"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION"

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the requirements
of

M.D. DEGREE EXAMINATION
BRANCH – VIII– RADIODIAGNOSIS

MADRAS MEDICAL COLLEGE
&
RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL
CHENNAI– 600 003

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI-TAMILNADU,INDIA.

APRIL 2017

CERTIFICATE

This is to certify that the dissertation titled **"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION"** submitted by **Dr.R.BHARATHIPRIYA** appearing for **M.D (Radiodiagnosis)** degree examination in April 2017 is a bonafide record of work done by her under my guidance and supervision in partial fulfillment of requirement of the TamilNadu Dr. M.G.R Medical University, Chennai. I forward this to the TamilNadu Dr. M.G.R Medical University, Chennai.

DR.S.BABU PETER , MDRD,D.N.B,
Guide,
Associate Professor,
Barnard institute of Radiology,
Madras Medical College,
Rajiv Gandhi Government General Hospital,
Chennai – 600 003.

PROF.R.RAVI, MD.,DMRD .,
Professor & Head of Department,
Barnard Institute of Radiology,
Madras Medical College &
Rajiv Gandhi Government General Hospital,
Chennai - 600 003.

PROF.N.KAILASANATHAN MD.,D.M.R.D.,
Director & Professor,
Barnard Institute of Radiology,
Madras Medical College,
Rajiv Gandhi Government General Hospital,
Chennai - 600 003.

PROF.DR.MK. MURALIDHARAN MS,MCH.,
Dean,
Madras medical college,
Rajiv Gandhi Government General Hospital,
Chennai - 600 003.

DECLARATION

I **Dr.R.BHARATHIPRIYA**, solemnly declare that this dissertation titled **"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION**" is a bonafide work done by me at the Barnard Institute of Radiology, Madras Medical College and Government General Hospital, under the supervision of the Professor N.KAILASANATHAN,M.D.,D.M.R.D., Director, Barnard Institute of Radiology, Madras Medical College and Rajiv Gandhi Government General Hospital. This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of M.D. Degree Radiodiagnosis.

Place: Chennai

Date: 28.9.2016 **DR.R.BHARATHIPRIYA**

ACKNOWLEDGEMENT

I express my heartful gratitude to the Dean, **PROF.MK.MURALIDHARAN MS,MCH** Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai-3 for permitting me to do this study.

I gratefully acknowledge and sincerely thank our Director **Prof. N.KAILASANATHAN M.D.,D.M.R.D.,** Barnard Institute of Radiology, for his valuable suggestions, guidance, constant supervision and moral support without which this study would not have been possible.

I express my gratitude to **Prof.R. RAVI, M.D., D.M.R.D.,** Head of the Department, Barnard Institute of Radiology, for his valuable guidance in doing the dissertation work.

I owe a lot to my guide, **DR.S.BABU PETER, M.D., D.N.B.,** whose expert guidance constant encouragement created an interest for me to pursue this study on advanced MRI imaging. It is his constant supervision and support, that made me possible to finish this study without much difficulty.

I am extremely thankful to my Professor, **Dr.K.MALATHY, M.D.,D.M.R.D.,Associate professor, Dr.D.RAMESH,M.D., Dr.S.KALPANA, M.D.,D.N.B., Prof. A.P.ANNADURAI, M.D., D.M.R.D.,Dr.MANIMEGALA MDRD,DNB.,Dr.KASIVISALAKSHI**

MDRD, and assistant professors Dr.J.CHEZHIAN,M.D., Dr.K.GEETHA, M.D., Dr.MOHIDEEN ASHRAF MD,DMRD.,Dr.G.GEETHA,MDRD,Dr.IYENGARAN MDRD and Tutors **Dr.SARANYA, D.M.R.D, and Dr. M.P.BALAN, D.M.R.D,** in the Barnard Institute of Radiology for their constant support, encouragement and advice during my study.

I also thank **my past and present fellow postgraduates** who helped me in carrying out my work and preparing this dissertation.

I thank **all Radiology technicians, Staff Nurses and all the Paramedical staff members** in Barnard Institute of Radiology, for their co- operation in conducting the study.

I thank my family members for their understanding and co-operation in completion of this work.

Last but not the least; I owe my sincere gratitude to the patients and their relatives who co-operated for this study, without whom the study could not have been possible.

INDEX

INTRODUCTION

A spectrum of diagnostic considerations may affect the spinal cord which includes developmental anomalies, inflammatory and infectious processes, vascular disease, degenerative conditions as well as benign and malignant neoplasms. Patients with intramedullary spinal cord lesions commonly present with tingling pain, numbness and weakness.

Magnetic resonance imaging is the current imaging modality of choice in the evaluation of patients presenting with myelopathic symptoms in the search for spinal cord lesions. It is important to recognize and differentiate non neoplastic from the neoplastic process of the spinal cord as the two entities differentiation is extremely crucial to the neurosurgeon.

Magnetic resonance spectroscopy is a noninvasive tool which helps to characterise the chemical composition of human tissue[1] . Thereby it can help to better characterize pathologic processes affecting the spinal cord and also helps to provide important clinical markers for differential diagnosis.

RATIONALE FOR THE STUDY

For several years Magnetic resonance spectroscopy has been applied in the investigation of pathologic processes involving the brain and has gained an increased acceptance by its potential in differentiating high- versus low-grade tumors,distinguishing tumor from nontumoral tissue[2],differentiating solid lesions from cysts or abscesses[3], monitoring the results of treatment, and occasionally predicting outcome.

The information obtained by Magnetic resonance spectroscopy helps to differentiate benign vs malignant lesions and may often prevent unnecessary invasive interventions like surgery &biopsy thus it can avoid further negative impact on patient outcome[1].

ANATOMY OF SPINAL CORD

The spinal cord is about **45 cm** in length and with the thickness of 13 mm. It extends from the foramen magnum upto the level of first or second lumbar vertebrae.

It is enclosed by the protective bony spinalcanal . At the lower end of the cord, the fibers separates into the cauda equina. The spinal cord is the most important communicating structure between the body and brain.

The brain transmit motor impulses to the all four limbs and the body through the spinal cord for allowing movement. “Spinal reflexes” protect our body immediately from acute harm.

Quadriplegia or tetraplegia (paralysis of all four limbs)occur from cervical spinal cord injury, sensory and motor function loss usually occur below the level of spinal cord injury.

SPINAL NERVES

- 31 pairs of spinal nerves arise from the spinal cord.
- 8 cervical ,12 thoracic, 5 lumbar,5 sacral, 1 coccygeal

The spinal nerves act as "telephone lines," carrying impulses between body and spinal cord to control sensation and movement. Each spinal nerve has two(2) roots -ventral and dorsal root.

"The ventral (front) root carries motor impulses **from** the brain and the dorsal (back) root carries sensory impulses **to** the brain".

The two ventral and dorsal nerve roots fused together to form a spinal nerve, which enters in the spinal canal, along with the cord, it reaches its exit hole - the inter vertebral foramen'. When the nerve passes into the inter vertebral foramen, it branches and each branch has both motor and sensory fibers.

SPINAL NERVES

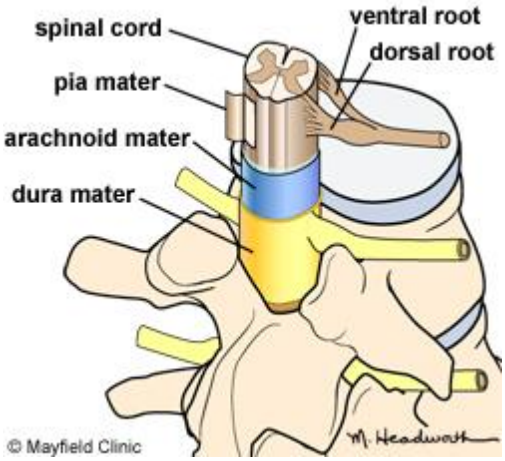


Fig :1 Both the dorsal and venral nerve roots join to form the spinal nerve. The cord is covered by three layers of meninges: inner pia, middle arachnoid and outer dura mater.

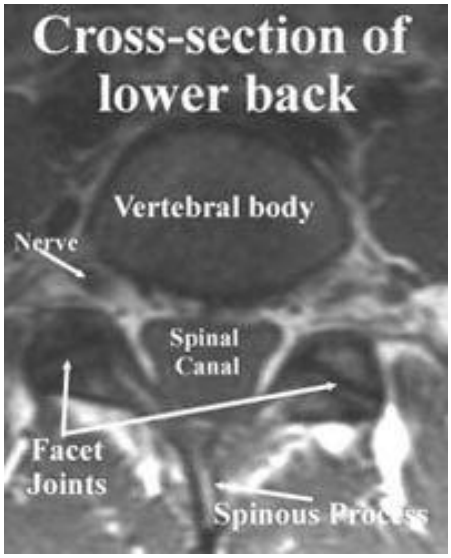


Fig:2 cross sectional view of spinal cord in Magnetic resonance imaging

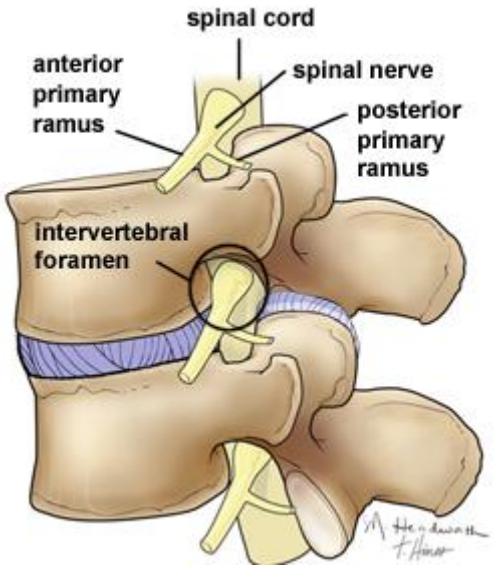


Fig:3 Spinal nerves leave the spinal canal through the intervertebral foramen below each pedicle.

- Dermatomes are specific region of the body innervated by a specific nerve, this pattern is useful to identify the location of a spinal problem based on the area of pain or muscle weakness.

For ex: sciatica usually denotes a problem at the level of L4-S3 nerves.

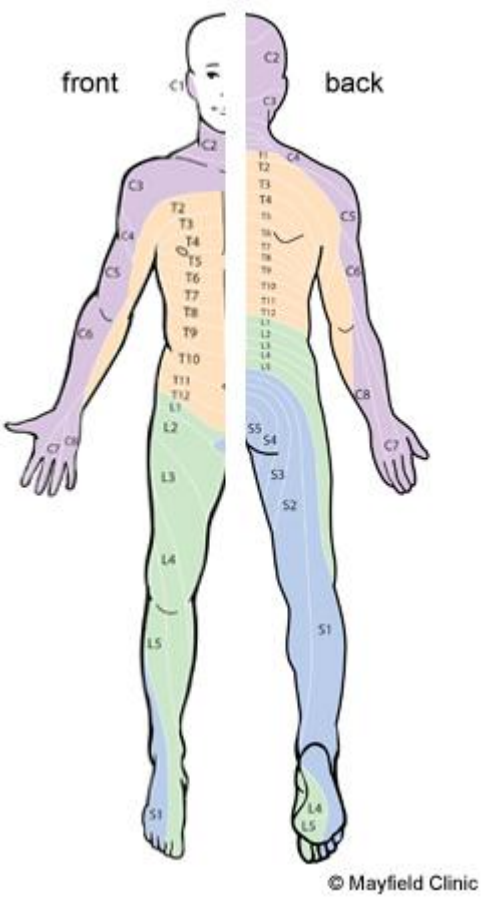


Fig:4 Dermatome pattern.

COVERINGS & SPACES

The spinal cord is covered by three layers of meninges with inner membrane is the pia mater, which is intimately attached to the cord,middle membrane is the arachnoid mater and the outer membrane is the tough dura mater.Between these membranes are subdural and subarachnoid spaces.

SPINAL COVERINGS AND SPACES

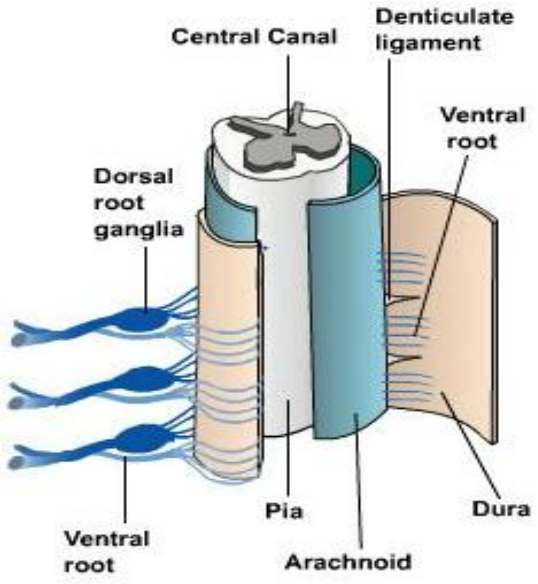


Fig :5 Coverings and spaces of spinal cord

Subarachnoid space - The space between the pia and arachnoid mater which surrounds the cord and contains CSF

Epidural space – The space between the dura mater and the bone

Subdural space – the space between the dura and arachnoid

SPINAL CORD STRUCTURE

The Central nervous system usually categorised into white matter & gray matter. White matter is the one that contains myelinated axons which form the tracts ,that conducts information between multiple regions & structures in the central nervous system . “Gray matter made up of the cell bodies & dendrites -site of synaptic transmission”.

The gray matter of the cord usually arranged in three(3) horns.

Anterior horn - motor

Lateral - visceral efferent & afferent

Posterior horn - sensory function

The anterior horn is further divided into a ventral part,head,dorsal part and the base.

In the brain, cortical (outer) region, made up of mainly gray matter while the the deep brain tissues contains various white matter tracts with some exceptions as deep grey nuclei (basal ganglia & thalamic nuclei) are composed of gray matter

Unlike brain, central butterfly shaped gray matter is surrounded by white matter in spinal cord indicating- "the spinal cord tracts carry information up and down along the outer aspects, and synaptic transmission occur more centrally".

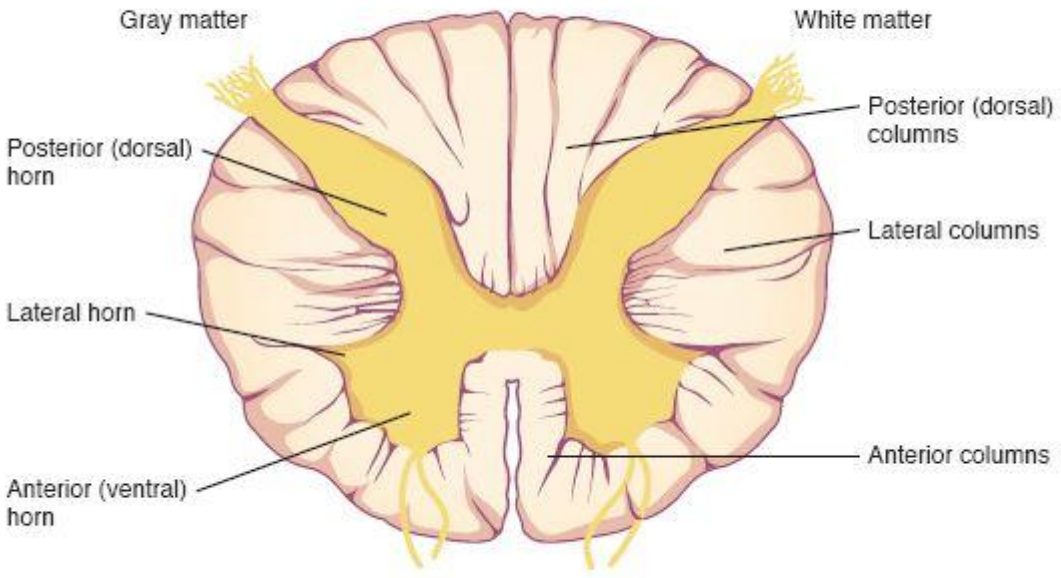


Fig:6 Spinal cord segment - cross section view

LAMINAR ORGANISATION

In thick cross section ,the cord appears to have a laminar pattern of arrangement.ten layers are identified known as laminae of rexed,-numbered from the tip of the dorsal horn moving ventrally into ventral horn.

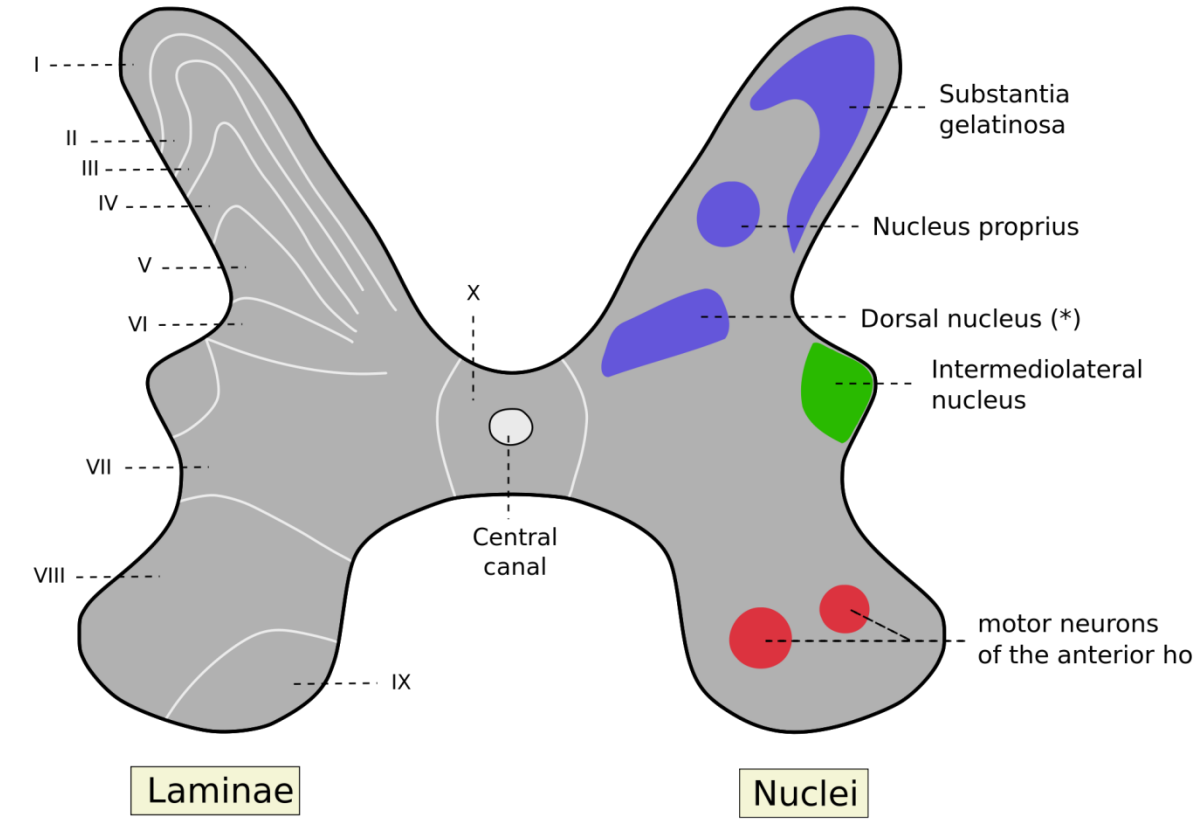


Fig:7 Cell groups and various laminae in spinal cord

In the spinal cord , the central grey matter is usually "butterfly shaped", with each side of the "butterfly" contains a dorsal horn (posterior) and ventral (anterior) horn. Each horns is connected with the spinal nerve roots.

WHITE MATTER OF SPINAL CORD

↓

SEGMENT

↓

COLUMNS

↓

TRACTS

COLUMNS:	**TRACTS:**
Anterior	Ascending
Posterior	Desending
Lateral	

SPINAL CORD TRACTS

A TRACT is a collection of multiple nerve fibers that connects two grey matter masses with in the CNS(central nervous system)

Tracts may be ascending or desending named after the grey matter masses connected by them.

Tracts are also called “Fasiculi or leminisci”

The WM of the cord is divided into 1.posterior (dorsal), 2.lateral, 3.anterior (ventral) columns. These columns are sometimes called “funiculi”.

Sensory information carried from the periphery to the brain by ascending tracts , while the motor signals carried from brain to muscles and glands by the desending tracts.

The columns further divided into tracts (sometimes called “fascicule”) they are named according to the structures that they connect which. For EX: “the spinothalamic tract - carrying information from the spinal cord to the thalamus of the brainstem, and it is an ascending tract, so it carries sensory information.

Most of the motor control is contralateral.

For Ex: right upper and lower limb is controlled by the motor area in the left cerebral cortex . Some tract have an origin and destination on the same side of the body known as - an ispsilateral relationship.

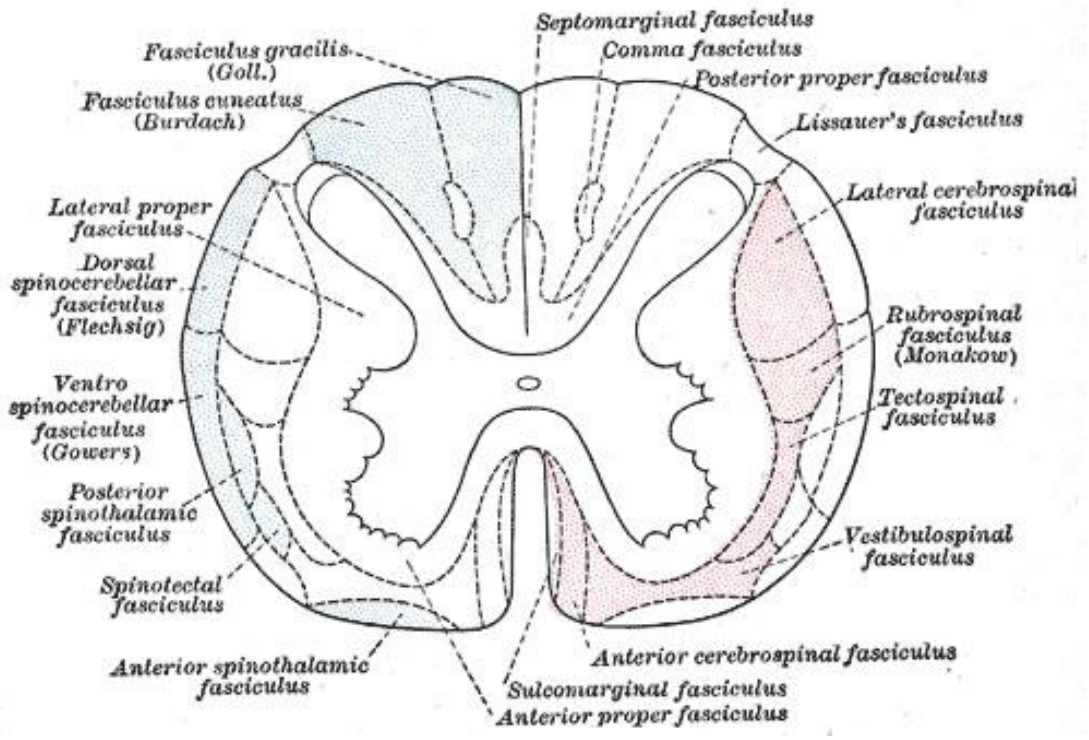


Fig :8 White matter columns &various tracts –cross section

Table:1

Spinal Tracts

Tract	Column Location	Dessusitation location	Function
Ascending Tracts - Sensory			
Gracile fasciculus	posterior	medulla	below level T6: limb and trunk position sensations; deep touch; visceral pain; vibration
Cuneate fasciculus	posterior	medulla	level T6 and above: limb and trunk position sensations; deep touch; visceral pain; vibration
Spinothalamic	lateral and anterior	spinal cord	light touch, tickle, itch, temperature, pain, and pressure sensations
Spinoreticular	lateral and anterior	some fibers of the spinal cord	pain sensation from tissue injury
Posterior spinocerebellar	lateral	none	proprioception - feedback from muscles
Anterior spinocerebellar	lateral	spinal cord	proprioception - feedback from muscles
Descending Tracts - Motor			
Lateral corticospinal	lateral	medulla	fine limb control
Anterior corticospinal	anterior	spinal cord	fine limb control
Tectospinal	anterior	midbrain	head-turning reflex in response to visual and auditory stimuli
Lateral reticulospinal	lateral	none	posture and balance; awareness of pain regulation
Medial reticulospinal	anterior	none	posture and balance; awareness of pain regulation
Lateral vestibulospinal	anterior	none	posture and balance
Medial vestibulospinal	anterior	some fibers of the medulla	head position control

This table lists the major spinal tracts, indicates if they decussate, and provides a brief description of the types of information that they carry.

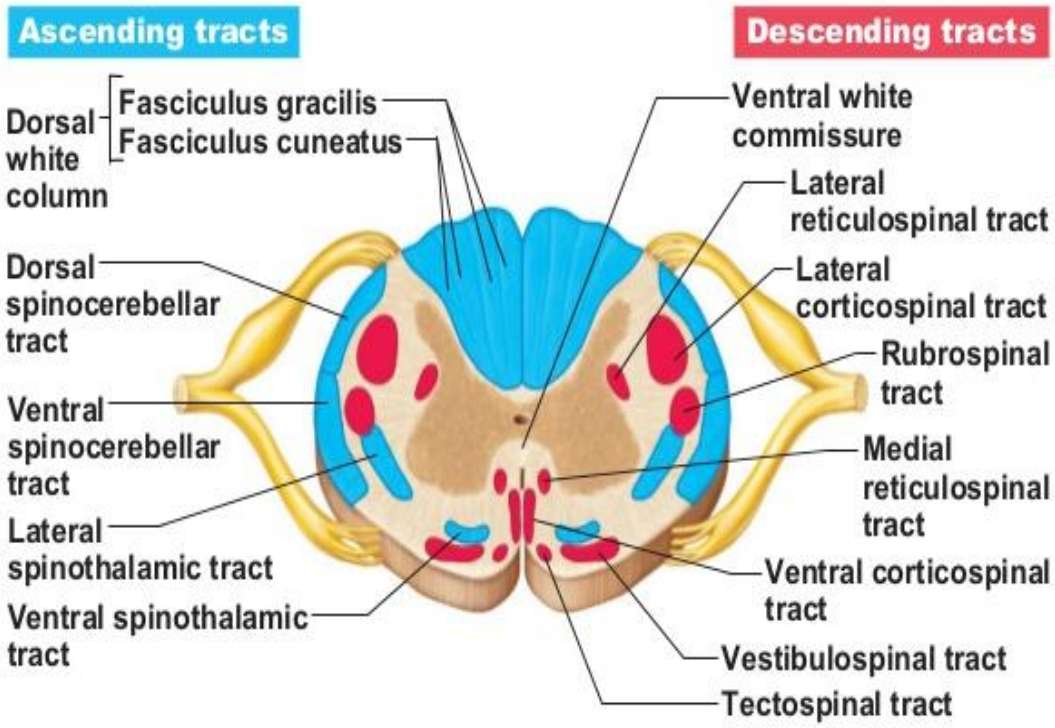


Fig: 8 Graphic representation of spinal cord tracts

INTRA MEDULLARY SPINAL CORD LESIONS

A spectrum of lesions may affect the spinal cord that includes developmental anomalies, inflammatory and infectious processes, degenerative disease, vascular disease, as well as benign and malignant neoplasms. Patients with intramedullary spinal cord lesions usually come with symptoms like tingling sensation,pain and numbness or weakness.

CLASSIFICATION OF INTRAMEDULLARY SPINAL CORD LESIONS

1. NEOPLASMS

A. Glial Neoplasms

I. Ependymoma

II. Astrocytoma

i. Juvenile Pilocytic Astrocytoma

ii. Anaplastic Astrocytoma

iii. Glioblastoma Multiforme

III. Ganglioglioma

IV. Subependymoma

B. Non-glial Neoplasms

I. Hemangioblastoma

II. Intramedullary Metastasis

III. Solitary fibrous tumor

IV. Spinal cord lipoma

2. VASCULAR

A. Cavernous malformation

B. Spinal dural arteriovenous fistula

C. Arterial venous malformation

D. Spinal cord ischemia/infarction

3. INFLAMMATORY/INFECTION

A. Multiple Sclerosis

B. Neuromyelitis Optica

C. Granulomatous Angiitis

D. Neurosarcoid

E. Tuberculosis

4. TRAUMA/DEGENERATIVE

A. Spinal cord contusion

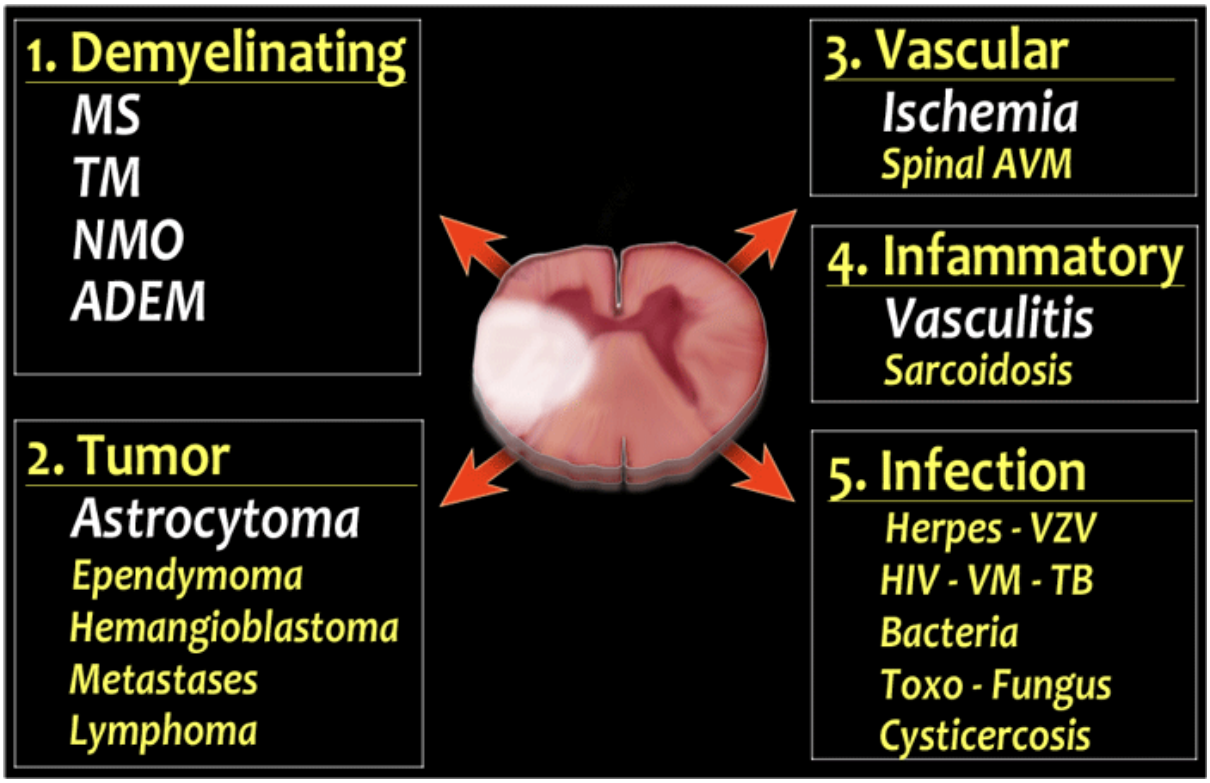


Fig:9 Diagram representing various lesions of spinal cord

INTRA MEDULLARY SPINAL CORD NEOPLASMS

- Spinal cord intramedullary neoplasms constitute about 4%–10% of all central nervous system (CNS) tumors and about 2%–4% of CNS glial tumors[4].
- They account for 20% of all intraspinal tumors in adults and 35% of all intraspinal tumors in paediatric age group[5].
- Intramedullary spinal cord neoplasms are more often common in patients with NF-1 (astrocytomas), NF-2 (ependymomas), and VHL (hemangioblastomas).
- 70% of tumors are associated with cysts. Two types of recognized cysts:
- Intratumoral cysts:
    - Contained within the tumor itself
    - demonstrate peripheral enhancement
    - Most commonly occurs in ganglioglioma (46%), ependymoma (22%), astrocytoma (21%), and hemangioblastoma (2-4%)[6]
- Non-tumoral cysts:
    - seen at the rostral or caudal part of the tumor
    - Occurs because of dilatation of the central canal
    - Do not show enhancement
    - Present in 60% of all intramedullary spinal tumors

- Syrinx occurs in approximately 50% of all intramedullary tumors but is most often associated with hemangioblastomas.

- In contrast to intracranial neoplasms, the vast majority of spinal cord neoplasms, including even low-grade forms, enhance after the administration of contrast material to some degree. more active portions of the tumors are the areas showing contrast enhancement and may indicate potential sites for biopsy if resection is not feasible.

EPENDYMOMA

- Most common intramedullary neoplasm in adults, comprising 60% of all glial spinal cord tumors.
- Second most common intramedullary neoplasm in the pediatric population, representing 30% of pediatric intramedullary spinal neoplasms.
- Classified as WHO Grade I or II neoplasm.
- Most commonly occurs in the cervical region (44-67%).
- Characterized by slow growth and tends to compress rather than infiltrate adjacent spinal cord neural tissue.
- Well circumscribed lesion that almost always has a cleave plane, which facilitates microsurgical resection (treatment of choice).
- Symmetric cord expansion as it arises from the ependymal cells of the central canal within the spinal cord.
- Rostral and caudal non-tumoral polar cysts are common (Intratumoral cysts are less common).
- Intratumoral hemorrhage, cystic degeneration, and hemosiderin cap is common. Unlike intracranial ependymomas, calcifications are uncommon.
- Multiple ependymomas can be seen in the setting of Neurofibromatosis-2 (MISME Syndrome – Meningioma, Schwannoma, and Ependymoma).

ASTROCYTOMA

Characterized as:

1. WHO Grade I – Juvenile Pilocytic Astrocytoma
2. WHO Grade II – Fibrillary Astrocytoma
3. WHO Grade III – Anaplastic Astryocytoma
4. WHO Grade IV – Glioblastoma Multiforme (GBM)

- Most common in children, 2nd most common tumor in adults.
- Comprises approximately 40% of spinal tumors.
- Generally large, diffuse fusiform enlargement without obvious (infiltrative) margins.Patchy ill-defined enhancement.
- Can involve the full diameter of the cord but are more eccentric in location compared to ependymomas.
- Both polar and intratumoral cysts are common. Leptomeningeal spread in seen in 60% of intramedullary GBM.

Table :2 Difference between ependymoma and astrocytoma

	Ependymoma	Astrocytoma
Population	Adult	Pediatric
Location	Central	Eccentric
Morphologic Appearance	Well-circumscribed	Ill-defined
Hemosiderin Cap	Common	Rare
Enhancement	Intense, homogeneous	Patchy, irregular
Cysts	Common	Common

SPINAL GANGLIOGLIOMA

- Spinal ganglioglioma are rare tumours that constitutes 1.1% of all spinal cord lesions
- Common in children representing 15% of intramedullary neoplasm in the paediatric age group.they are WHO grade 1 or 2 neoplasms[7].
- LOCATION:cervical cord followed by thoracic cord in eccentric location.
- Typically involve long segment of spinal cord extending more than eight vertebral body segments.
- 46% of these tumours contain intratumoural cysts.
- Mixed signal intensity in T1 and high signal intensity in T2 WI with patchy enhancement.
- Calcifications common.

SPINAL HEMANGIOBLASTOMA

- Constitutes 2-6% of intramedullary spinal tumours.peak presentation in fourth decade[8].
- One third of the pts with this tumour have von hippel lindau syndrome.
- They are vascular benign tumours and do not undergo malignant tansformation.
- Location:thoracic cord followed by cervical cord.
- Most of the hemangioblastoma have intramedullary component with two thirds located ecentricaly wih exophytic component.
- Only 25% are entirely intramedullary
- Associated tumour cysts and syrinx are common.

- Variable signal intensiy in T1 and iso- hyper in T2WI with areas of flow voids.
- vivid enhancement in post contrast study.

INTRA MEDULLARY SPINAL METASTASIS

- Very rare occurring approximately 1% of spinal tumours
- Primaries –lung carcinoma,breast ca ,lymphoma,leukemia,malignant melanoma, renal cell ca ,colonic carcinoma.
- Location: cervical cord followed by thoracic and lumbar cord
- Lesions are usually solitary and involve 2-3 vertebral segments
- Lesion is hypointense in T1 and hyperintense in T2 with surrounding edema which usually shows avid homogenous contrast enhancement.

INFLAMMATORY INTRAMEDULLARY SPINAL CORD LESIONS

ACUTE TRANSVERSE MYELITIS

- Rapidly progressive clinical course
- Etiology-

  idiopathic,postinfectious(ADEM),postvaccination,autoimmune,systemic malignancy.
- Usually long segment cord involvement-extend for 3-4 spinal segments
- Lesion occupy more than two third of cross sectional area of spinal cord
- Variable contrast enhancement

MULTIPLE SCLEROSIS

- Plaques are usually multiple,shorter than two vertebral body segments in length and involves less than half the cross sectional area of spinal cord
- Additional lesions in brain
- Less prominent perilesional edema
- Less cord expansion may not demonstrate enhancement

NEUROMYELITIS OPTICA

- Also known as Devics disease is a severe demyelinating disorder.
- Characterised by bilateral optic nerve involvement –swollen,hyperintense on T2 WI with enhancement extending into optic chiasma
- Spinal cord involvement is extensive with high T2 SI extending three or more vertebral segments with involvement of central portion of the spinal cord with patchy cloud like enhancement

INTRA MEDULARY TUBERCULOMA

- The Magnetic resonance imaging findings in cases of spinal intramedullary tuberculoma can vary during the different phases of tuberculoma.
- In the early phase, the tuberculoma is characterized by severe infective reactions, poor formation of the gel capsule, and severe edema around the lesion. During this phase, T1WI and T2WI both show equal signal intensity and they are evenly enhanced after being intensified.
- As the gel content in the tuberculoma increases, the peripheral edema is alleviated or may disappear. As a result, T1WI shows equal signal intensity; meanwhile, T2WI shows equal or low signal intensity. After enhanced scanning, there is rim enhancement and low signal in the central region.
- With the development of caseation, T2WI shows a typical “target sign” which means that it exhibits a range from the low signal target to the high signal rim and also from the center of the low signal rim to the peripheral parts.

- The caseous substance forms the target center, whereas the peripheral infective granulation tissues form the high signal rim. The low signal rim in the external region is composed of collagen fibers produced by fibroblasts. Because the contents of collagen fibers vary, the low signal rim may be incomplete or absent.
- The “target sign” is a valuable indicator that helps differentiate spinal tuberculoma from other intramedullary lesions. Rim enhancement is usually observed in spinal tuberculosis. Compared with tumors, spinal tuberculoma has a sharper margin and lower T2WI signals, and it is particularly easy to differentiate the disease when there is a “target sign.”

MAGNETIC RESONANCE SPECTROSCOPY

Magnetic resonance spectroscopy (MRS) is an exciting application of magnetic resonance to noninvasively assess various metabolites or biochemicals from the body tissues. This metabolite information is then used to diagnose diseases, monitoring diseases and assessing response to the treatment. Even though theoretically MRS can be performed with spins or nuclei of 1H, 13C, 19F, 23Na and 31P, MRS in present clinical use are mainly 1H (Hydrogen) and 31P (Phosphorus) spectroscopy.

BASIC PRINCIPLES:

The basic principles of MRS are as same as magnetic resonance imaging. However, a few differences exist.

1. Usual Magnetic resonance imaging images are reconstructed from the signal from all protons in the tissue that is dominated by water and fat protons. The protons from other metabolites do not contribute to imaging because of their negligible concentration. Contrary to routine Magnetic resonance imaging, the aim in Magnetic resonance spectroscopy itself is to detect these small metabolites. Most metabolites of clinical interest have their signal resonate between resonant frequencies of water and fat.

   These small metabolites are detected only when the large signal from the water protons is suppressed.

2. **How are small metabolites from the tissue detected?**

Chemical shiftforms the basis of Magnetic resonance spectroscopy. The precessional frequency of protons is determined by the chemical environment or electron cloud surrounding it. A proton in the water will precess at a different frequency than the proton in fat and the same proton in another metabolite (for example NAA) will precess at a different frequency than in water and fat. This change in the precessional frequency of protons in different chemical environment is chemical shift. So, by determining the frequency of protons we can detect their chemical environment, i.e. metabolites in which they are precessing.

3. In homogenous field, Frequency of protons in a given metabolites = chemical shift = position of metabolite peak. Since the precessional frequency of any proton is directly proportional to the external magnetic field strength (Larmor frequency), chemical shift in Hz will be different at different magnetic field strength. To avoid this confusion, chemical shift is expressed in parts per million (ppm), which will be same for a particular metabolite at all the field strengths.
4. Since chemical shift is proportional to the external magnetic field, smaller chemical shift will not be detected at low field strength. Even though Magnetic resonance spectroscopy can be performed on 0.5 Tesla or above, field strength of 1.5tesla or above is required for improved spectral separation and increased Signal to noise ratio.

5. **Magnetic field homogeneity:**

   Magneticfield should be homogenous, i.e. of same strength throughout its entire extent for all Magnetic resonance applications. Magnetic resonance spectroscopy requires much more homogenous field than Magnetic resonance imaging because the smaller concentration of metabolites with smaller chemical shift needs to be detected. Since the chemical shift is proportional to the external magnetic field, smaller chemical shift will be misinterpreted and incorrect concentration will be recorded in an inhomogeneous field. The homogeneity required for Magnetic resonance imaging is about 0.5 ppm whereas for Magnetic resonance spectroscopy it is about 0.1 ppm. This process of making the magnetic field homogeneous is called as shimming.

6. No frequency encoding gradient in Magnetic resonance spectroscopy.

   Similar to MRI, localization in Magnetic resonance spectroscopy is done by slice selection and phase encoding gradients. However, the frequency encoding gradient is not used in MRS to preserve optimal homogeneity and chemical shift information.

   One more phenomenon needs to be discussed in Magnetic resonance spectroscopy, the spin-spin or J- coupling. Spins (protons) with a small difference of precessional frequency, for example spins within a molecule, interact with each other. This is facilitated by electrons around the nuclei.

This spin-spin interaction modifies the resonant frequency of the spins involved in it. J- Coupling causes fusion of peaks on spectral map, e.g. doublet of lactate at 1.3 ppm.

Localization of techniques in magnetic resonance spectroscopy:

In initial days, localization of the volume of interest was done by the surface coil. The area (volume) covered by the coil was the volume of interest from which metabolite information is obtained. In present clinical practice, four methods are commonly used for the localization of the volume of interest. They are STEAM, PRESS, ISIS and CSI (MRSI). STEAM, PRESS and ISIS are used for single voxel spectroscopy (SVS). CSI is a multivoxel (MVS) technique.

STEAM: Stimulated Echo Acquisition Method

The volume of interest is excited by three 90 degree pulses in three orthogonal planes. Since the echo is stimulated signal is weak. STEAM is used for short TE (20 –30ms) spectroscopy.

PRESS: Point Resolved Spectroscopy

In PRESS, one 90 degree and two 180 degree pulses are applied along three orthogonal planes. The signal is strong with better Signal to noise ratio. Hence PRESS is used for longer TE (135, 270 ms) spectroscopy. PRESS cannot be used for shorter TEs.

ISIS: Image Selected In vivo Spectroscopy

Three frequency selective inversion pulses are applied in presence of the orthogonal gradients. Fourth non- selective pulse is used for the observation of signal. ISIS is used in the 31P spectroscopy.

CSI: Chemical Shift Imaging

CSI is used for multivoxel spectroscopy, where a large area is covered and divided into multiple voxels. CSI is also called as Magnetic Resonance Spectroscopic Imaging (MRSI) as it combines features of both imaging and spectroscopy. Spatial localization is done by phase encoding in one, two or three directions to get one, two or three dimensional spectroscopy respectively. Metabolite maps or metabolite ratio maps can be seen overlaid over the images.

Steps in MRS Acquisition:

1. Patient positioning.
2. Global shimming.

   Optimization of magnetic field homogeneity is done over the entire volume detected by the receiver coil. Global shimming provides starting value for local shimming.
3. Acquisition of MR images for localization.

   Images are obtained in all three planes (coronal, axial and sagittal) for placement of a voxel. In routine practice, MR images already obtained during routine imaging are used for localization purpose if patient is not moved.

4. Selection of MRS measurement and parameters.

TR and TE are important parameters. Improved SNR are obtained at a longer TR. Commonly used TEs are 20 – 30 ms, 135- 145 ms and 270 ms. At TEs longer than 135 ms, only peaks of major metabolite like choline, Creatine, NAA and lactate are visible. The lipid, glutamate, glutamine, GABA and inositol peaks are suppressed at higher TEs because of their short T2. Shorter TEs are used for detection of these metabolites. There is less noise at higher TEs.

5.Selection of VOI (Volume of Interest)

SVS can be used for local or diffuse diseases. CSI is used in irregularly shaped large lesion and where comparison on both sides is required.

6.Local shimming

This is optimization of homogeneity over the selected volume of interest. Good local shim results into narrower metabolite peaks, better spectral resolution and good SNR. Full width at half height of the water peak is used as an indicator of shim. A local shim of 4-10 Hz is desirable.

7.Water suppression

The water peak is suppressed so that smaller metabolite peaks are visible. Water peak suppression is done by CHESS (Chemical Shift Selective Spectroscopy) technique.

8.MRS data collection.On modern machines in use, single voxel spectroscopy usually takes 3 – 6 minutes and CSI usually takes upto 12 minutes for data acquisition.

9.Data processing and display

Acquired data is processed to get spectrum and spectral maps. Zero point of spectrum is set in the software itself by frequency of a particular compound called Tetramethylsilane (TMS).

10.Interpretation

The area under the peak of any metabolite is directly proportional to the number of spins contributing to the peak. Absolute values for each metabolite may vary with age and population. Interpretation should always be based on ratios of metabolites and comparison with normal side.

Metabolites of 1H(PROTON) MRS

1. NAA: N- Acetyl aspartate

   Peak position: 2.02 ppm

   There is some contribution from NAAG and glutamate to the NAA peak.

   It is a neuronal marker and any insult causing neuronal loss or degeneration causes reduction in NAA. It is absent in tissues/lesions with no neurons e.g. metastasis and meningioma.

NAA is reduced in: hypoxia, infarction, Alzheimer's, herpes encephalitis, hydrocephalus, Alexander disease, epilepsy, some neoplasms, stroke, NPH, closed head trauma (diffuse axonal injury).

NAA increased in: Canavan's disease.

Table :3 major metabolites and its peak position

Metabolites	Peak position in ppm	Approx. concentration in mmol/kg in the white matter
NAA	2.02	10 – 15
Creatine	3.0	8
Choline	3.2	1.5
Myo –inositol	3.56	5

2. **Cr :** Creatine

Peak position**:** 3.0ppm. Second peak at 3.94ppm.

The Cr peak contains contribution from Creatine, CrPO4, GABA, Lysine and Glutathione.

Cr serves as high energy phosphate and as a buffer in ATP/ADP reservoir. It increases in amount with age.

Cr is increased in: hypo metabolic states and in trauma.

Cr is reduced in: hyper metabolic states, hypoxia, stroke and some tumor.

Cr remains stable in many diseases hence serves as reference or control peak for the comparison.

3. **Cho:** Choline

   Peak position: 3.22 ppm

   Choline is a constituent of phospholipids of the cell membrane. It is a precursor of acetyl choline and phosphatidylcholine. Choline is an indicator of cell membrane integrity. Cho increases with increased cell membrane synthesis and increased cell turnover.

   Cho increased in: chronic hypoxia, epilepsy, Alzheimer's disease, gliomas and some other tumors, trauma, infarction, hyperosmolar states, diabetes mellitus.

   Cho is reduced in: hepatic encephalopathy and stroke.

4. **MI:** Myo – inositol

   Peak position: 3.56 ppm. Second peak: 4.1 ppm.

   mI acts as an osmolyte and is a marker of gliosis. It is involved in hormone sensitive neuroreception and is precursor of glucuronic acid. It is the dominant peak in new born babies and decreases with age.

   mI is increased in: Alzheimer's disease, frontal lobe dementias, diabetes and hyperosmolar states.

   mI is decreased in: hepatic and hypoxic encephalopathy, stroke, tumor, osmotic pontine myelinolysis and hyponatremia.

5. **Lac :** Lactate

   Peak position: 1.3 ppm

It is a doublet. It is inverted at TE of 135 ms with PRESS, upright at other TEs on PRESS and at all the TEs with STEAM sequences. It is not seen in the normal brain spectrum.

It can be elevated in hypoxia, tumor, mitochondrial encephalopathy, intracranial haemorrhage, stroke, hypoventilation, Canavan's disease, Alexander's disease and hydrocephalus.

6.**Glx :** Glutamate (Glu) and Glutamine (Gln)

Peak position: 2 – 2.45 ppm for beta and gamma Glx. Second peak of alpha Glx at3.6 – 3.8 ppm.

Glu is an excitatory neurotransmitter and GABA is a product of Glu.

Gln has role in detoxification and regulation of neurotransmitter activity.

Glx peak is elevated in head injury, hepatic encephalopathy and hypoxia.

7.Lipids

Peak position: 0.9, 1.3, 1.5 ppm.

Not seen in normal brain spectrum.

Seen in acute destruction of myelin.

Increased in high grade tumors (reflects necrosis), stroke and multiple sclerosis lesions.

8.Amino acids :

Alanine (at 1.3 – 1.4 ppm**), Valine (**at 0.9 ppm**), Leucine (**at 3.6 ppm**)** are usually multiplets visualized at short TE. They invert at TE of 135 ms.

Alanine is seen in the meningioma whereas Valine and Leucine are markers of an abscess.

9.**Glucose:**

When present, it is seen next to Cho peak on its left side. It may increase in diabetes, parenteral feeding and hepatic encephalopathy.

10.GABA:

Peak position: 1.9 and 2.3 ppm

Used for monitoring vigabatrin therapy given in children with myoclonic jerks.

CLINICAL USES OF MAGNETIC RESONANCE SPECTROSCOPY:

1H (Proton) MRS has its role in almost every neurological condition. Role of MRS in a few common conditions is discussed below.

1. Tumors:

   There is increase in Choline, lactate and lipid in tumors. There is reduction in N-acetyl aspartate and Creatine in tumors.

a.**MRS in tumor evaluation** –

MRS can differentiate neoplasm from non-neoplastic lesions. MRS also helps to grade gliomas based on metabolite ratios.

b. **In treatment planning**

MRS guides the biopsy. Biopsy of higher choline area has been shown to have higher success and increased diagnostic confidence. Inclusion of the peritumoral area with increased Cho in the radiation field improves survival.

c.**In treatment monitoring**

MRS helps to differentiate radiation necrosis and gliosis from the residual or recurrent neoplasm. Patient with radiation necrosis will have reduced peaks of all metabolites whereas recurrent or residual tumor will have characteristic spectrum of tumor with elevated choline.

2. Multiple sclerosis

In MS plaques, there is decrease in NAA/Cr and increased Cho/Cr and mI/Cr. Active plaque shows elevated lipid, lactate, Cho/Cr ratio and mI. Progression can be monitored by NAA/Cr ratio.

3.**Abscess**

Abscess can be difficult to differentiate from neoplasm. The changes in MR spectra in abscess include visualization of amino acid peaks at 0.9 ppm. These amino acids include Valine, Leucine and isoleucine. The abscess may show peaks representing acetate, pyruvate, lactate and succinate, which are end products arising from some microorganisms[9].

REVIEW OF LITERATURE

A review of the literature revealed several publications using H-MR spectroscopy in clinical studies in which different pathologies were investigated.

"Five investigations focused on multiple sclerosis and reported a general reduction in the NAA/Cr ratio &increase in Cho/Cr and myoinositol/Cr in the spinal cord of patients with MS[10]"- Marliani et al.

Kim et al "investigated 14 mass lesions. They reported specific MR spectroscopy changes, similar to changes seen in MR spectroscopy of central nervous system tumors[2]." they didnt show any quantification or metabolite ratio and quality evaluation of their results.

Dydak et al investigated one patient with a malignant lesion in the cervical spinal cord, they showed that "lactate and myo-inositol are increased compared with healthy spinal cord tissue by visual inspection of the spectrum[11]" but they did not provide metabolite concentrations and quality indicators.

Henning et al" reported two patients with spinal cord tumor (one located at C4–5 and one at T9) with sufficient shim of12 Hz (FWHM of the water peak) at 3T.Besides a decrease of NAA and Cr, an increase of Cho and myo-

inositol was observed. In addition, they reported an increased lactate and glutamine/glutamate peak in the tumor tissue[12]"

Hock et al "presented 3 patients with neoplastic spinal cord lesions and compared the spectra with those from 13 healthy volunteers and 13 patients with MS .Spectra were of good quality,. However just acquisitions with short TEs were acquired. The measured ependymoma showed strongly reduced NAA/Cr, increased Cho/Cr, and strongly increased myo-inositol/Cr in addition to lipids and lactate compared to metabolite over Cr ratios measured in healthy controls[1]"

Holly et al "investigated 21 patients with cervical spondylotic myelopathy and compared the results with those in 13 healthy volunteer and identified the presence of lactate in this patient group[13]".

Rapalino et al investigated "cervical spondylosis in 8 patients and compared the results with those in 6 healthy volunteers. They noticed a decrease of the NAA/Cr ratio and an increase of Cho/NAA in patients, without presenting quality indicators apart from 2 exemplary spectra with relatively low SNR[14]"

Carew et al and Pineda-Alonso et al "investigate amyotrophic lateral sclerosis in 14 patients and 16 controls, show that NAA/Cr and NAA/myo-inositol were reduced in patients with amyotrophic lateral sclerosis, and the reduction correlated significantly with clinical parameters[15]"

De Vita et al “ presented data from 14 healthy volunteers and 8 patients after brachial plexus root re-implantation. They found a significant increase of the myo-inositol/Cr ratios in the patient data sets[16]”.

Hock et al also “presented data of patients with traumatic injuries A decrease in NAA/Cr and an increase of myo-inositol/Cr were observed, the spectral quality was reduced in these patients in comparison with healthy volunteers and patients with MS, most likely due to implants and the presence of hemorrhage[1]”

All published studies report specific changes when comparing patients with healthy volunteers, indicating the feasibility and the potential of applying MR spectroscopy to the spinal cord. Especially, NAA and myo-inositol seem to be meaningful markers for the investigation of various disease.

AIM OF THE STUDY

The purpose of the study is to determine the role of 3 TESLA MR SPECTROSOPY in evaluation of patients with INTRAMEDULLARY spinal cord lesions to enable reliable metabolite concentration and to correlate with its histopathological examination.

STUDY CENTRE:

BARNARD INSTITUTE OF RADIOLOGY,

MADRAS MEDICAL COLLEGE,

RAJIV GANDHI GOVT GENERAL HOSPITAL,CHENNAI-3.

- NUMBER OF PATIENTS:50
- NUMBER OF CONROLS:15
- DURATION OF STUDY:ONE YEAR (from may 2015-may 2016)
- STUDY DESIGN:Prospective study

- The study was done after getting approval from Madras Medical College ethics committee.
- The study was done only after getting written consent from the patients or control .

INCLUSION CRITERIA

1.patients with intramedullary spinal cord lesions

EXCLUSION CRITERIA:

1.Recurrent spinal lesions

2.Previously operated spinal lesions

3.Patients with contraindications for MR(MR incompatible pacemaker,cochlear implant)

4.Nonconsenting&uncooperative patients

METHODOLOGY:(materials and methods)

All patients with intramedullary spinal lesions admitted at madras medical college and RGGGH who is willing for MRI are included in this study during the period from may 2015 to may 2016 .

Clinical history and physical examination were obtained by the evaluating neurosurgeon which included back pain, upper or lower limb weakness,paraparesis.

Standard MRI SPINE and MR SPECTROSCOPY were done on 3TESLA MRI scanner.All Magnetic resonance Experiments were performed on a SIEMENS skyra 3T scanner using the integerated body coil for transmission and a sense neurovascular coil for reception.

Standard MRI of the spine was taken in T1 and T2 sagittal ,T2axial, post contrast fat suppressed T1sagittal images.Location of the lesion was identified and the charater of the lesion like solid/cystic,post contrast enhancement characters,any associated syrinx ,cord expansion were noted.

Single voxel Magnetic resonance Spectroscopy was applied either in the T2 sagittal or post contrast T1 sagittal fat suppressed image.If the patient motion is identified voxel position is updated and the measurement is repeated. size of the voxel adjusted according to the size of the lesion .pulse gating was applied to reduce the pulsation artifact. TE was set at 135 and TR at 2000.

Inner volume suppression bands were applied to minimize the chemical shift displacement artifact and to reduce the influence of cerebrospinal fluid Slice thickness was 3mm. This sequence lasts about 5minutes.

Magnetic resonance spectroscopy DATA were acquired. post processing done to obtain good spectrum of metabolites.integeral values of the metabolites in each intramedullary spinal cord lesions were obtained.These patients were followed for their post operative tissue biopsy and Histopathological examination results to compare the MRS findings.

MR Spectroscopy was also done in normal subjects at cervical and dorsal level for comparison and to detect the metabolite peaks.

SPINAL MRS PROTOCOL

MRI SPINE	**TR**	**2000ms**
T1 SAGITTAL	**TE**	**135**
T2 SAGGITAL	**FLIP ANGLE**	**90degrees**
T2 AXIAL	**SNR**	**1**
T1 POST CONTRAST	**VECTOR SIZE** **BAND WIDTH**	**1024** **1200HZ**

CONTROL

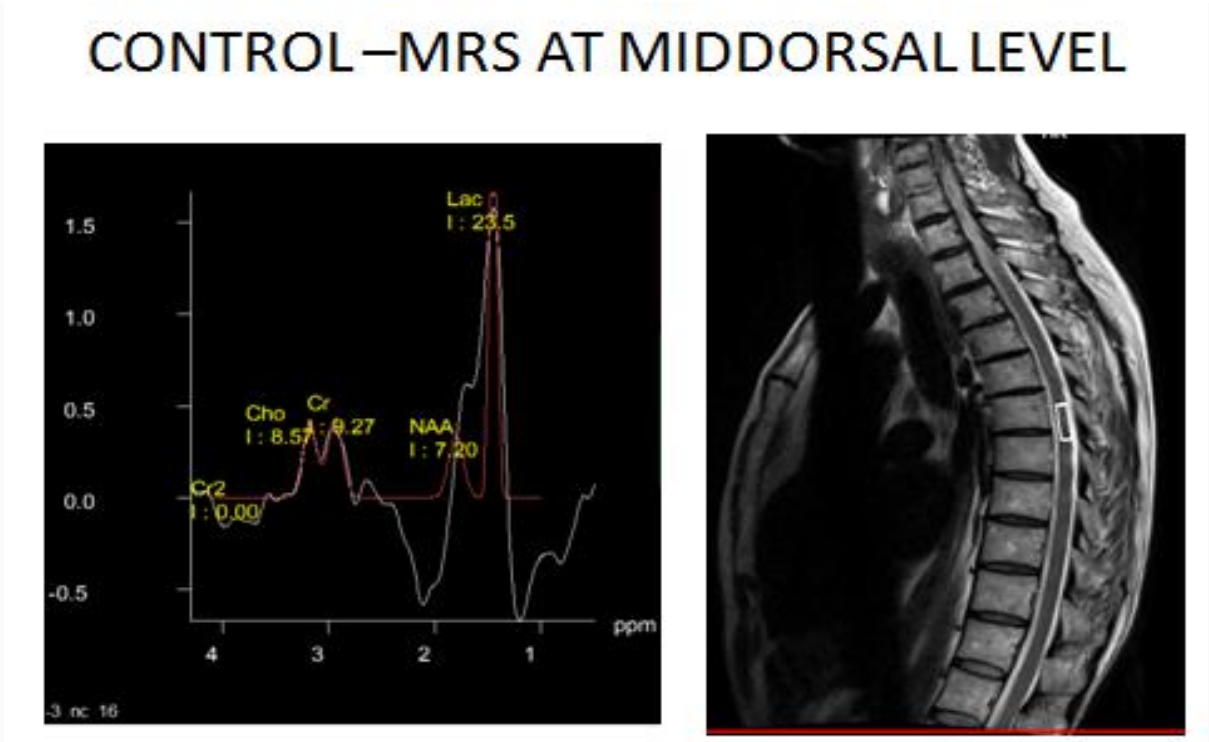


FIG:10 Single voxel MRspectroscopy at the mid dorsal cord level shows normal concentration of lactate,NAA,creatine with reduced choline .

CONTROL MRS AT CERVICAL LEVEL

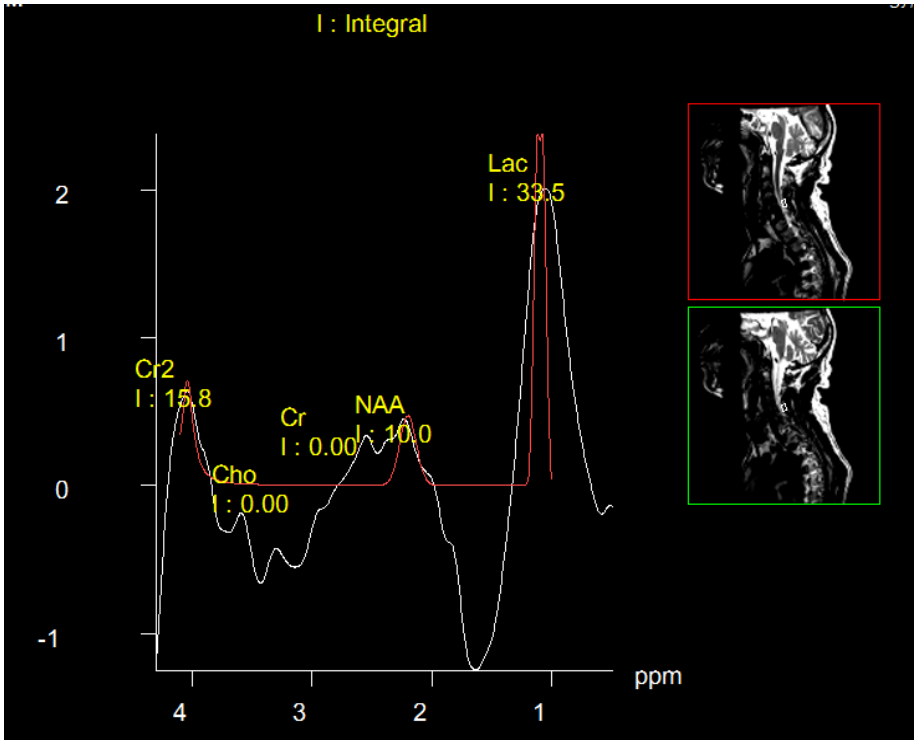


FIG:11 Single voxel MRspectroscopy at the cervical cord level shows mild increase in the lactate concentration probably due to contamination with no detectable choline .

CONTROL MRS AT CERVICAL CORD LEVEL

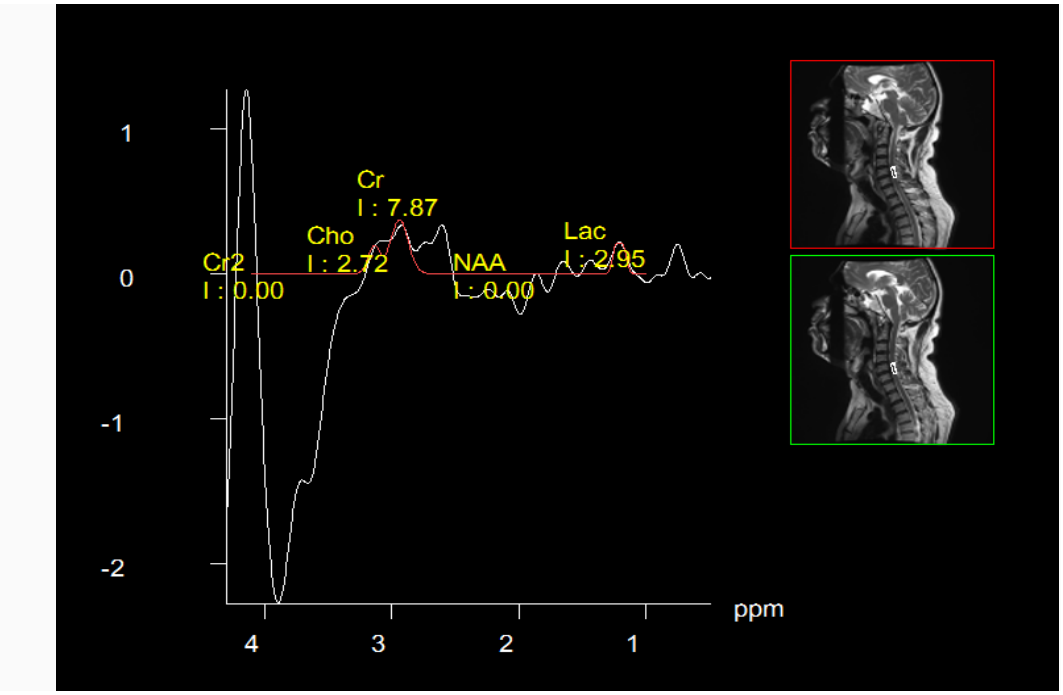


FIG:12 Single voxel MR spectroscopy at the cervical cord level in another patient shows no significant increase in any specific metabolite .

CASE 1

- 30 years old male pt came with the c/o of numbness involving all four limbs.

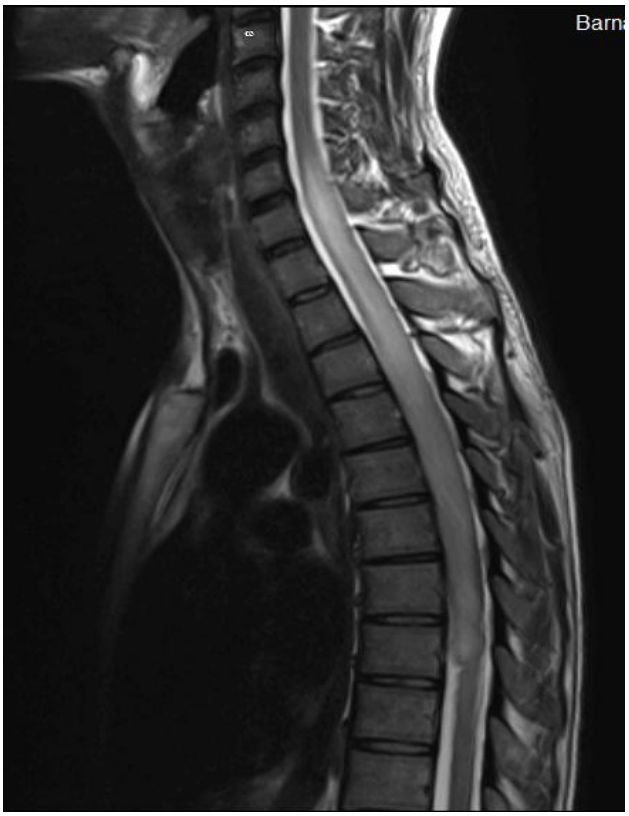


FIG:12 T2 WEIGHTED MRI shows – LONG SEGMENT INTRAMEDULLARY HYPERINTENSITY FROM C6-D8 LEVEL WITH MILD CORD EXPANSION.

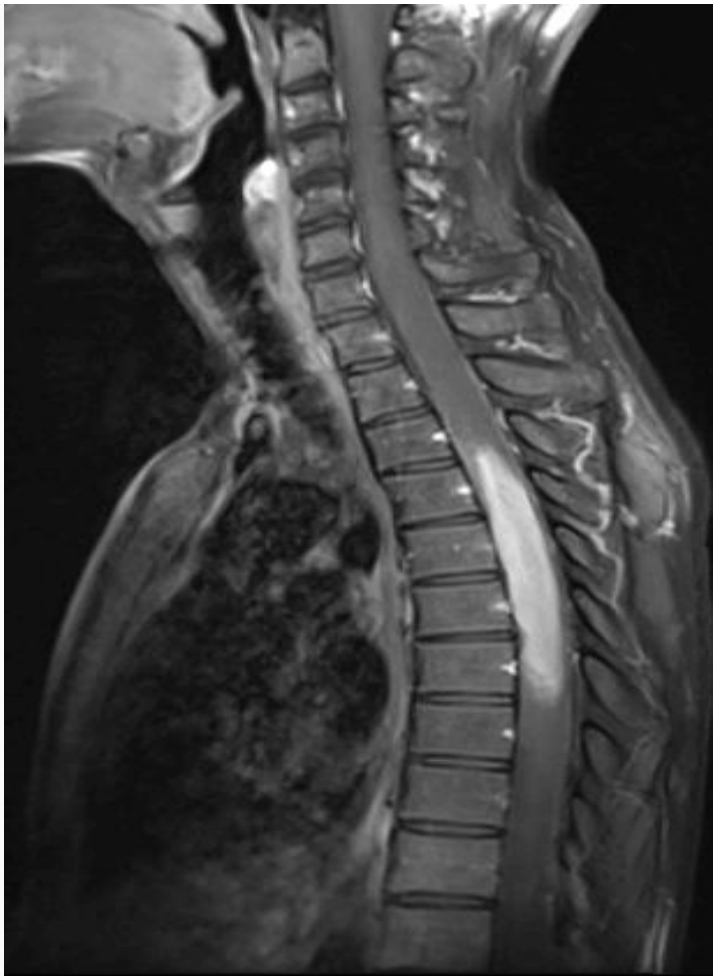


FIG:13 T1 FAT SAT POST CONTRAST image shows intense homogenous enhancement from D4 to D8 level.

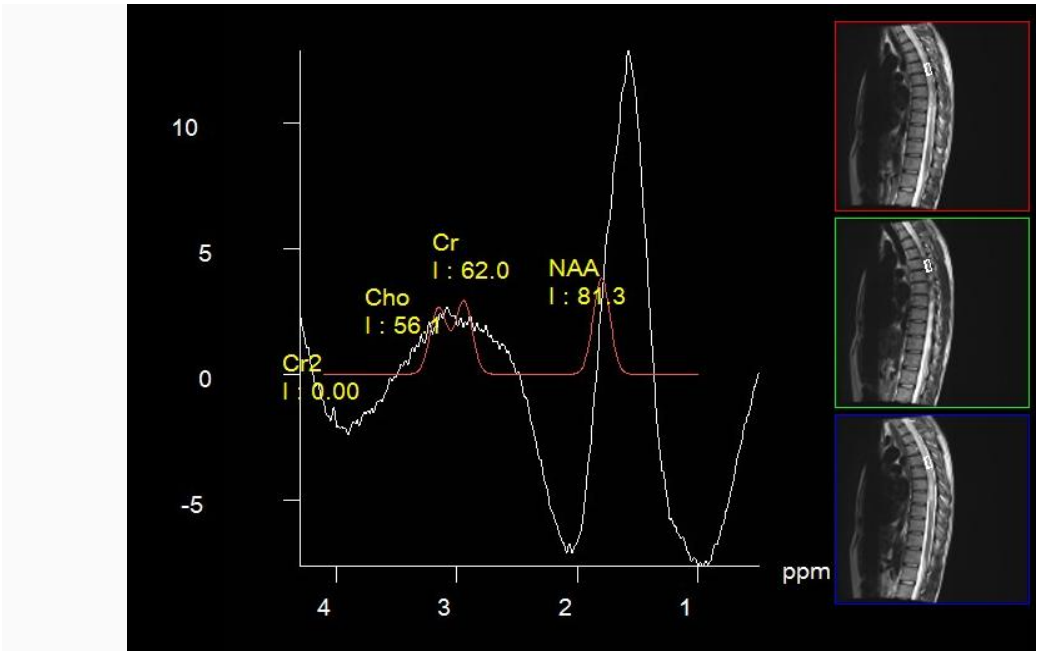
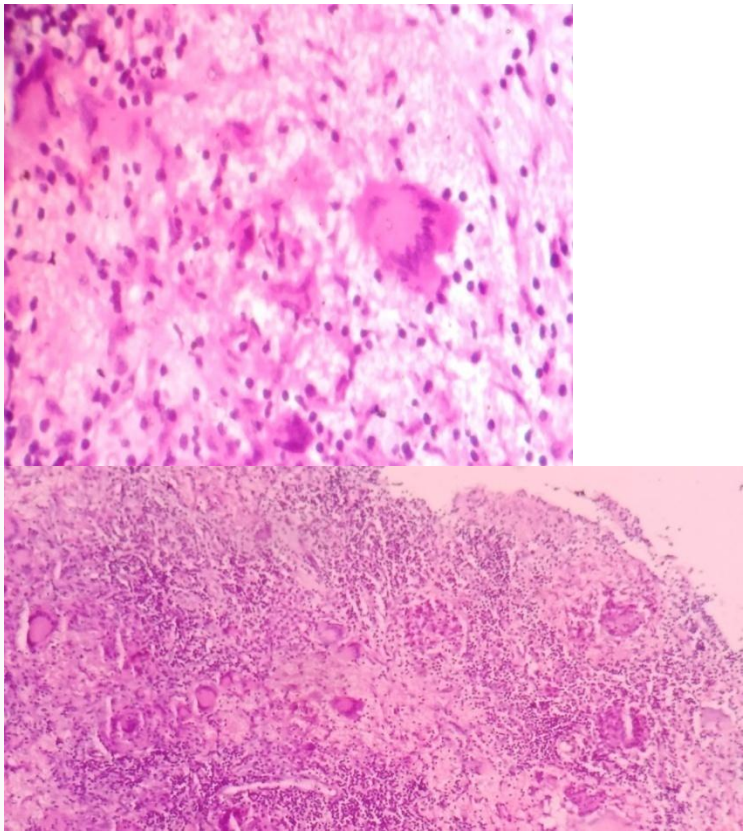


FIG:14 MR SPECTROSCOPY SHOWS reduced NAA and creatine with increased lactatate peak at 1.3 ppm

DIAGNOSIS:INTRAMEDULLARY TUBERCULOMA

HPE:



- **FIG:15 HPE Showed - extensive areas of caseation necrosis with scattered epitheloid histocytes,lymphocytes,plasma cells and few neutrophils suggestive of tuberculous etiology.**

CASE 2

- 20 yrs Old female patient presented with the c/o gradual onset of quadriparesis
- No h/o fever

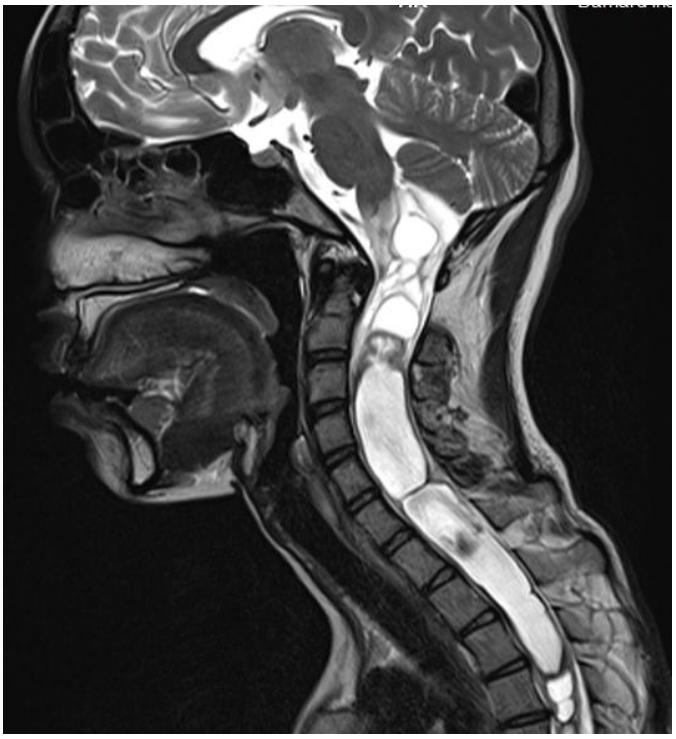


FIG:16 T2w saggital image shows heterointense solid lesion with hyperintense cystic areas causing cord expansion occupying the medulla,and cervico dorsal region.

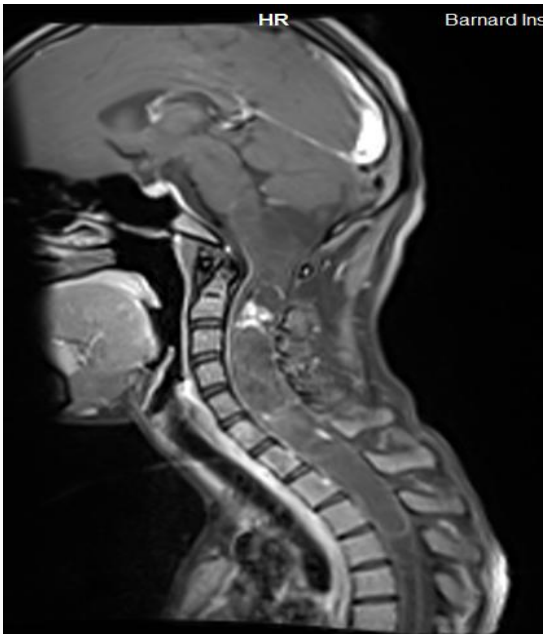


Fig:17 T1-postcontrast-heterogenouslyenhancing intramedullary lesion at cervicomedullary junction

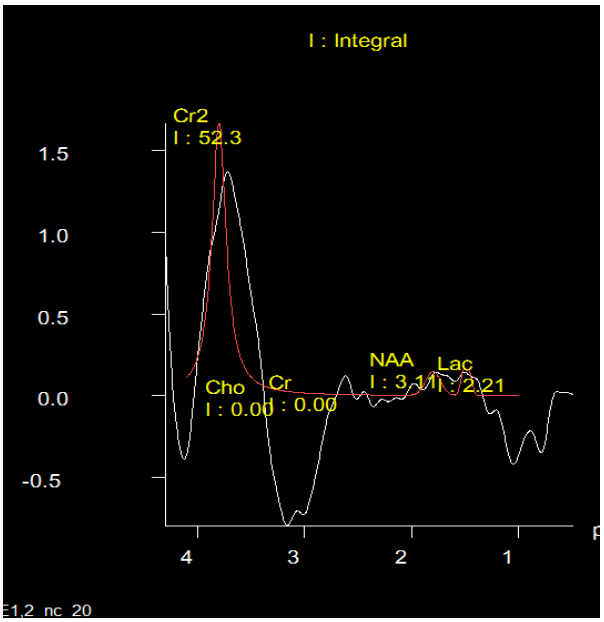
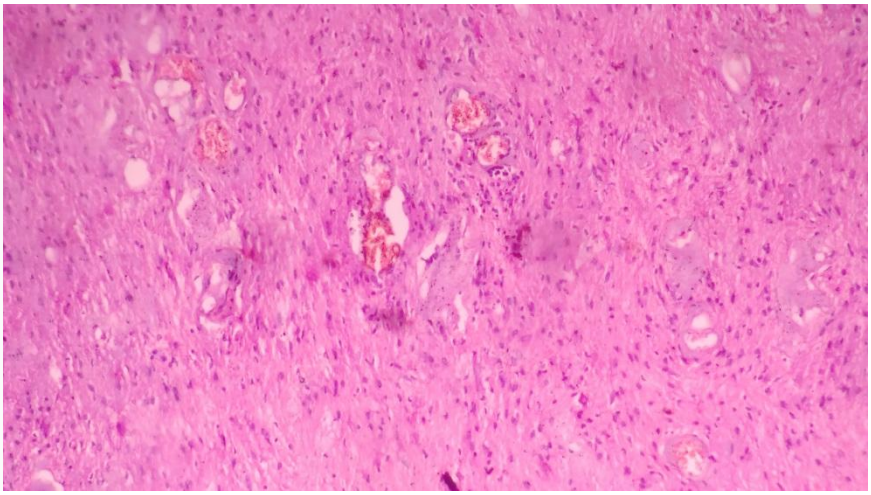
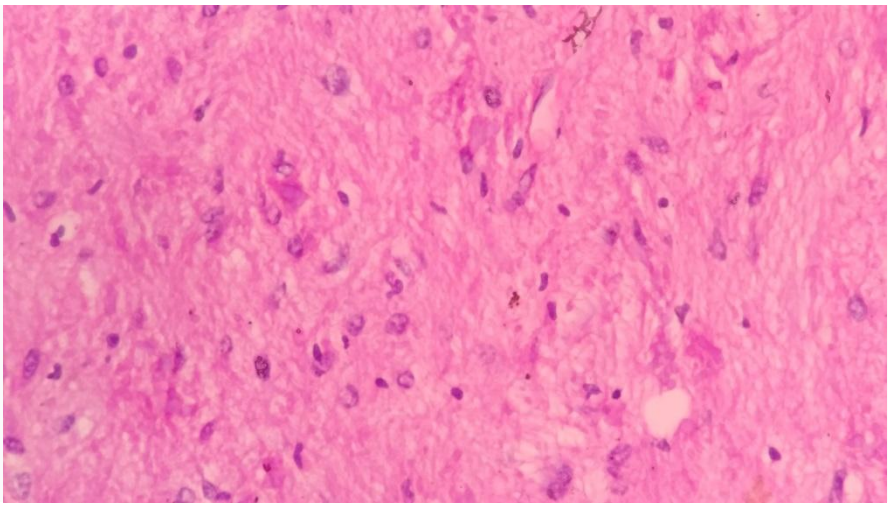


FIG:18 MR spectroscopy- NAA decreased, lactate not very much increased and creatine increased.DIAGNOSIS: EPENDYMOMA

HPE





HPE:Fig 19 Biphasic neoplasm composed of looser (and cystic) areas with protoplasmic astrocytes and densely cellular areas composed of hair-like (piloid) cells, Eosinophilic granular bodies, Rosenthal fibers and Oligodendroglial-like cells -LOW GRADE GLIOMA.

CASE 3

30yrs old male patient presented with progressive weakness involving both upper limb.

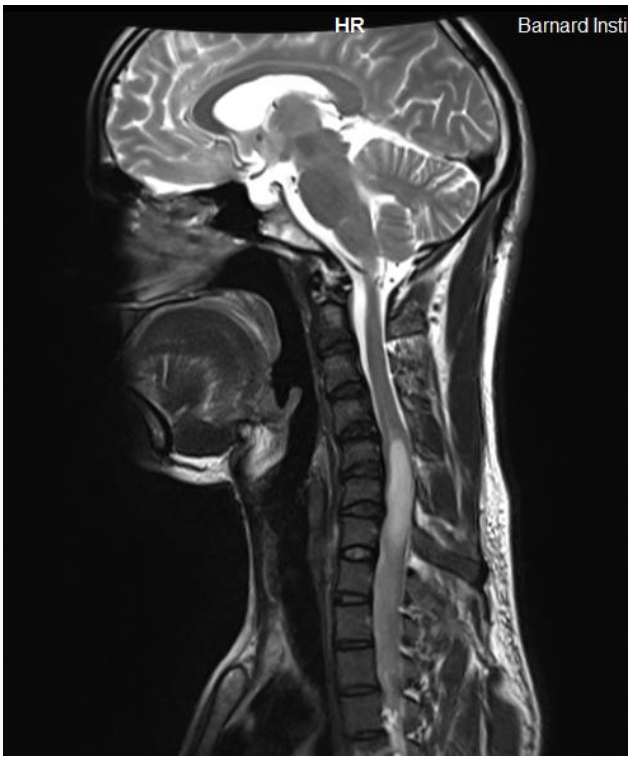


FIG:20 T2weighted MRI shows intramedullary T2 hyperintensity with cordexpansion extending from C5-C7 level .

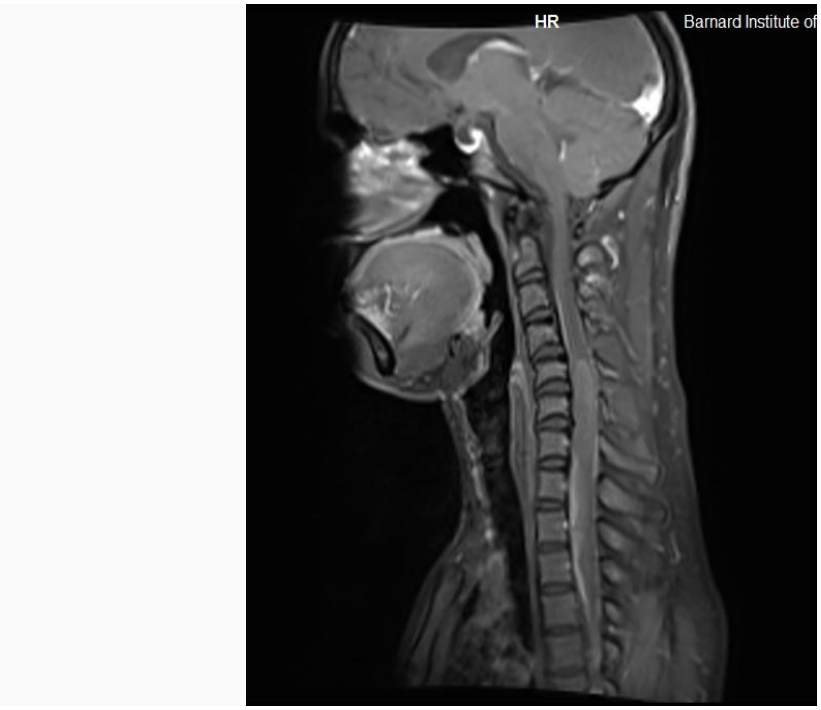


FIG:21T1 post contrast image show subtle enhancement within the lesion.

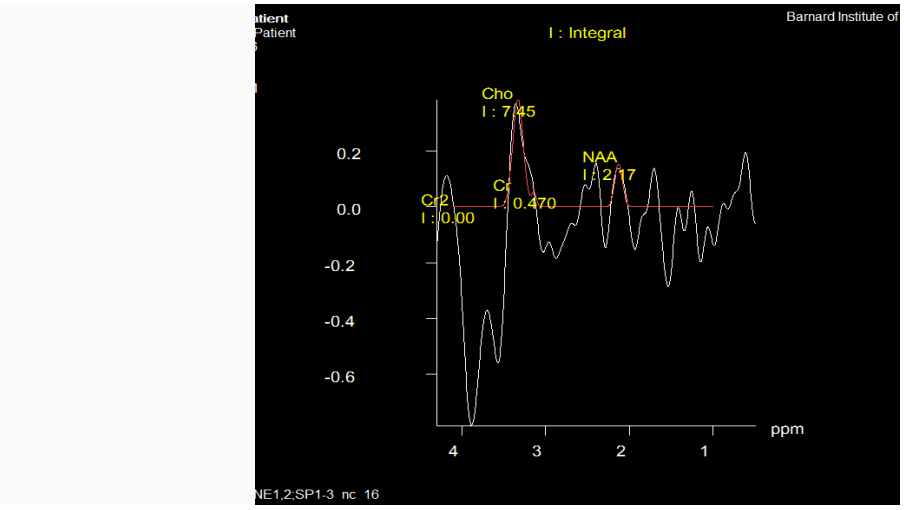


FIG:22 MR spectroscopy- Choline increased, creatine almost zero, NAA decreased .

DIAGNOSIS:GLIOMA PROBABLY ASTROCYTOMA

HPE:

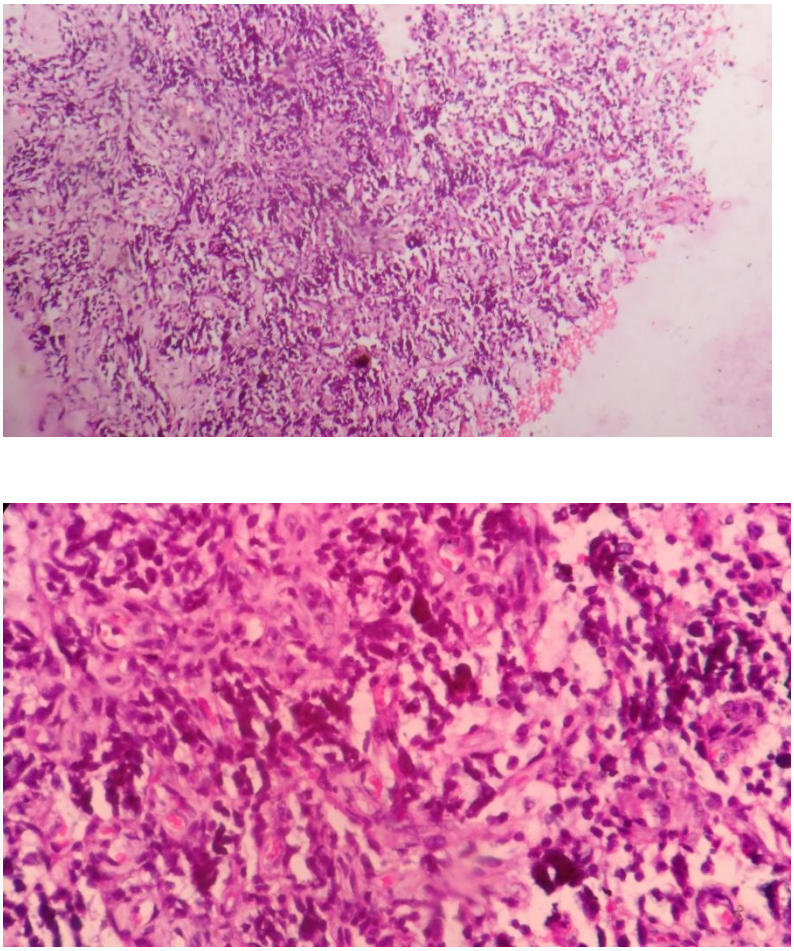


FIG:23 HPE- Increased cellularity and mitotic figures with microvascular proliferation and pseudopalisading necrosis-GBM WHO GRADE 4.

CASE 4

42 yrs old female patient admitted with the complaints of back pain associated with weakness of both lower limbs.

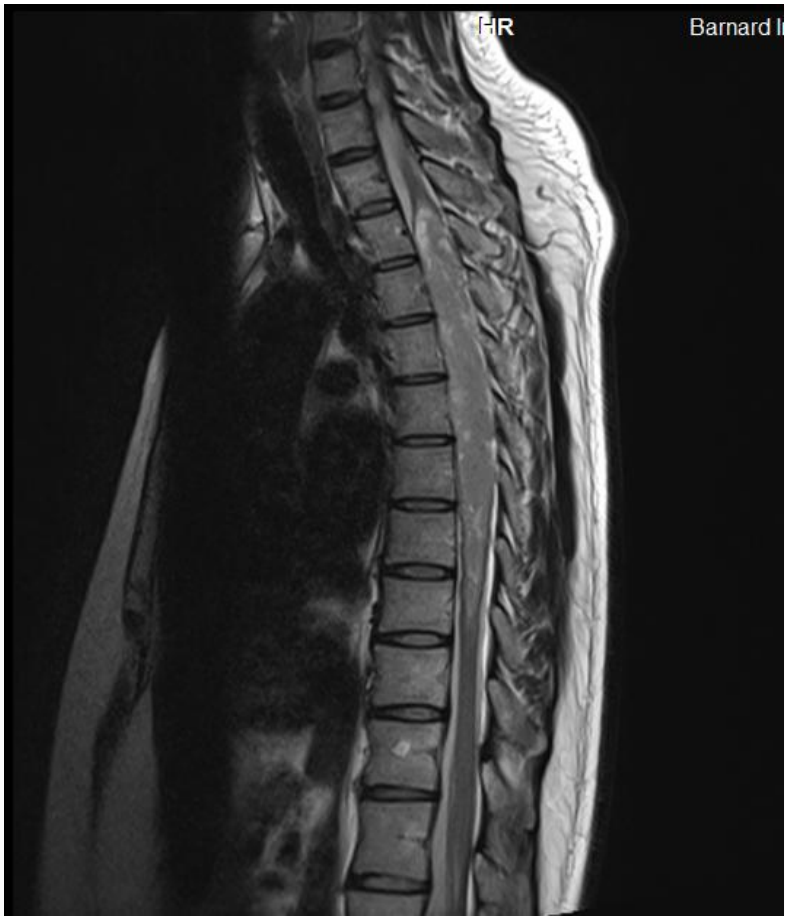


FIG:24 T2WI shows heterointense intramedullary expansile lesion from T4 to T11 level.

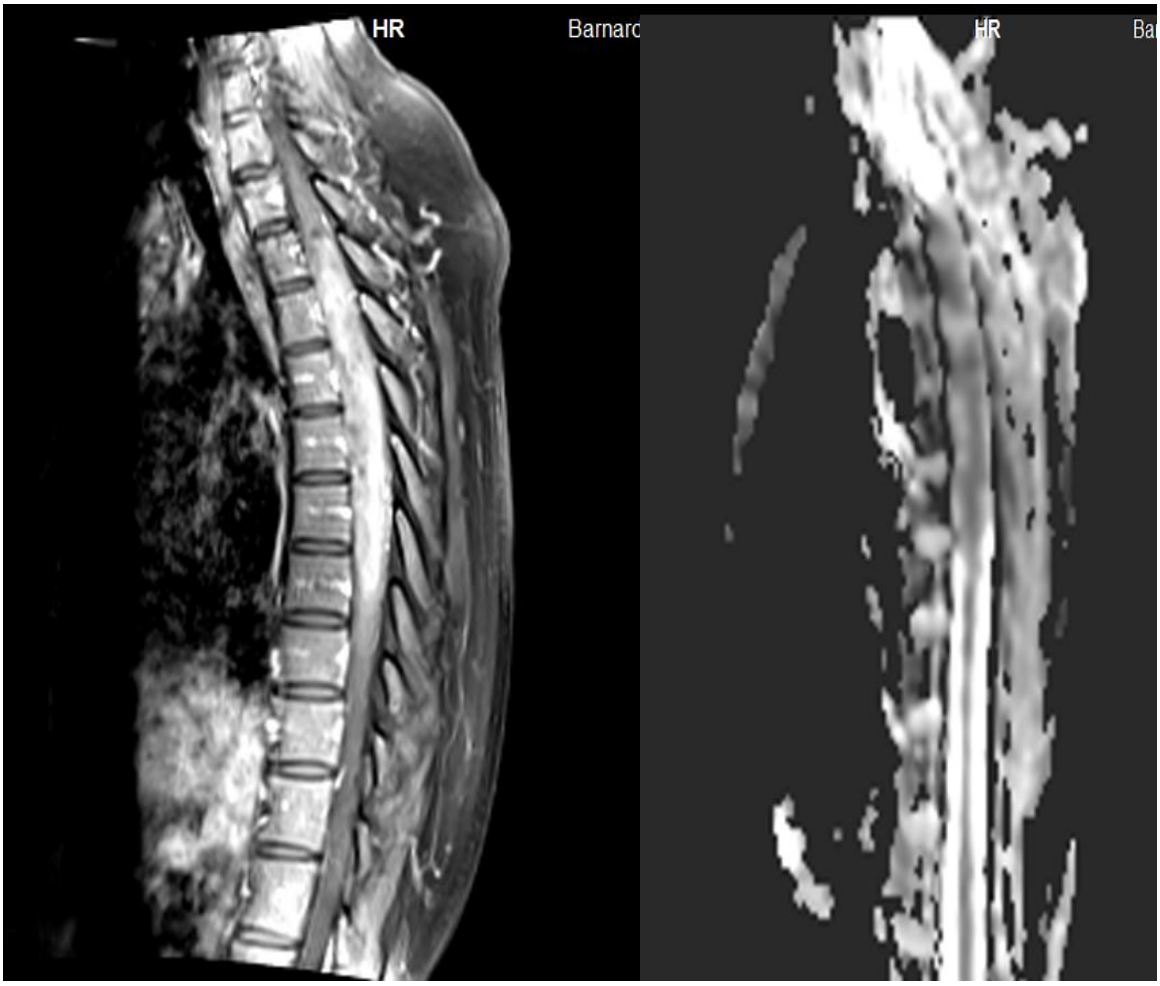


FIG25 :DWI-Lesion shows diffusion restriction.

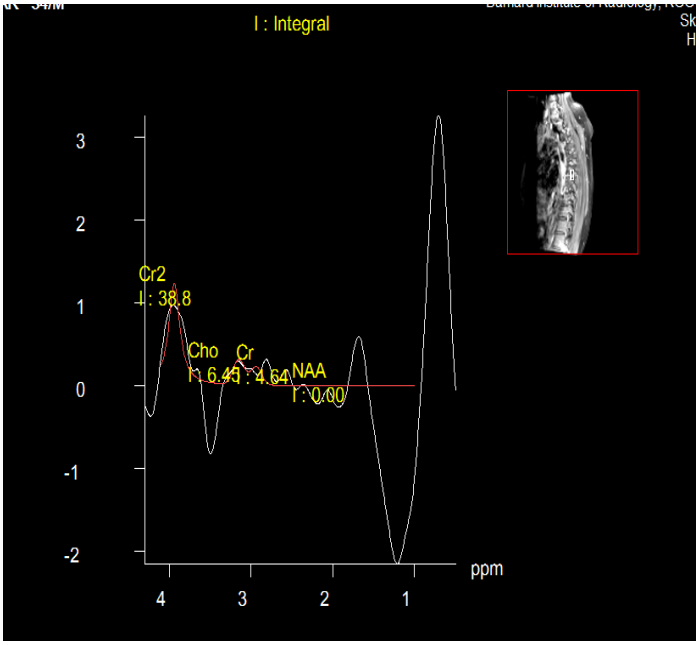


FIG:26 MRS Shows elevated lipid lactate level.

DIAGNOSIS:INTRAMEDULLARY TUBERCULOMA

HPE:

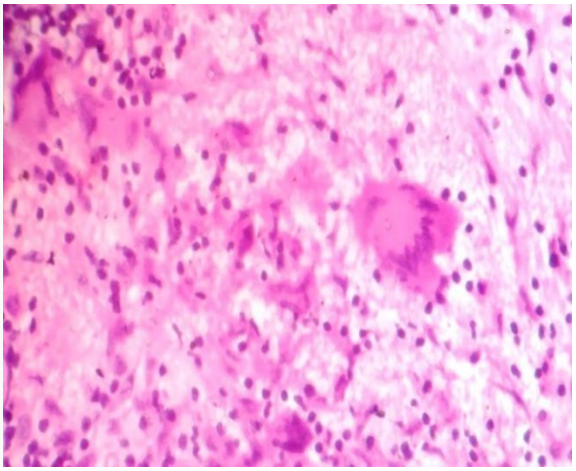


FIG:27 HPE- Extensive areas of caseation necrosis with scattered epitheloid histocytes,lymphocytes,plasma cells and few neutrophils suggestive of tuberculous etiology.

STATISICAL ANALYSIS

- The collected data was analysed with SPSS 16.0 version.

- To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and for continuous variables the mean and StandardDeviation were used.

- To find the significance between conventional Magnetic resonance imaging & Magnetic resonance spectroscopy the McNemar test was used.

- In the above statistical tool the probability value .05 is considered as significant level.

SEX DISTRIBUTION

FREQUENCIES:

sex

	Frequency	percent	Valid percent
female	**21**	**42**	**42**
male	**29**	**58**	**58**
total	**50**	**100**	**100**

Table 4: shows the sex distribution of experimental group.

The table shows the predominance of male patients in the study group.

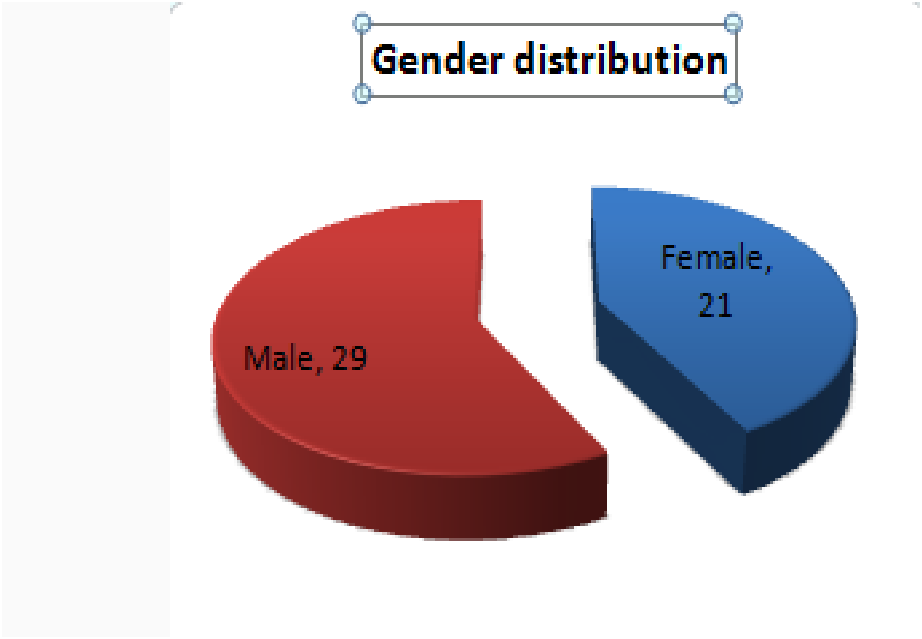


Fig:28 PIE CHART shows graphic representation of the percentage of male & female patient in the study group

AGE DISTRIBUTION

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	50	7	53	33.28	9.424
Valid N (listwise)	50				

Table 5: shows the AGE distribution of experimental group,mean age in the study group is 33 years

DISTRIBUTION OF INTRAMEDULLARY LESIONS

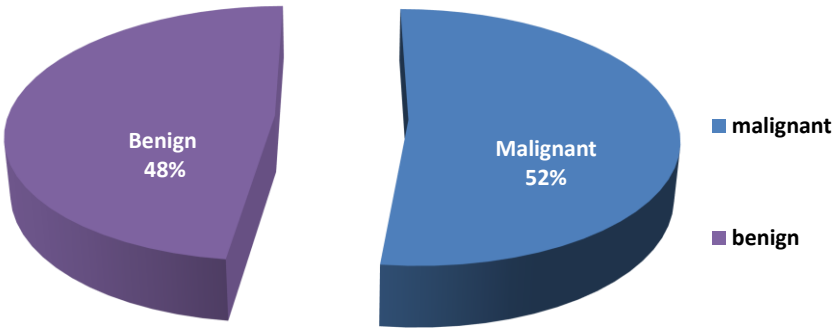


FIG:29 pie chart represents percentage of benign and malignant lesions among the study group.slight predominance of malignant lesions was observed.

FREQUENCY DISTRIBUTION OF IM LESIONS

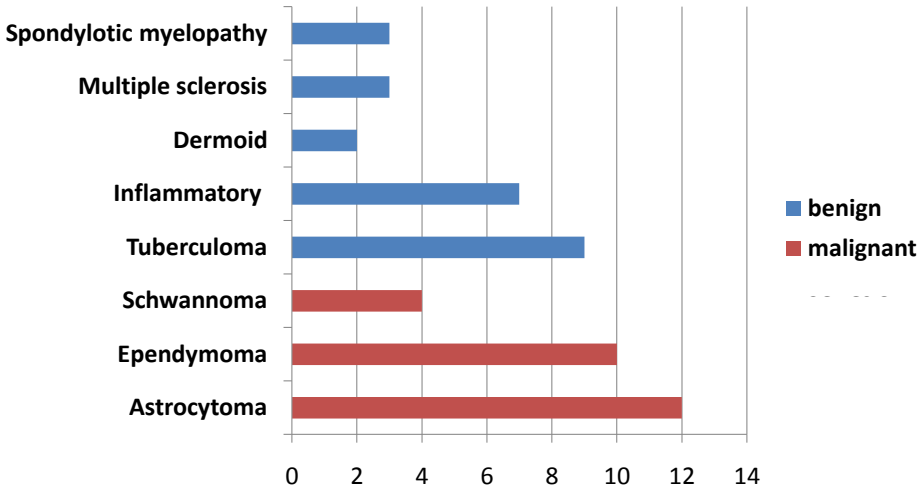


FIG:30 bar diagram represents the frequency distribution of various benign and malignant lesions among the study group.

FREQUENCYDISRIBUTION OF INDIVIDUAL MALIGNANTLESION

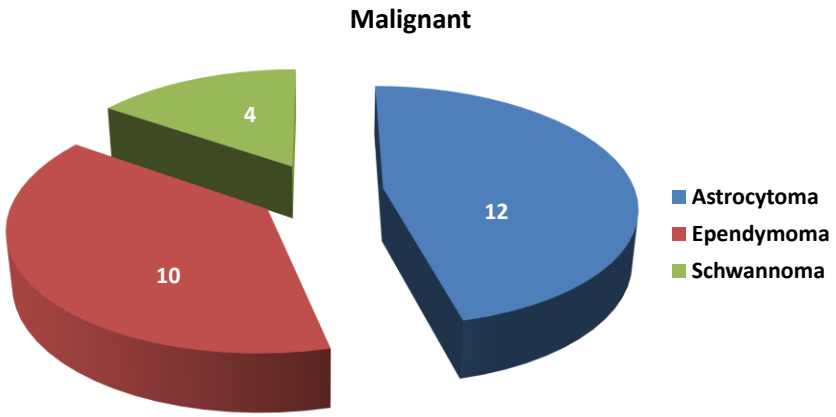


Fig:31pie chart represents the percentage distribution of individual malignant lesions among the study group

FREQUENCY DISTRIBUTION OF INDIVIDUAL BENIGN LESION

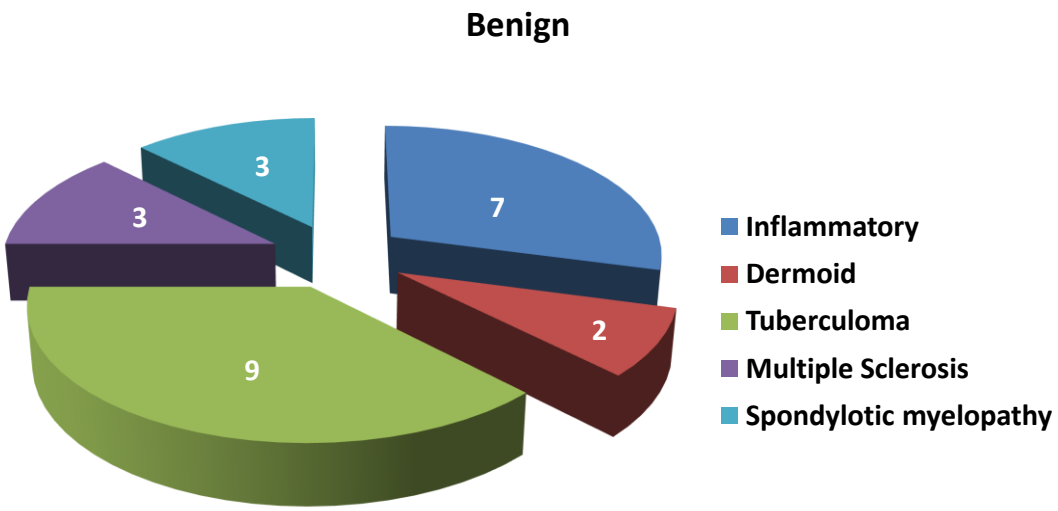


Fig:32pie chart represents the the percentage distribution of individual benign lesions among the study group.

FREQUENCY TABLE

CONVENTIONAL

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	29	58.0	58.0	58.0
	No	21	42.0	42.0	100.0
	Total	50	100.0	100.0	

MRS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Yes	42	84.0	84.0	84.0
	No	8	16.0	16.0	100.0
	Total	50	100.0	100.0	

Table6: shows that applying conventional imaging alone no of cases detected correctly was 58%.when Magnetic resonance spectroscopy was done along with the conventional imaging no of cases detected was significantly increased to 84%.

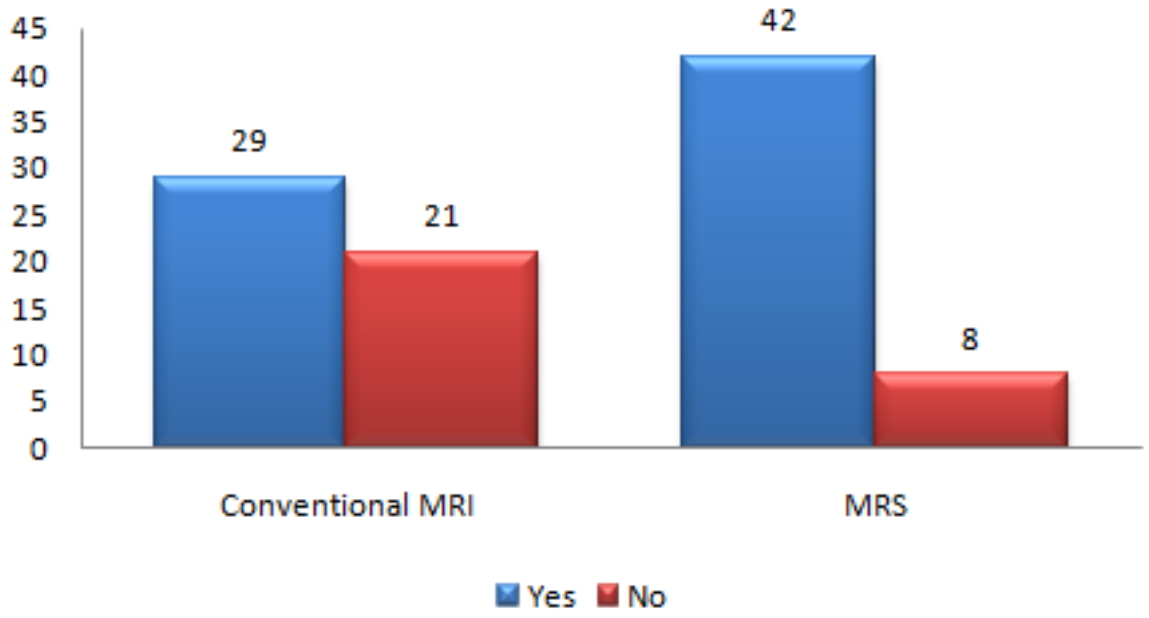


Fig:33 Bar diagram represents the comparison of case detection using conventional MRI and MR SPECTROSCOPY.

CROSS TABS

CONVENTIONAL & MRS

CONVENTIONAL	MRS	
	Yes	No
Yes	25	4
No	17	4

Test Statistics[a]

	CONVENTIONAL & MRS
N	50
Exact Sig. (2-tailed)	.007[b]

Table 7 ** Highly statistical Significance with P < 0.01 level

Comparison using McNemar Test

Comparison using McNemar Test		
Results	Conventional MRI	MRS
Yes	29	42
No	21	8
P-Values	0.007 **	
** Highly statistical Significance with P < 0.01 level		

- **Table 8 .On comparing the conventional MRI and MR Spectroscopy with Histo pathological examination by using MCNEMAR TESTS it was found that statistically significant difference exists with a p value of 0.007**

DISCUSSION

To completely image a spinal lesion of a patient, including T1 sagittal,T2sagittal,T2axial,postcontrastimagingand magnetic resonance spectroscopy it took around 30 minutes.

Spectral processing separately took about 5minutes including data transfer to a workstation. In magnetic resonance spectroscopy single voxel was applied by square or rectangle shape and it was difficult procedure to include a region of interest in a normal appearing spinal cord fully on any imaging plane. In some cases it produce potential partial volume effect or signal contamination from surrounding tissue.

Incase of large spinal lesions this difficulty was not the problem. It was possible to detect the signals.Amplitude of the spectrum represents the presence of a certain amount of a metabolite concentration.

Out of 50 subjects who underwent standard MRI spine and MR spectroscopy,21 were female and 29 subjects were male with a maximum age of 53yrs. minimum age included in this study was 7 yrs with a mean age group of 33yrs.

Of these 50 patients ,26 patients were found to have malignant lesions that is 52%.among these 12 patients had HPE proven high grade glioma 10 patients had ependymoma,4 patients had schwannoma.

Remaining 24 patients were found to have benign lesions including 9 intramedullary tuberculoma,7 patients with inflammatory lesions like acute transverse myelitis,neuromyelitis optica(excluding cases of multiple sclerosis) who presented with acute symptoms,3 known patients of multiple sclerosis,3 patients with spondylotic myelopathy,2 patients with dermoid cysts.

Magnetic resonance spectroscopy was done in 15 normal subjects without any symptoms as controls to find out the spectrum of metabolites in a normal looking spinal cord at cervical and dorsal levels. Even in normal appearing spinal cord, lipids signals were observed in a spreadout manner,It is difficult to determine whether this is due to contamination from surrounding tissues or because of intrinsic properties.

Elevated choline was nearly always observed in many malignant lesions like astrocytoma.In case of benign lesions like tuberculomas lipid lactate peak was merely observed.In inflammatory conditions like multiple sclerosis reduction in the N-acetyl aspartate with absent choline was observed.

From this study we observed that On applying conventional magnetic resonance imaging alone no of cases detected correctly is around 58%.when magnetic resonance spectroscopy was also done along with the conventional magnetic resonance imaging no of cases detected is significantly increased to 84%.

In the statistical analysis we observed that On comparing the conventional MRI and MR Spectroscopy with Histo pathological examination by using MCNEMAR TESTS it was found that statistically significant difference exists with a p value of 0.007 **.**

RESULTS

- Among the 15 normal subjects taken as control mild increase in the lactate peak was observed at 1.3 ppm in some subjects but rest of the metabolites were within normal limit
- In contrast to controls, spectra measured in different pathologies in the spinal cord show a distinct change in the metabolite fingerprint, with high correlation to connatural MR spectroscopy acquisitions in the brain.
- In **Tuberculoma** - Significant increase in Lipid and lactate with decreased NAA . Choline not significantly increased.
- In **Dermoid cyst** - found lipids mixed with lactate; lactate was also clearly observed at 1.3 ppm.
- In **Ependymoma** - Choline significantly increased, NAA decreased and lactate not very much decreased.In 3 patients we observed significant increase in the creatine peak at 3.9ppm.
- In **Low Grade Glioma** - NAA decreased, lactate not very much increased, creatine increased and Choline was found to be increased.
- In **GBM**- Choline increased, creatine almost zero, NAA decreased.
- In **MULTIPLE SCLEROSIS** and other inflammatory lesions significant reduction in NAA was observed.

- Kim et al investigated 14 mass lesions,he reported the following findings: " in a patient with tuberculosis, a very large lipid/lactate mixture was observed". **In our cases also we observed very large lipid/lactate peak.**

- In a dermoid cyst, found lipids mixed with lactate; lactate was also clearly observed at 4.18 and 1.33 ppm in one cyst. **In our 2 cases we observed lipid peak.**

- One intramedullary lesion around C5 was suggested to be ependymoma and glycine was detected at 3.45 ppm, whereas choline was increased and NAA decreased, **In our ependymoma cases Choline significantly increased and NAA decreased.in 3 cases we observed creatine pak at 3.9 ppm.**

- They reported spinal MR spectroscopy changes, similar to changes seen in the brain MR spectroscopy, however they did not show any quantification or metabolite ratios and quality evaluation of their results.

- Dydak et al. investigated one patient with a tumor in the cervical spinal cord.,they showed that lactate and myo-inositol are increased compared with healthy spinal cord tissue by visual inspection of the spectrum.

- They also did not provide metabolite concentrations and quality indicators.

- Henning et al. reported 2 patients with spinal cord tumor (1 located at C4–5 and 1 at T9) with 3T.

- Besides a decrease of NAA and Cr, an increase of Choline and myo-inositol was observed. **In our tumor cases also decrease of NAA and Cr, an increase in Choline was observed.**
- In addition, they reported an increased lactate and glutamine/glutamate peak in the tumor tissue.
- However, the spectra had a low SNR
- Hock et al. presented 3 patients with neoplastic spinal cord lesions and compared the spectra with those from 13 healthy volunteers and 13 patients with MS.
- The measured ependymoma showed strongly reduced NAA/Cr, increased Cho/Cr, and lactate compared to metabolite over Cr ratios measured in healthy controls. In our cases Choline significantly increased and NAA decreased.

However, more studies with high spectral quality in larger patient cohorts are needed to increase diagnostic confidence with respect to the degree that MR spectroscopy can enhance specific differential diagnoses or to provide a tool for monitoring the course of a disease

LIMITATIONS OF THIS STUDY:

1.susceptebility changes due to anatomic inhomogenecity

2.small size of the spinal cord for single voxel

3.CSF flow affecting the spectrum of metabolite

4.cardiac ,respiratory and patient motion influences the spectral quality

CONCLUSION

- MR spectroscopy of the spinal cord is a valuable noninvasive tool for research and diagnosis because it can provide additional information which is complementary to other noninvasive imaging methods.
- It is also an emerging tool which adds a new biomarker information to characterize the spinal cord tumors, to differentiate benign and malignant lesions thereby helps to prevent unnecessary biopsies and surgeries.
- However, the application of MR spectroscopy in the spinal cord is not straightforward, and great care is required to attain optimal spectral quality.

BIBLIOGRAPHY

1. Hock, A. Henning, P. Boesiger, and S.S. Kollias. **1H-MR Spectroscopy in the Human Spinal Cord.** AJNR Am J Neuroradiol Sep 2013 34:1682–89.

2. Kim YG, Choi GH, Kim DH, et al. **In vivo proton magnetic resonance spectroscopy of human spinal mass lesions.** *J Spinal Disord Tech* 2004;17:405–11

3. Kollias S, Goldstein R, Cogen P, et al. **Prenatally detected myelomeningoceles: sonographic accuracy in estimation of the spinal level.** *Radiology* 1992;185:109–12

4. Dahnert WF.radiology review manual.(2007)ISBN:0781766206

5. Abul-kasim K,ThurnerMM,Mckeever P et-al.intradural spinal tumours:current classification and MRI features.Neuroradiology.2008;50(4):301-14.

6. Seo HS,kimJH,Lee DH et al.non enhancing intramedullary astrocytomas and other MR imaging features: a retrospective study and systematic review.AJNR Am J Neuroradiol.2010;31(3);498-503

7. Smith AB ,Soderlund KA,Rushing EJ et-al.radiologic and pathologic correlation of pediatric and adolescent spinal neoplasms:part 1, intramedullary spinal neoplasm.AJR Am J Roentgenol.2012;198(1);34-43.

8. Baker KB,Moran CJ,Wippold FJ et-al.MR imaging of spinal hemangioblastoma.AJR Am J Roentgenol.2000;174(2):377-82.

9. Govind B,Chavan ,et al.MRI made easy:magnetic resonance spectroscopy.2006;17:158-171.

10. MarlianiAF,ClementiV,Albini-RiccioliL,etal.Quantitativecervical spinal cord 3T proton MR spectroscopy in multiple sclerosis. AJNR Am J Neuroradiol 2010;31:180–84

11. Dydak U, Kollias S, Scha¨r M, et al. MR spectroscopy in different regions of the spinal cord and in spinal cord tumors. In: Proceedings of the Annual Meeting of the International Society of Magnetic Resonance in Medicine, Miami Beach, Florida. May 7–13, 2005:813

12. Henning A, Schar M, Kollias SS, et al. Quantitative magnetic resonance spectroscopy in the entire human cervical spinal cord and beyond at 3T. Magn Reson Med 2008;59:1250–58

13. HollyLT,FreitasB,McArthurDL,etal.Protonmagneticresonance spectroscopytoevaluatespinalcordaxonalinjuryincervicalspondylotic myelopathy. J Neurosurg Spine 2009;10:194–200

14. Rapalino O, Law M, Salibi N, et al. Metabolite changes from MR spectroscopy in the cervical spinal cord in patients with cervical spondylosis.In:ProceedingsoftheAnnualMeetingoftheInternational Society of Magnetic Resonance in Medicine, Seattle, Washington. May 6–12, 2006:3139

15. Carew JD, Nair G, Pineda-Alonso N, et al. Magnetic resonance spectroscopy of the cervical cord in amyotrophic lateral sclerosis. Amyotroph Lateral Scler 2011;12:185

16. de Vita E, Kachramanoglou C, Wheeler-Kingshott CA, et al. Spinal cord1H-MRspectroscopyinpatientsafterbrachialplexusrootreimplantation. In: Proceedings of the Annual Meeting of the International Society of Magnetic Resonance in Medicine, Montreal, Que´bec, Canada. May 7–13, 2011:4290

17. Preul MC, Caramanos Z, Collins DL, et al. **Accurate, noninvasive diagnosis of human brain tumors by using proton magnetic resonance spectroscopy.** *Nat Med* 1996;2:323–25

18. Cooke FJ, Blamire AM, Manners DN, et al. **Quantitative proton magnetic resonance spectroscopy of the cervical spinal cord.** *Magn Reson Med* 2004;51:1122–28

19. Kendi AT, Tan FU, Kendi M, et al. **MR spectroscopy of cervical spinal cord in patients with multiple sclerosis.** *Neuroradiol* 2004;46:764–69

20. Anke Henning,1* Michael Scha¨r,1 Spyros S. Kollias,2 Peter Boesiger,1 and Ulrike Dydak1 **Quantitative Magnetic Resonance Spectroscopy in the Entire Human Cervical Spinal Cord and Beyond at 3T;** Magnetic Resonance in Medicine 59:1250–1258 (2008)

21. Loth F, Yardimci MA, Alperin N. **Hydrodynamic modeling of cerebrospinal fluid motion within the spinal cavity.** *J Biomech Eng* 2001;123:71–79.

ABBREVATIONS

1.MRS-MAGNETIC RESONANCE SPECTROSCOPY

2.TE-TIMED ECHO

3.TR-REPETITION TIME

4.CSF-CEREBRO SPINAL FLUID

5.CNS-CENTRAL NERVOUS SYSTEM

6.NF-NEURO FIBROMATOSIS

7.VHL-VON HIPPEL LINDAU

8.WHO-WORLD HEALTH ORGANISATION

9.MISME-MULTIPLE INHERITED SCHWANNOMA MENINGIOMA AND EPENDYMOMA

10.GBM-GLIOBLASTOMA MULTIFORME

11.NMO-NEUROMYELITIS OPTICA

12.NAA-N –ACETYL ASPARTATE

13.PPM-POINTS PER MILLION

14.1.5 T-TESLA

15.SNR-SIGNAL TO NOISE RATIO

16.STEAM-STIMULATED ECHO ACQUISITION METHOD

17.PRESS-POINT RESOLVED SPECTROSCOPY

18.ISIS-IMAGE SELECTED IN VIVO SPECTROSCOPY

19.SVS-SINGLE VOXEL SPECTROSCOPY

20.MVS-MULTIVOXEL SPECTROSCOPY

21.CSI-CHEMICAL SHIFT IMAGING

22.VOI-VOLUME OF INTEREST

24.CHESS-CHEMICAL SHIFT SELECTIVE SPECTROSCOPY

25.TMS-TETRA METHYLSILANE

PROFORMA

TITLE:

ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION

NAME:

AGE/SEX:

OCCUPATION:

ADDRESS:

PRESENTING COMPLAINTS

PAIN:

SIDE OF WEAKNESS:

SENSORY SYMPTOMS:

OTHER SYMPTOMS:

PAST HISTORY:

DIABETES MELLITUS:

HYPERTENSION:

TREATMENT HISTORY:

OTHERS:

VITAL SIGNS:

PULSE:

BP:

RESPIRATORY RATE:

GENERAL EXAMINATION:

LOCAL EXAMINATION:

INVESTIGATIONS:

MRI FINDINGS:

T1 WEIGHTED SEQUENCE	T2 WEIGHTED SEQUENCE	CONTRST ENHANCEMENT	DIFFUSION WEIGHTED SEQUENCE	GRADIENT ECHO SEQUENCE	SHORT TAU INVERSION RECOVERY (STIR)

MR SPECTROSCOPY FINDINGS

S.NO	METABOLITES	(ppm)	PEAK OBSERVED
1.	LACTATE	1.3	
2.	LIPID	1.3	
3.	ALANINE	1.48	
4.	N-ACETYL ASPARTATE	2	
5.	GLUTAMINE/ GLUTAMATE	2.2-2.4	
6.	GABA	2.2-2.4	
7.	CITRATE	2.6	
8.	CREATINE	3	
9.	CHOLINE	3.2	
10.	MYOINOSITOL	3.5	

GRAPHICAL REPRESENTATION:

INTERPRETATION

PATIENT INFORMATION SHEET

We are conducting a study on“ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION”

- Your cooperation would be valuable for the same
- The privacy of patients in the research will be maintained throughout the study.In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.
- Taking part of the study is voluntary.you are free to decide whether to participate in this study or to withdraw at any time.your decision will not result in any loss of benefits to which your are otherwise entitled.
- The result of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator Signature ofparticipant

(DR.R.BHARATHIPRIYA)

PATIENT CONSENT FORM

STUDY TITLE :

"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION"

STUDY CENTRE:

BARNARD INSTITUTE OR RADIOLOGY,
MADRAS MEDICAL COLLEGE,
RAJIV GANDHI GOVT GENERAL HOSPITAL,
CHENNAI-600003

PARTICIPANT

Name: Age: Sex:

I confirm that I have understood the purpose of procedure for the above study.I have the opportunity to ask the questions and all my questions and doubts have been answered to my satisfaction.

I have been explained about the pitfall in the procedure.I have been explained about the safety,advantage and disadvantage of the technique. I understand that my participation in the study is voluntary and that iam free to withdraw at anytime withoutgiving any reason.

I understand that investigator ,regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it,even if I withdraw from the study.I understand that my identity will not be revealed in any information released to third parties or published,unless as required under the law.I agree not to restrict the use of any data or results that arise from the study.

I have been explained that the MRS STUDY technique is a standard and approved technique.This may help in future research in the field of radiology.I consent to undergo this procedure.

DATE:

SIGNATURE/THUMB IMPRESSION OF PATIENT

S. No	NAME	AGE	SEX	LOCATION	MRI -T1	T2	MRS LIPID	LACTATE	CHOLINE	NAA	CREATINE	DIAGNOSIS	HPE
1	raji	23	F	CERVICAL	HYPO	HYPER	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	TUBERCULOMA
2	parvathy	33	F	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	MULTIPLE SCLEROSIS	-
3	tamil	27	M	DORSAL	HYPO	HYPER	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	TUBERCULOMA
4	gnanam	53	M	DORSAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	TRANSVERSE MYELITIS	-
5	mani	44	M	CERVICAL	HYPO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	HIGH GRADE GLIOMA
6	sundaram	32	M	CERVICAL	HETERO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	ELEVATED	EPENDYMOMA	GLIOMA
7	veena	42	F	CERVICAL	HYPO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	ELEVATED	EPENDYMOMA	HIGH GRADE GLIOMA
8	KANNAPAN	36	M	CERVICAL	HETERO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	EPENDYMOMA	EPENDYMOMA
9	poongavanam	36	M	DORSAL	HYPO	HYPER	NORMAL	NORMAL	INCONCLUSIVE		NORMAL	ASTROCYTOMA	ASTROCYTOMA
10	mari	48	M	CERVICAL	HYPO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	INFLAMMATORY ETIOLOGY
11	durai	52	M	DORSAL	HYPO	HETERO	INCONCLUSIVE					TUBERCULOMA	INFLAMMATORY ETIOLOGY
12	chinnapillai	50	F	DORSAL	HETERO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	ELEVATED	ASTROCYTOMA	HIGH GRADE GLIOMA
13	mala	35	F	CERVICAL	HYPO	HYPER	NORMAL	ELEVATED	NORMAL	REDUCED	NORMAL	MULTIPLE SCLEROSIS	-
14	rohini	33	F	DORSAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	TRANSVERSE MYELITIS	-
15	VASANTHAN	41	M	CERVICAL	HETERO	HETERO	INCONCLUSIVE					EPENDYMOMA	GLIOMA
16	ravi	53	M	CERVICAL	HYPO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	SPONDYLOTIC MYELOPATHY	-
17	radha	36	F	CERVICAL	HETERO	HETERO	NORMAL	NORMAL	ELEVATED	REDUCED	ELEVATED	EPENDYMOMA	EPENDYMOMA
18	roshni	22	F	DORSAL	HYPO	HYPER	INCONCLUSIVE					NEUROMYELITIS OPTICA	-
19	devi	7	F	DORSAL	HYPO	HETERO	NORMAL	ELEVATED	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	
20	mariappan	31	M	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	TRANSVERSE MYELITIS	TUBERCULOMA
21	karupu	28	M	CERVICODORSAL	HYPO	HYPER	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	INFLAMMATORY ETIOLOGY
22	naveen	27	M	LUMBAR	HYPO	HETERO	NORMAL	ELEVATED	NORMAL	REDUCED	NORMAL	NECROTIC SCHWANNOMA	SCHWANNOMA
23	priya	37	F	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	INCONCLUSIVE			INFLAMMATORY	ASTROCYTOMA
24	sundaravalli	32	F	DORSAL	HYPO	HYPER	NORMAL	NORMAL	INCONCLUSIVE			MULTIPLE SCLEROSIS	-
25	mohana	28	F	DORSAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	TRANSVERSE MYELITIS	-
26	ramasamy	31	M	CERVICAL	HETERO	HETERO	NORMAL	ELEVATED	ELEVATED	REDUCED	NORMAL	EPENDYMOMA	GLIOMA
27	narayanan	48	M	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	ASTROCYTOMA
28	seenu	52	M	CERVICAL	HYPO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	SPONDYLOTIC MYELOPATHY	-
29	mayilu	39	F	DORSAL	HYPO	HYPER		INCONCLUSIVE			NORMAL	ASTROCYTOMA	HIGH GRADE GLIOMA
30	rajan	35	M	DORSAL	HYPO	HYPER	ELEVATED	NORMAL	NORMAL	NORMAL	NORMAL	TUBERCULOMA	TUBERCULOMA
31	suganthi	34	F	CERVICAL	HETERO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	ELEVATED	EPENDYMOMA	EPENDYMOMA
32	subramani	49	M	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	SPONDYLOTIC MYELOPATHY	-

S. No	NAME	AGE	SEX	LOCATION	MRI -T1	T2	MRS LIPID	LACTATE	CHOLINE	NAA	CREATINE	DIAGNOSIS	HPE
33	mohana	24	F	CERVICAL	HYPO	HYPER	INCONCLUSIVE					NEUROMYELITIS OPTICA	-
34	karunakaran	44	M	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	NORMAL	SCHWANNOMA	SCHWANNOMA
35	alliammal	42	F	DORSAL	HYPO	HYPER	NORMAL	NORMAL	NORMAL	REDUCED	ELEVATED	EPENDYMOMA	HIGH GRADE GLIOMA
36	rengan	37	M	DORSAL	HYPO	HYPER	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	TUBERCULOMA
37	srinivasan	44	M	DORSAL	HYPO	HYPER	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	HIGH GRADE GLIOMA
38	revathi	32	F	LUMBAR	HETERO	HETERO	ELEVATED	NORMAL	NORMAL	NORMAL	NORMAL	DERMOID	DERMOID
39	muthu	38	M	LUMBAR	HYPO	HETERO	NORMAL	ELEVATED	NORMAL	NORMAL	NORMAL	SCHWANNOMA	SCHWANNOMA
40	rosy	33	F	CERVICAL	HETERO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	ELEVATED	EPENDYMOMA	EPENDYMOMA
41	basha	29	M	DORSAL	HYPO	HYPER	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	INFLAMMATORY ETIOLOGY
42	barani	40	M	DORSAL	HYPO	HYPER	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	ASTROCYTOMA
43	balachander	44	M	CERVICAL	HYPO	HETERO	ELEVATED	ELEVATED	NORMAL	NORMAL	NORMAL	TUBERCULOMA	INFLAMMATORY ETIOLOGY
44	malar	32	F	DORSAL	HYPO	HETERO	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	SCHWANNOMA	EPENDYMOMA
45	vinayagam	31	M	DORSAL	HYPO	HETERO	NORMAL	NORMAL	NORMAL	REDUCED	ELEVATED	EPENDYMOMA	EPENDYMOMA
46	pattu	41	F	CERVICODORSAL	HYPO	HYPER		INCONCLUSIVE				TRANSVERSE MYELITIS	INFLAMMATORY ETIOLOGY
47	sebastien	42	M	DORSAL	HYPO	HYPER	INCONCLUSIVE					ASTROCYTOMA	TUBERCULOMA
48	veeran	22	M	LUMBAR	HETERO	HETERO	ELEVATED	NORMAL	NORMAL	NORMAL	NORMAL	DERMOID	DERMOID
49	KALI	29	M	DORSAL	HYPO	HYPER	INCONCLUSIVE					TUBERCULOMA	TUBERCULOMA
50	veni	38	F	CERVICAL	HYPO	HYPER	NORMAL	NORMAL	ELEVATED	REDUCED	NORMAL	ASTROCYTOMA	GLIOMA

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.R.Bharathipriya
IInd Year P.G. in MD RD
Madras Medical College/RGGGH
Chennai 600 003

Dear Dr.R.Bharathipriya,

The Institutional Ethics Committee has considered your request and approved your study titled " **ROLE OF 3TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION "** - **NO.21012016.**

The following members of Ethics Committee were present in the meeting hold on **05.01.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD.,Inst.of Pharmacology,MMC,Ch-3	: Member
5.Prof.Md.Ali,MD.,DM.,HOD-MGE, MMC,Ch-3	: Member
6.Prof.K.Ramadevi,MD, Director, Inst. of Bio-Chem,MMC,Ch-3	: Member
7.Prof.M.Saraswathi, MD.,Director, Inst.of Path,MMC,Ch-3	: Member
8.Prof.Srinivasagalu,MD.Director,Inst.of Int.Med.MMC,Ch-3	:Member
9.Tmt.J.Rajalakshmi,JAO,MMC,Ch-3	: Lay Person
10.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
11.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

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

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

MADRAS MEDICAL COLLEGE
21 JAN 2016
CHENNAI-600 003

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION"

INTRODUCTION

A spectrum of diagnostic considerations may affect the spinal cord which includes developmental anomalies, inflammatory and infectious processes, vascular disease, degenerative conditions as well as benign and malignant neoplasms. Patients with intramedullary spinal cord lesions commonly present with tingling pain, numbness and weakness.

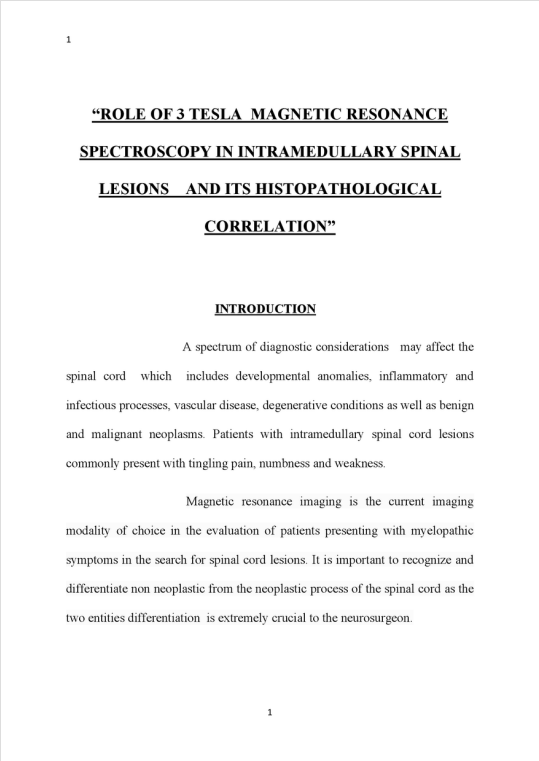
Magnetic resonance imaging is the current imaging modality of choice in the evaluation of patients presenting with myelopathic symptoms in the search for spinal cord lesions. It is important to recognize and differentiate non neoplastic from the neoplastic process of the spinal cord as the

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201418002 Md Radio Diag BHARAT.
Assignment title:	2015-2015 plagiarism
Submission title:	ROLE OF 3 TESLA MAGNETIC RES..
File name:	DISSERTATION.docx
File size:	5.38M
Page count:	86
Word count:	7,064
Character count:	39,980
Submission date:	13-Sep-2016 05:45PM
Submission ID:	704667014



1

"ROLE OF 3 TESLA MAGNETIC RESONANCE SPECTROSCOPY IN INTRAMEDULLARY SPINAL LESIONS AND ITS HISTOPATHOLOGICAL CORRELATION"

INTRODUCTION

A spectrum of diagnostic considerations may affect the spinal cord which includes developmental anomalies, inflammatory and infectious processes, vascular disease, degenerative conditions as well as benign and malignant neoplasms. Patients with intramedullary spinal cord lesions commonly present with tingling pain, numbness and weakness.

Magnetic resonance imaging is the current imaging modality of choice in the evaluation of patients presenting with myelopathic symptoms in the search for spinal cord lesions. It is important to recognize and differentiate non neoplastic from the neoplastic process of the spinal cord as the two entities differentiation is extremely crucial to the neurosurgeon.

1