6e : T1 W + C SAGGITAL



6f : ADC



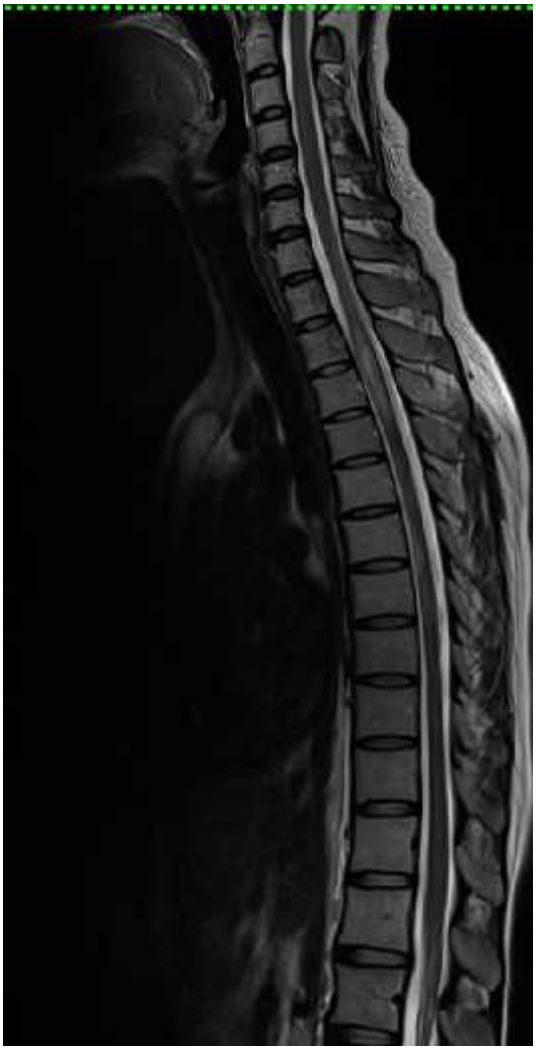
6g : DWI

Figure 14:Photographs and Description of case of idiopathic myelitis:

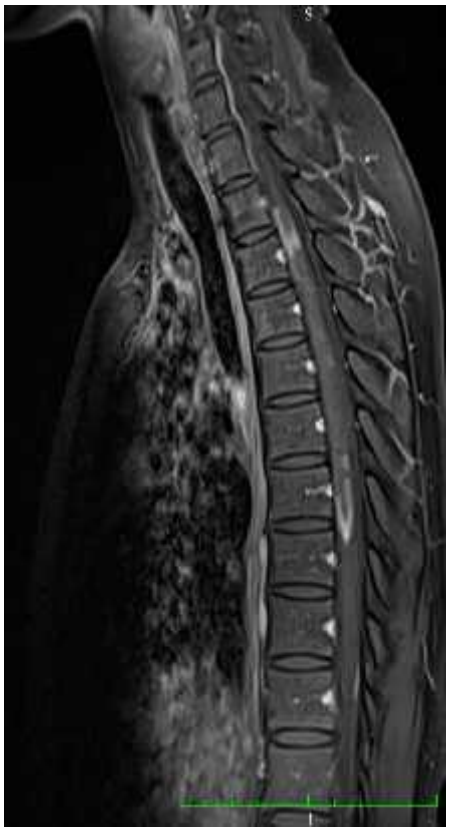
Case 7:-

27 year old female case ofidiopathic myelitis underwent MRI entire spine with contrast which revealed Longitudinally extending long segment T2 hyperintensities involving the dorsal spinal cord extending from upper margin of D2 to upper margin of D8 vertebra, for an approximate length of 11.5 cms. On contrast study, the lesion shows peripheral patchy enhancement.

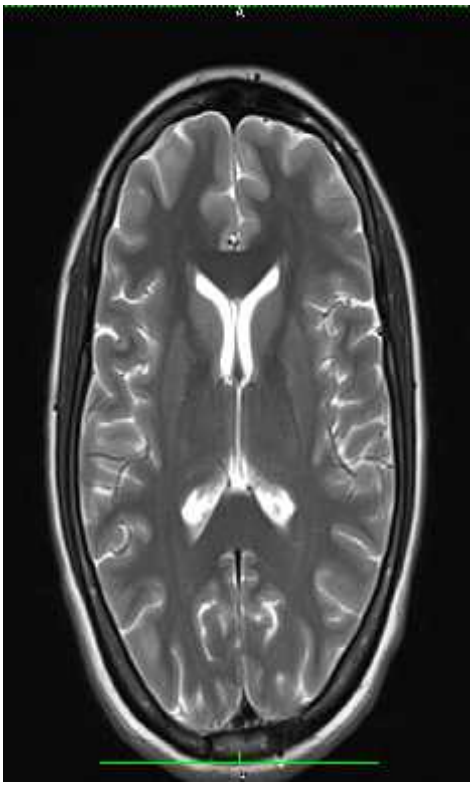
On brain screening no abnormal lesion noted.



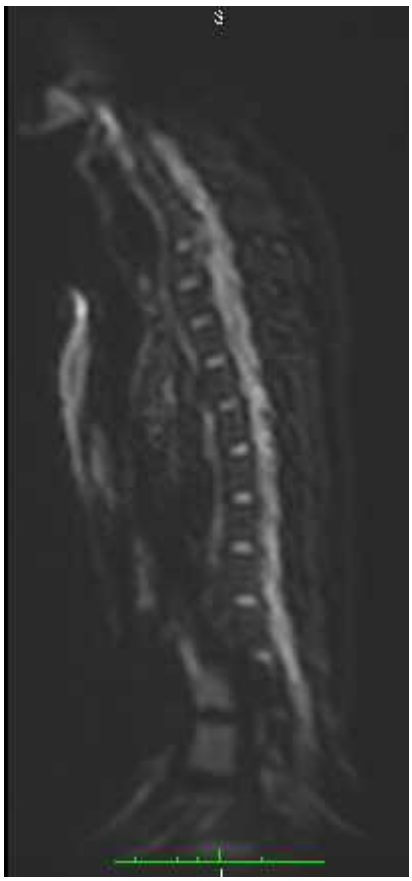
7a : T2 W SAGGITAL



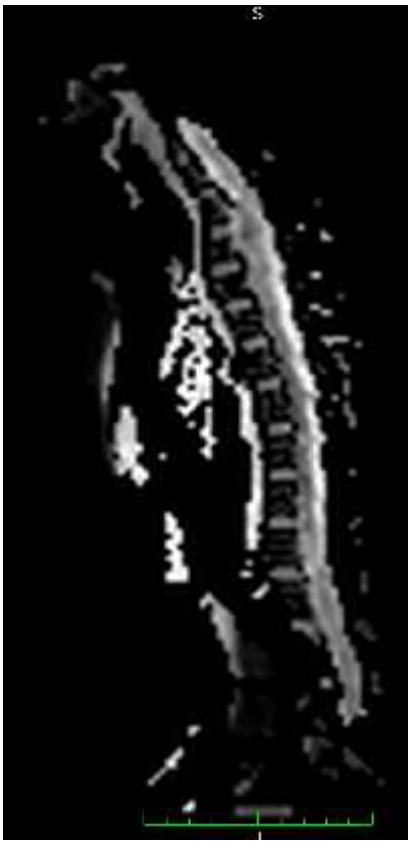
7b : T1 W + C SAGGITAL



7c : T2 W AXIAL BRAIN



7d : DWI



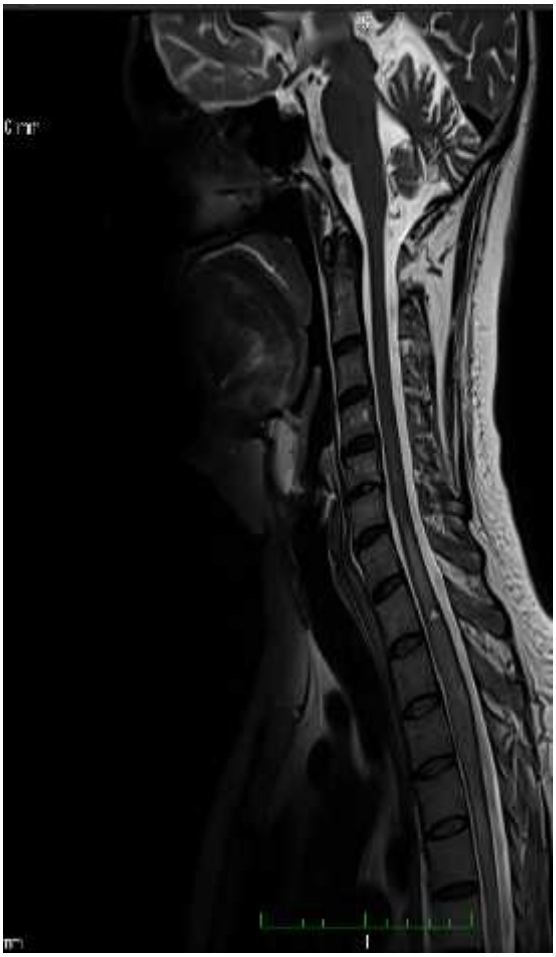
7e : ADC

Figure 15 : Photographs and Description of case of astrocytoma:

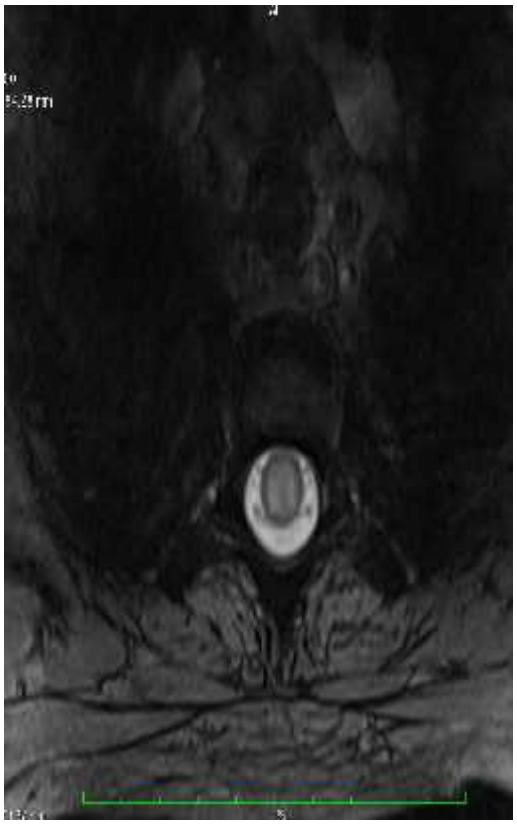
Case 8-

A 56 year old male case of astrocytoma underwent MRI spine screening with contrast which showed homogenously enhancing expansile intramedullary T2 hyperintense & T1 isointense lesion, approximately measuring 6.0 x 0.9 cms extending from the level of D1 to D4 vertebrae. The lesion shows diffusion restriction on DWI sequence.

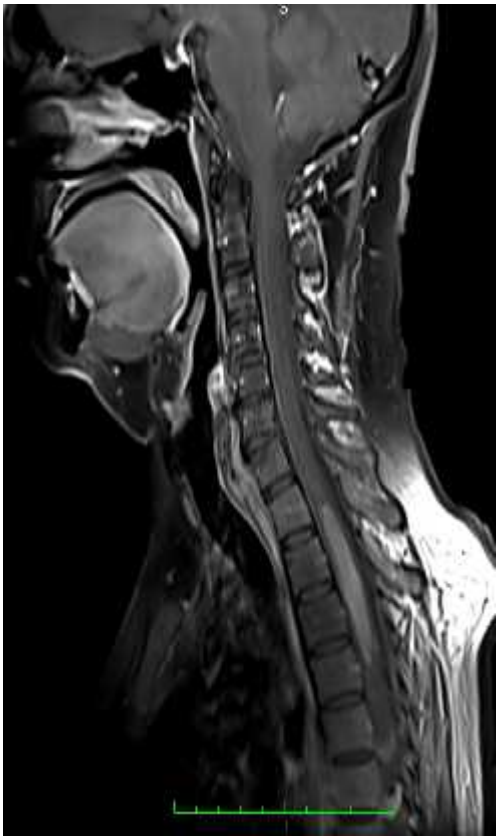
T2 hyperintensities noted in the cord extending from the lesion superiorly till C7 vertebra and inferiorly till the level of D7 vertebra likely suggestive of edema



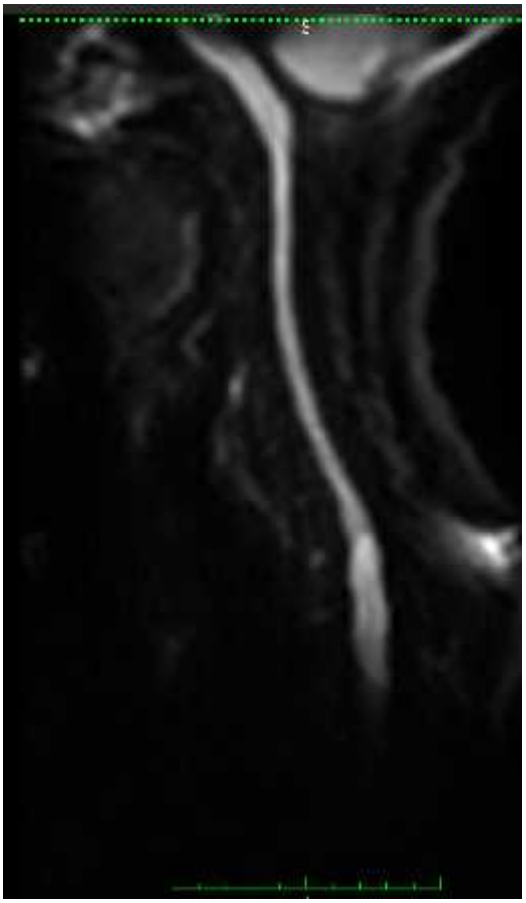
8a : T2 W SAGGITAL



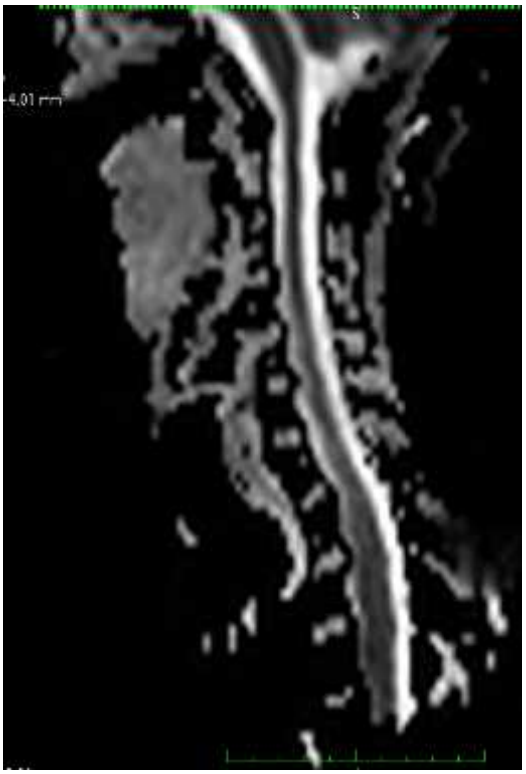
8b: T2 W AXIAL



8c : T1 W + C SAGGITAL



8d : DWI

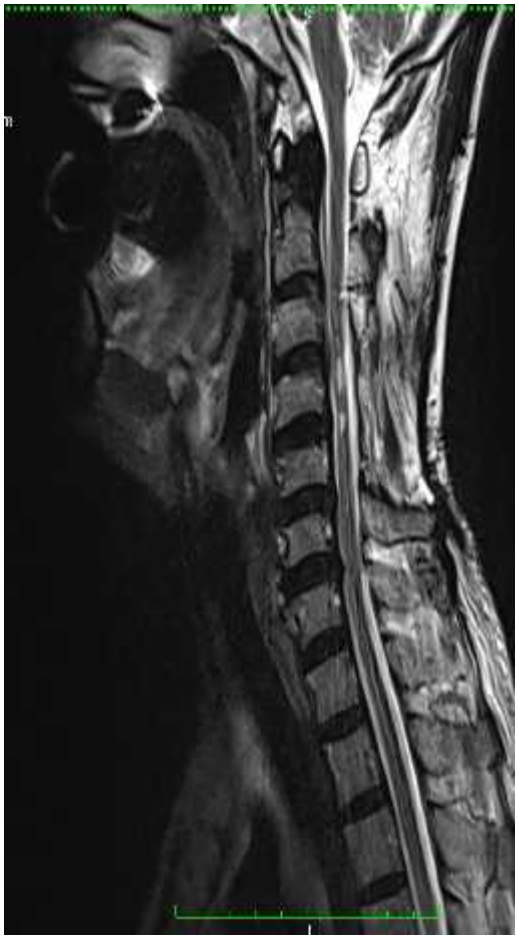


8e : ADC

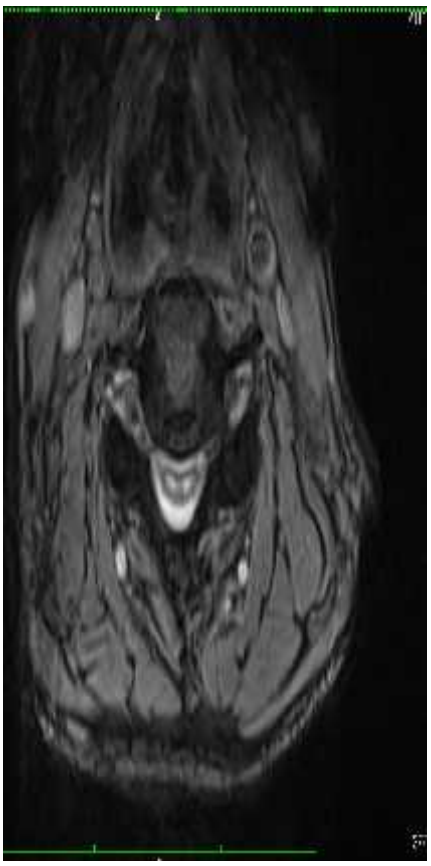
Figure 16 : Photographs and Description of case of cord myelomalacia:

Case 9:-

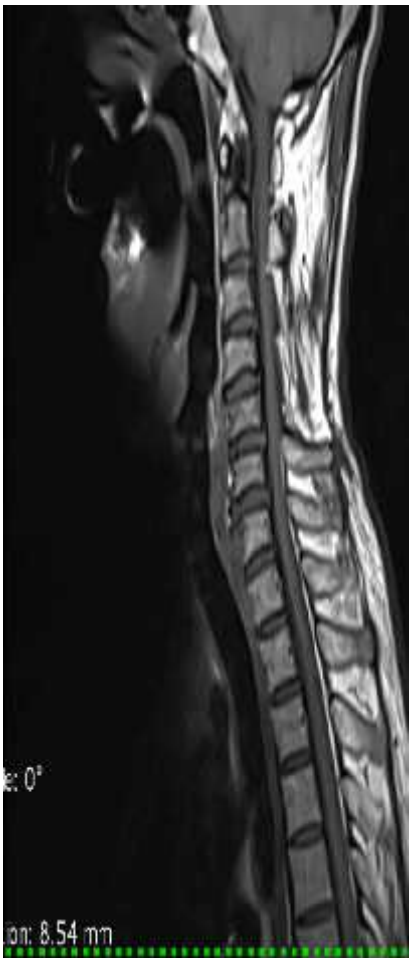
58 years old man case of cordmyelomalacia underwent MRI spine which revealed linear T2 hyperintensities in the cervical spinal cord extending from the C3 to C5 vertebrae with mild decrease in cord diameter for an approximate length of 2.7 cms suggestive of cord atrophy withmyelomalacia.



9 a : T2 W SAGGITAL



9b : T2 W AXIAL



9c : T1 W SAGGITAL

9d : DWI

9e : ADC

ANNEXURE IV - KEY TO MASTER CHART

M	-	MALE
F	-	FEMALE
N	-	NORMAL
AB	-	ABNORMAL
ND	-	NOT DONE
C	–	CERVICAL SPINE
T	-	THORACIC SPINE
L	-	LUMBAR SPINE
LT	-	LOWER THORACIC
UT	-	UPPER THORACIC
CVJ	-	CERVICOMEDULLARY JUNCTION
DB	-	DISC BULGE
DP	-	DISC PROTRUSION
EST	-	Extra Dural Soft Tissue
EML	-	Extra Medullary Lesion
CF	-	Compression Fracture
WCF	-	Wedge Compression Fracture
ODC	-	Osteophytic Disc Complex

NE	-	NON ENHANCEMENT
PE	-	PERIPHERAL ENHANCEMENT
HE	-	HOMOGENOUS ENHANCEMENT
0	-	PRESENT
1	-	NOT PRESENT
TM	-	Transverse Myelitis
CM	-	Cord Myelomalacia
CE	-	CORD EDEMA
CA	-	CORD ATROPHY
SACD	-	SUB ACUTE COMBINED DEGENERATION
DD	-	DEMYELINATING DISEASE
SEC	-	SPINAL EPIDERMOID CYST
CCS	-	CENTRAL CORD SYNDROME
MS	-	MULTIPLE SCLEROSIS
CD	-	CORRECT DIAGNOSIS
NMO	-	NEUROMYELITIS OPTICA
SCA	-	SPINAL CORD ASTROCYTOMA
ADEM	-	ACUTE DISSEMINATED ENCEPHALOMYELITIS

S.NO	MRI.NO	NAME	AGE	SEX
1	M9105	Basr daratty	27	M
2	M10857	Jasaram Sarang	40	M
3	M14507	Laxman	40	M
4	M13517	Kuber banu	60	M
5	M8892	Padmavati kurade	32	F
6	M14849	Vaman jambotkar	57	M
7	M15029	Ramappa ingalagi	50	M
8	M9614	Shanta devi	68	F
9	M13502	Sidramappa birads	68	M
10	M15354	Shahnaz maandar	3	F
11	M8668	Rajashekhar angadi	61	M
12	M13503	Rabiya mulla	33	F
13	M18516	Reshma kale	30	F
14	M15480	Manju Gupta	30	M
15	M15559	Prakash k	55	M
16	M14479	Laxman v kittur	69	M
17	M15057	Sagar ghazi	23	M
18	M9843	Sanjay patil	43	M
19	M10378	Shabana sharif	38	F
20	M13854	Tatyasab minache	68	M
21	M10315	Preeti asundi	7	F
22	M10428	Sonawwa bastwade	75	F
23	M6618	Anjana bommanawad	54	F
24	M14454	Imamsab nada	51	M
25	M11167	Imtiyaz patel	58	M
26	M13249	Mahadevi gouda	46	F
27	M10046	Mahaveer shirgavkar	68	M
28	M15069	Mallavva govvanko	35	F
29	M10293	Manjula rupan	45	F
30	M12784	Mudakkappa somana	62	M
31	M8590	Amar halakam	16	M
32	M13044	Nalini pattar	15	F
33	M6713	Nidhi rudrappa	17	F
34	M15375	Priyadarshini chavan	30	F
35	M11159	Sanmata patik	41	F
36	M14902	Shantabai A	75	F
37	M14579	Shivamma shivanagi	50	F
38	M14446	Sunita pishe	50	F
39	M13162	Suvarna dhavaleshwa	23	F
40	M12018	Vaishnavi mayannava	5	F
41	M13620	Vaishali galagali	44	F
42	M9567	Vilas malakannavar	48	M
43	M12121	Babu joshi	65	M
44	M9261	Shankar hubbali	58	M
45	M10831	Basagouda b	39	M
46	M122554	Hussain sahib	54	M
47	M9810	D Lingaih amarayya	59	M

48	M15123	Renuka jaggapur	27	F
49	M10155	Irappa basaki	53	M
50	M14815	Shivanand d	27	M
51	M9823	Irawwa paragond	60	F
52	M2872	Durgappa ankalgi	54	M
53	M	Saharada kaganole	20	F

CLINICAL SYMPTOMS	RADIOGRAPH	LEVEL OF LESION
Paraparesis with sphincter dysfunction	N	D3-D9 vertebrae
Cervical pain	AB	C5-C6 vertebrae
Loss of pain & temperature along both arms	N	C3-C6 & D1-D12 vertebrae
Cervical pain with paresthesia	AB	C3-C5 vertebrae
Quadriparesis with bowel incontinence	N	C1-C4 vertebrae
Upper back ache	AB	D6-D8, C5-C6 vertebrae
Radiating pain to bilateral upper limb	ND	D5-D10 vertebrae
Upper back ache		C4-C5 vertebrae
Neck pain radiating to both upper limbs	AB	C3-C4 vertebrae
Para-paresis with low grade fever	ND	D3-D8 vertebrae
Tingling & numbness in both hands	ND	At dens
Quadriparesis	AB	D1-D10 vertebrae
Loss of sensation in both hands & feet	N	C5-C7 vertebrae
Para paresis with sensory impairment	AB	D1-L2 vertebrae
Cervical pain	AB	D4-D6 vertebrae
Neck pain radiating to both upper limbs	AB	C5-C6 vertebrae
Cervical pain	ND	C4-C6 vertebrae
Cervical pain due to RTA	AB	D12 vertebra
Upper back ache	ND	C6-D7 vertebrae
Cervical pain	AB	C3-C4 vertebrae
Mild headache	N	C5-C6 vertebrae
Cervical pain radiating to bilateral limbs	AB	D12 vertebra
Bilateral upper limb motor impairment	N	C2-C8 vertebrae
Cervical pain with paresthesia	-	C2-C6 vertebrae
Tingling & numbnss in both hands	AB	C3-C5 vertebrae
Upper back ache	-	C4-C5 vertebrae
Right lower limb weakness	AB	D1-D2 vertebrae
Loss of sensation in both hands & feet	ND	C1-C5 vertebrae
Tetra paresis with sensory impairment	N	C2-D9 vertebrae
Neck pain radiating to both upper limbs	AB	C4-C6 vertebrae
Pain in left orbit with loss of vision	N	C6-C7 & D2-D10 vertebrae
Back pain with leg weakness	AB	D11-D12 vertebrae
Hemiparesis of left foot	AB	C3-C4 vertebrae
Paresthesia in both hands and feet	ND	CVJ-C6 vertebrae
Low back ache	AB	D12-L3 vertebrae
Bilateral lower limb weakness	ND	L1-L3 vertebrae
Paraparesis	N	D10-L2 vertebrae
Tetra paresis with sensory impairment	ND	C2-C6 vertebrae
Low back ache with limb weakness	ND	C5-C7, D2-D6 & D9-L1 vertebrae
No complaints	ND	D12-L2 vertebrae
Neck pain	N	C1 vertebra
Neck pain radiating to both upper limbs	AB	C5-C6 vertebrae
Upper back ache	AB	C2-C7 vertebrae
Back pain with lower limb weakness	AB	D1-D4 vertebrae
Upper back ache	ND	C6-D7 vertebrae
Back pain	AB	C3-C4 vertebrae
Neck pain	ND	C3-C4 vertebrae

Para paresis with sensory impairment	AB	D2-D8 vertebrae
Low back pain	ND	C3-C5 vertebrae
Quadriparesis with bowel incontinence	N	D6-D7 vertebrae
Radiating pain to bilateral upper limbs	AB	C5-C6 vertebrae
Quadriparesis with bowel incontinence	AB	D10-L1 vertebrae
Fever & left hemiparesis	N	D8-L1 vertebrae

C1-C4 vertebrae long ti kurade 32 F
Foc- AB D6-D8,

CAUSE OF SC CC	IMAGING FEATURES	POST CONTRAST	CORD CHANGES
	Long segment T2 hyperintensity	ND	-
DB	Subtle T2 hyper intensity	ND	+
	Long segment T2 hyper intensities	NE	+
	T2 cord hyper intensities	ND	+
	long segment T2 hyperintensity	ND	+
	Focal T2 hyper intensity	ND	+
	Focal T2 hyper intensity	NE	+
DB	Focal T2 hyper intensity	ND	+
DP	Cord T2 hyper intensities	ND	+
	Linear T2 cord hyper intensity	ND	+
EST	T2 cord hyper intensities	ND	+
	T2 intramedullary hyper intensities	NE	+
	T2 hyperintense lesion	ND	+
	Long segment T2 hyper intensities	PAE	+
	Linear central T2 hyper intensity	ND	+
DB	T2 cord hyper intensities	NE	+
EML	T2 cord hyper intensities	ND	+
CF	Subtle T2 hyperintesity	ND	+
DB	T2 cord hyper intensities	ND	+
DB	T2 hyper intensities	ND	+
	Linear T2 hyper intensity	ND	+
WCF	Subtle T2 hyper intensity	ND	+
	T2 hyper & T1hypo	ND	-
	Subtle T2 hyper intensity	ND	+
	Linear T2 hyper intensity	ND	+
	Focal T2 hyper intensity	ND	+
	Focal T2 hyper intensity	NE	+
	Posterior column T2 hyper intensity	NE	+
	long segment diffuse T2 hyperintensity	PE	+
	Focal T2 hyper intensity	ND	+
	long segment intramedullary T2 hyperintensity	NE	+
	T2 hyper & T1hypo DWI- bright	HE	+
	Few T2hyperintense lesions	NE	+
	Inverted V shaped T2 hyperintensity	ND	+
	T2-heterogenous DWI-bright ADC- low	NE	+
	T2 hyper & T1 iso	HIE	+
	Long segment T2 hyperintensity	NE	+
	SubtleT2 hyperintensity	ND	+
	T2-hyper DWI-bright ADC- low	NE	+
	T2 hyperintensity	ND	-
	Focal T2 hyper intensity	ND	+
ODC	Focal T2 hyper intensity	ND	+
ODC	Focal T2 hyper intensity	ND	+
	Expansile T2 hyper & T1hypo DWI- bright	HE	+
	Linear central T2 hyper intensity	ND	+
DP	SubtleT2 hyperintensity	ND	+
	SubtleT2 hyperintensity	ND	+

	long segment T2 hyperintensity	PE	+
	T2 cord hyper intensities	ND	+
Lesion	T2 hyper & T1 hypodense	ND	-
	T2 cord hyper intensities	ND	+
	T2 intramedullary hyper intensity	ND	+
	long segment T2 hyperintensity	ND	+

LIGAMENTOUS DISRUPTION	POSTERIOR	PRE & PARA VERTEBRAL COLLECTION	MR DIAGN	FOLLOW U	REMARKS
-	N	-	TM	-	-
-	AB	+	CM	Y	CD
-	AB	-	Syringohdr	Y	CD
-	AB	-	CM	Y	CD
-	N	-	TM	Y	CD
-	N	-	Syrinx, CM	Y	CD
-	AB	+	Syrinx	Y	CD
+	AB	-	CM	-	-
+	N	-	CE	Y	Equivocal
-	N	+	Syringomye	Y	CD
+	AB	-	CM	Y	Equivocal
+	AB	+	TM	-	-
-	N	-	Syringohdr	Y	CD
-	AB	+	Myelitis	Y	Equivocal
-	AB	-	Syrinx	Y	CD
+	AB	+	CM	-	-
-	AB	-	CM	Y	CD
+	N	-	CE	Y	CD
+	N	-	CM	Y	Equivocal
-	AB	-	CM	Expired	CD
-	N	-	Syrinx	Y	CD
+	AB	+	CM	Expired	CD
-	AB	-	Syringomye	Y	CD
-	N	-	Metastase	-	-
-	AB	-	CA with CM	-	#NAME?
-	N	-	Focal syrin	Y	Equivocal
-	N	-	VCH	-	-
-	AB	-	SACD	Y	CD
-	AB	-	TM	Y	Equivocal
+	N	-	CM	-	-
-	N	-	TM	Y	NMO
-	AB	-	Neoplasm	Y	SCA
-	N	-	DD	Y	MS
-	N	-	SACD	Y	CD
+	AB	-	SEC	Y	CD
-	AB	-	Neoplasm	Y	SCA
+	N	-	TM	Y	CD
+	N	-	TM	Y	CD
-	N	-	SCI	Y	CD
-	N	-	Syrinx	-	-
-	N	-	Syrinx	Y	CD
+	AB	N	CM	Expired	CD
+	AB	-	CM	-	-
-	N	-	Astrocytom	Expired	CD
-	AB	-	Syrinx	Y	CD
-	AB	-	CM	-	-
+	N	-	CM	Y	CD

-	N	-	TM	Y	CD
-	AB	-	CC	-	-
-	N	-	Meningiom	Expired	CD
+	AB	-	CCS	Y	CD
+	AB	-	TM	Y	CD
-	N	-	NMO	Y	ADEM

POST CONTRAST	
NE	
HE	
HIE	
ND	
NE	
PAE	
PE	
PE	
Grand Total	

POST CO
NE
HE
HIE
ND
NE
PAE
PE
PE
Grand Tot
0
0
0
0
0
0

44.45
17.61

0	
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00

Count of FOLLOW UP	
FOLLOW UP	Total
Expired	5
Y	37
(blank)	
Grand Total	42

FOLLOW
Expired
Y
(blank)
Grand Tot
0
0
0
0
0
0
0
0
0
0
0
0
0
0
0
0
0
0
0

Total	
5	9.43
37	69.81
0	0.00
42	79.25
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00

Count of REMARKS	
REMARKS	Total
ADEM	1
CD	31
Equivocal	6
MS	1
NMO	1
SCA	2
(blank)	
Grand Total	42

REMARK
ADEM
CD
Equivocal
MS
NMO
SCA
(blank)
Grand Tot
0
0
0
0
0
0
0
0
0
0
0
0
0
0

Total	
1	1.89
31	58.49
6	11.32
1	1.89
1	1.89
2	3.77
0	0.00
42	79.25
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00
0	0.00

S.NO	MRL NO.	AGE	SEX	CLINICAL SYMPTOMS	DURATION OF SYMPTOMS	RADIOGRAPH	LEVEL OF LESION	LOCATION OF LESION IN CORD	CAUSE OF SC COMPRESSION	POST CONTRAST	CORD CHANGES	LIGAMENTOUS DISRUPTION	POSTERIOR ELEMENTS	PRE & PARA VERTEBRAL COLLECTION	MR DIAGNOSIS	FOLLOW UP	REMARKS
1	M9105	27	M	Para paresis	ACUTE	N	LT	CENTRAL		ND	0	0	N	0	TM	N	CD
2	M10857	40	M	Cervical pain	CHRONIC	AB	C	CENTRAL	DB	ND	1	0	AB	1	CM	Y	CD
3	M14507	40	M	Loss of sensation in both hands & feet	CHRONIC	N	C,T	CENTRAL		NE	1	0	AB	0	Syringohdromyelia	Y	CD
4	M13517	60	M	Cervical pain	SUBACUTE	AB	C	CENTRAL		ND	1	0	AB	0	CM	Y	CD
5	M8892	32	F	Quadriparesis	ACUTE	N	C	CENTRAL		ND	1	0	N	0	TM	Y	CD
6	M14849	57	M	Back ache	CHRONIC	AB	C,LT	CENTRAL		ND	1	0	N	0	Syrinx, CM	Y	CD
7	M15029	50	M	Radiating pain to bilateral upper limbs	CHRONIC	ND	LT	CENTRAL		NE	1	0	AB	1	Syrinx	Y	CD
8	M9614	68	F	Back ache	CHRONIC	ND	C	CENTRAL	DB	ND	1	1	AB	0	CM	N	
9	M13502	68	M	Cervical pain	ACUTE	AB	C	CENTRAL	DP	ND	1	1	N	0	CE	Y	Equivocal
10	M15354	13	F	Para paresis	CHRONIC	ND	T	CENTRAL		ND	1	0	N	1	Syringomyelia	Y	CD
11	M8668	61	M	Tingling & numbness in both hands	CHRONIC	ND	Atdens	CENTRAL	EST	ND	1	1	AB	0	CM	Y	Equivocal
12	M13503	33	F	Quadriparesis	SUBACUTE	AB	T	CENTRAL		NE	1	1	AB	1	TM	N	
13	M18516	30	F	Loss of sensation in both hands & feet	CHRONIC	N	C	CENTRAL		ND	1	0	N	0	Syringohdromyelia	Y	CD
14	M15480	30	M	Para paresis	SUBACUTE	AB	T,L	CENTRAL		PAE	1	0	AB	1	Myelitis	Y	Equivocal
15	M15559	55	M	Cervical pain	CHRONIC	AB	LT	CENTRAL		ND	1	0	AB	0	Syrinx	Y	CD
16	M14479	69	M	Cervical pain	CHRONIC	AB	C	CENTRAL	DB	NE	1	1	AB	1	CM	N	
17	M15057	23	M	Cervical pain	CHRONIC	ND	C	CENTRAL	EML	ND	1	0	AB	0	CM	Y	CD
18	M9843	43	M	Cervical pain	ACUTE	AB	T	CENTRAL	CF	ND	1	1	N	0	CE	Y	CD
19	M10378	38	F	Back ache	CHRONIC	ND	C,T	CENTRAL	DB	ND	1	1	N	0	CM	Y	Equivocal
20	M13854	68	M	Cervical pain	CHRONIC	AB	C	CENTRAL	DB	ND	1	0	AB	0	CM	Expired	CD
21	M10315	17	F	Mild headache	CHRONIC	N	C	CENTRAL		ND	1	0	N	0	Syrinx	Y	CD
22	M10428	75	F	Cervical pain	CHRONIC	AB	T	CENTRAL	WCF	ND	1	1	AB	1	CM	Expired	CD
23	M6618	54	F	Tingling & numbness in both hands	CHRONIC	N	C	CENTRAL		ND	0	0	AB	0	Syringomyelia	Y	CD
24	M14454	51	M	Cervical pain	SUBACUTE	ND	C	CENTRAL		ND	1	0	N	0	Metastase	N	
25	M11167	58	M	Tingling & numbness in both hands	CHRONIC	AB	C	CENTRAL		ND	1	0	AB	0	CA with CM	Y	CD
26	M13249	46	F	Back ache	SUBACTE	ND	C	CENTRAL		ND	1	0	N	0	Focal syrinx	Y	Equivocal
27	M10046	68	M	Hemiparesis of left foot	CHRONIC	AB	T	CENTRAL		NE	1	0	N	0	CM	N	
28	M15069	35	F	Loss of sensation in both hands & feet	SUBACUTE	ND	C	POSTERIOR		NE	1	0	AB	0	SACD	Y	CD
29	M10293	45	F	Tetra paresis	ACUTE	N	C,LT	CENTRAL		PE	1	0	AB	0	TM	Y	Equivocal
30	M12784	62	M	Cervical pain	CHRONIC	AB	C	CENTRAL		ND	1	1	N	0	CM	N	
31	M8590	16	M	Pain in left orbit with loss of vision	ACUTE	N	C,T	CENTRAL		NE	1	0	N	0	TM	Y	NMO
32	M13044	15	F	Back ache	SUBACUTE	AB	LT	CENTRAL		HE	1	0	AB	0	Neoplasm	Y	SCA
33	M6713	17	F	Hemiparesis of left foot	ACUTE	AB	C	ANTERIOR		NE	1	0	N	0	DD	Y	MS
34	M15375	30	F	Loss of sensation in both hands & feet	SUBACUTE	ND	CVJ,C	POSTERIOR		ND	1	0	N	0	SACD	Y	CD
35	M11159	41	F	Back ache	CHRONIC	AB	LT,L	CENTRAL		NE	1	1	AB	0	SEC	Y	CD
36	M14902	75	F	Bilateral lower limb weakness	SUBACUTE	ND	L	CENTRAL		HIE	1	0	AB	0	Neoplasm	Y	lymphoma
37	M14579	50	F	Para paresis	ACUTE	N	LT,L	CENTRAL		NE	1	1	N	0	TM	Y	CD
38	M14446	50	F	Tetra paresis	ACUTE	ND	C	CENTRAL		ND	1	1	N	0	TM	Y	CD
39	M13162	23	F	Back ache	ACUTE	ND	C,T,L	ANTERIOR		NE	1	0	N	0	SCI	Y	CD
40	M12018	15	F	No complaints	CHRONIC	ND	LT,L	CENTRAL		ND	0	0	N	0	Syrinx	N	
41	M13620	44	F	Cervical pain	CHRONIC	N	C	CENTRAL		ND	1	0	N	0	Syrinx	Y	CD
42	M9567	48	M	Cervical pain	CHRONIC	AB	C	CENTRAL	ODC	ND	1	1	AB	0	CM	Expired	CD
43	M12121	65	M	Back ache	CHRONIC	AB	C	CENTRAL	ODC	ND	1	1	AB	0	CM	N	
44	M9261	58	M	Back ache	SUBACUTE	AB	UT	CENTRAL		HE	1	0	N	0	Astrocytoma	Expired	CD
45	M10831	39	M	Back ache	CHRONIC	ND	C,T	CENTRAL		ND	1	0	AB	0	Syrinx	Y	CD
46	M122554	54	M	Back ache	CHRONIC	AB	C	CENTRAL	DP	ND	1	0	AB	0	CM	N	
47	M9810	59	M	Cervical pain	CHRONIC	ND	C	CENTRAL		ND	1	1	N	0	CM	Y	CD
48	M15123	27	F	Para paresis	SUBACUTE	AB	UT	CENTRAL		PE	1	0	N	0	TM	Y	CD
49	M10155	53	M	Back ache	ACUTE	ND	C	CENTRAL		ND	1	0	AB	0	CC	N	
50	M14815	27	M	Quadriparesis	CHRONIC	N	T	CENTRAL	Lesion	ND	0	0	N	0	EPENDYMOMA	Expired	CD
51	M9823	60	F	Radiating pain to bilateral upper limbs	SUBACUTE	AB	C	CENTRAL		ND	1	1	AB	0	CCS	Y	CD

Pre & para	No of patients
Negative	46
Positive	7
Not done	17
Loss of sen	7.55
Quadripare	7.55
Hemiparesi	5.66
Tingling & r	5.66
Radiating p	3.77
Tetra pares	3.77
Bilateral lo	1.89
Mild headа	1.89
Pain in left	1.89

Clinical sym	Cervical pa	Back ache	Para paresi	Loss of sen	Quadripare
% of patien	26.42	22.64	9.43	7.55	7.55

Pre & para	Negative	Positive	Not done
No of patie	46	7	17

Hemiparesi	Tingling &	Radiating p	Tetra pares	Bilateral lo	Mild headа	Pain in left orbit with loss of vision
5.66	5.66	3.77	3.77	1.89	1.89	1.89