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Role of MRI in Evaluation of Cerebral Demyelinating Disorders

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Abstract
Aims &Objectives :-
- *To evaluate the role of magnetic resonance imaging in demyelinating diseases and to demonstrate the different patterns of abnormal myelination.*
- *To establish an accurate diagnosis.*
- *To narrow down the differential diagnosis in various Demyelinating disorders and also to assess the severity and extent of the underlying lesion in various conditions of Demyelinatingdisorders.*

Material and Methods :-
This is a study of 20 patients referred to the department of radiodiagnosis with clinical history suspicious of Demyelinating Diseases & underwent Magnetic resonance imaging. The study was performed on 1.5T SIEMENS MRI machine at GCS Hospital, AHMEDABAD over a period of 24 months from January 2021 to December 2022.

Results:
Multiple sclerosis was most commonly encountered demyelinating disease in my study of 20 cases, common age of presentation was 31-40 years. It was followed by ADEM, PRES, Metabolic encephalopathy, CADASIL and extrapontinemyelinosis. MRI has important role in differentiating active from inactive disease phase based on enhancement pattern and diffusion restriction. Different pattern of abnormal myelination on T2W and FLAIR images in different demyelinating disease in adjunct with detailed clinical history helped to reach an accurate diagnosis.

Conclusion:
Magnetic resonance imaging (MRI) has become a very important tool in diagnosis and differentiation of different demyelinating disorders. Disease monitoring throughout its course, treatment response and prognosis are often based on the combination of clinical symptoms and neuroimaging findings. MRI is the most commonly used imaging modality as it offers high-resolution images in a noninvasive and safe method, without exposing patients to ionizing radiation.

Keywords: *Magnetic resonance imaging (MRI), cerebral demyelinating diseases, neurological complaints*

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I. Introduction

White matter diseases include a wide spectrum of disorders that have in common impairment of normal myelination, either by secondary destruction of previously myelinated structures **(demyelinating processes)** or by primary abnormalities of myelin formation **(dysmyelinating processes)**. The pathogenesis of many white matter diseases remains poorly understood.

Demyelinating disorders are further divided into autoimmune, infectious, vascular, and toxic-metabolic processes.

The imaging spectrum can vary widely from small multifocal white matter lesions to confluent or extensive white matter involvement. A systematic approach to the radiologic findings, in correlation with clinical and laboratory data, is crucial for narrowing the differential diagnosis.

II. Material And Methods

This is a study of 20 patients referred to the department of radiodiagnosis with clinical history suspicious of Demyelinating Diseases & underwent Magnetic resonance imaging. The study was performed on 1.5T SIEMENS MRI machine at GCS Hospital, AHMEDABAD over a period of 24 months from January 2021 to December 2022.

Inclusion criteria : -

Signs & symptoms consistent with demyelinating disease

Visual: Blurred vision, Unilateral loss of vision, Afferent pupillary defect (APD), Diplopia, Nystagmus

Motor : Muscle weakness, Spasticity, Hyperreflexia, Gait disturbance, Imbalance problems

Sensory: Numbness, Paresthesias, Trigeminal neuralgia

Cerebellar : Tremor, Ataxia, Incoordination

Genitourinary: Urgency/frequency/retention, Incontinence, Frequent UTI, Constipation, Impotence, Anorgasmia, Dyspareunia

Neuropsychiatric: Cognitive impairment, Depression, Irritability

Exclusion criteria : -

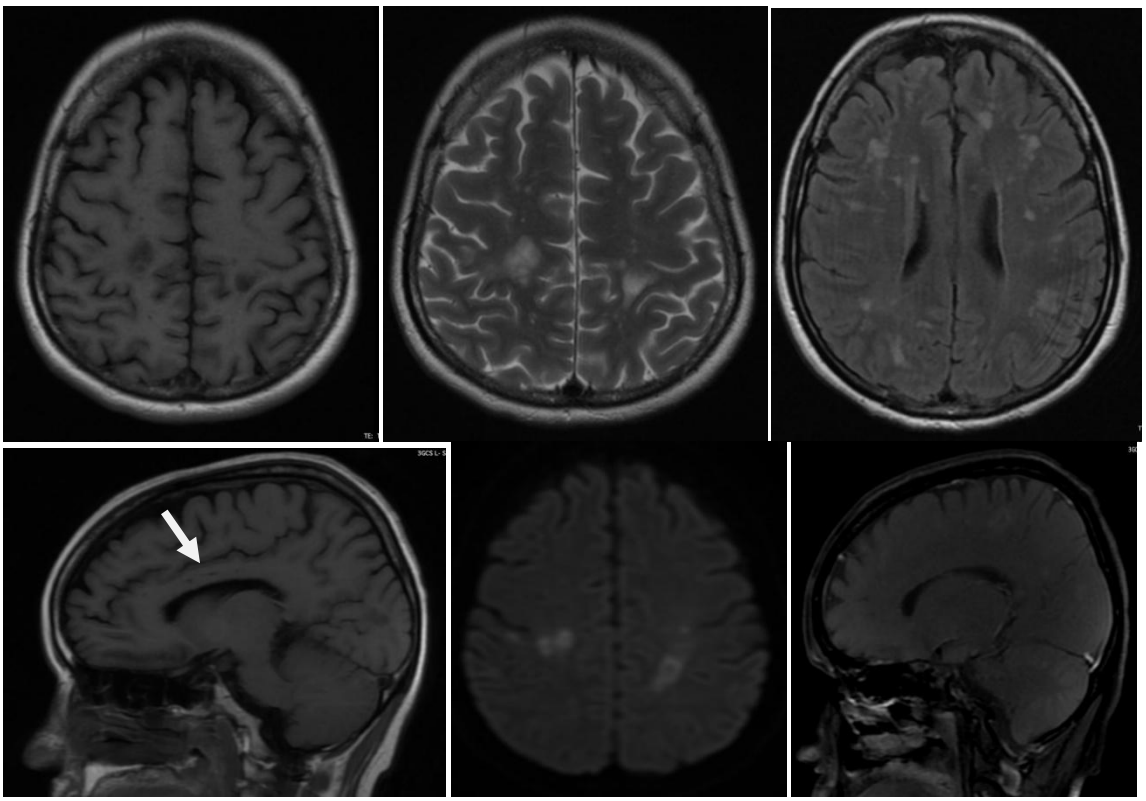
- Implanted electric and electronic devices are a strict contraindication to the magnetic resonance imaging, and in particular - heart pacemakers (especially older types), insulin pumps, implanted hearing aids, neurostimulators, intracranial metal clips, metallic bodies in the eye
- Metal hip replacements (old type), sutures or foreign bodies in other sites are relative contraindications to the MRI because they obscure the visualization of normal anatomy due to artifact effect.

III. Result

CASE 1

Case of Multiple Sclerosis : -

35 year old female with long-term neurologic complaints - heat intolerance ,stumbling gait and a tendency to fall, periodical changes in visual acuity during several years.

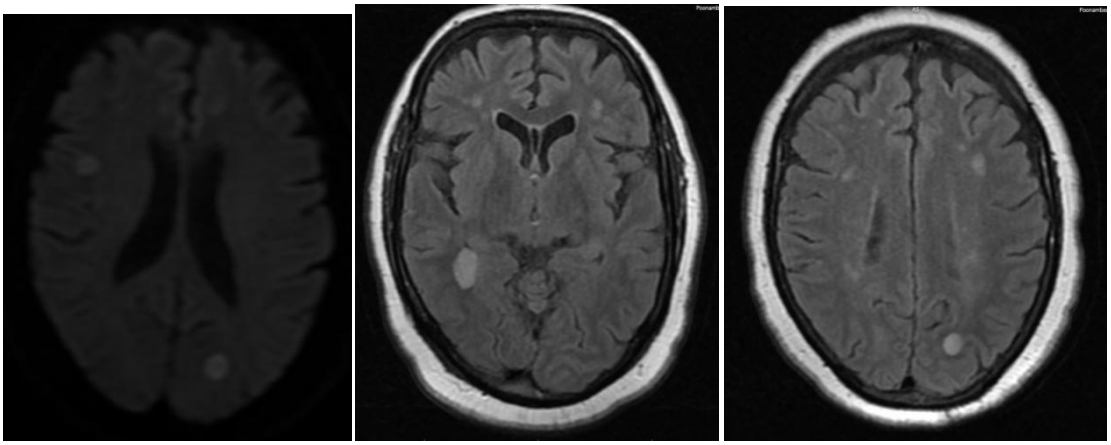


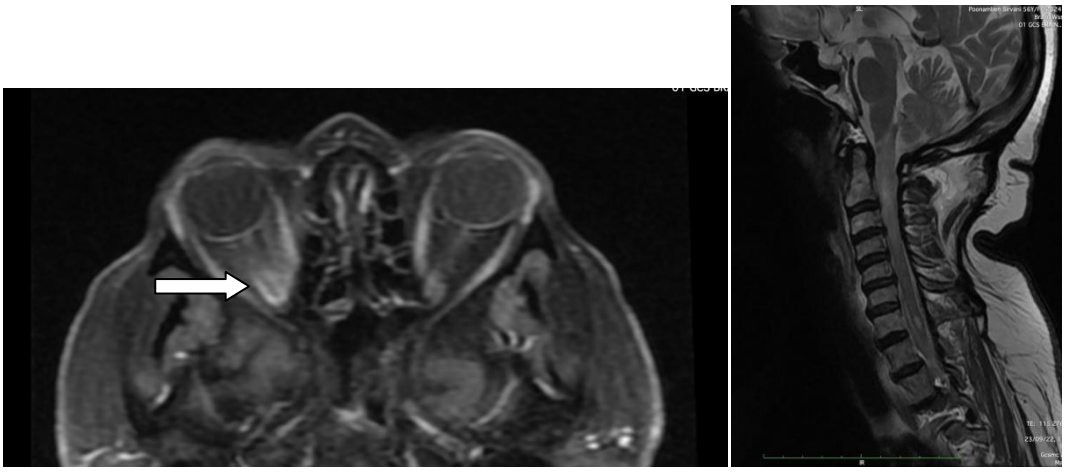
- **Multiple ill-defined variable sized altered signal intensity lesions are noted in periventricular, subcortical white matter in bilateral cerebral hemispheres,bilateral corona radiata, centrum semiovale.**
- **Few of the lesions are seen perpendicular upto the lateral ventricle and are seen in callososeptal interface.**
- **These lesions are hypointense on T1W, hyperintense on T2W and FLAIR images.**
- **ON DWI, few of the lesions are showing patchy areas of diffusion restriction with moderately low ADC.**
- **On post contrast study, the lesions show ring enhancement.**

CASE 2

Case of Neuromyelitis Optica:

56-year-old female with complaints of weakness of all four limbs, impaired vision, urinary incontinence, and dyspnea.



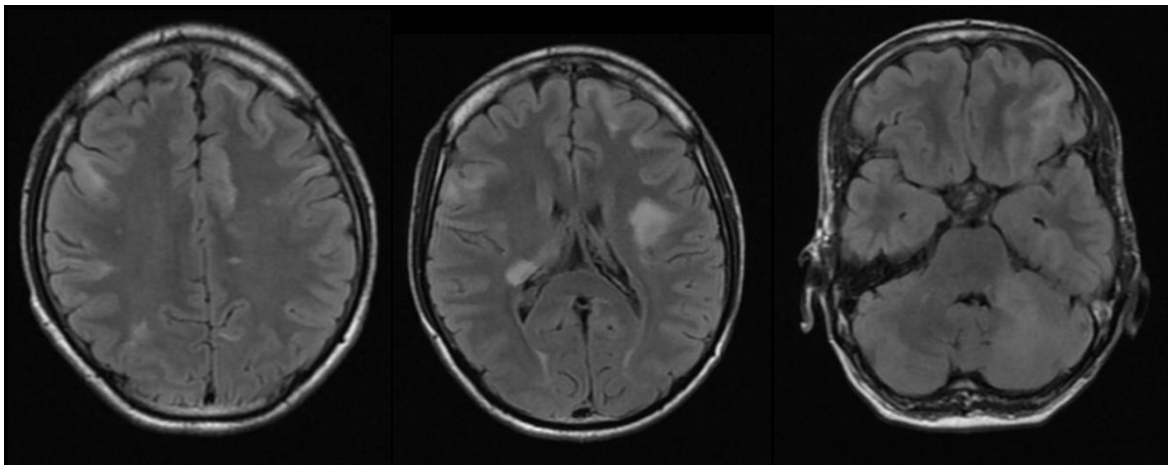


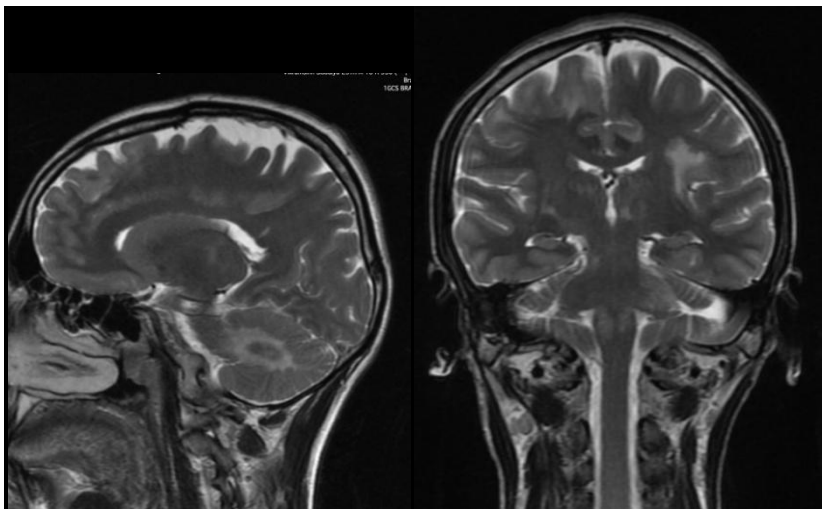
- **Few variable sized FLAIR hyperintense lesions are noted involving subcortical deep white matter of bilateral cerebral hemisphere which shows partial loss of grey-white matter differentiation.**
- **On DWI, it shows peripheral diffusion restriction.**
- **Presence of intramedullary T2W hyperintese area is noted involving lower part of medulla, cervicomedullary junction and long segment of cervical spinal cord upto C4 level.**
- **Right optic nerve appears swollen and shows enhancement on post contrast study.**

CASE 3

Case of Progressive Multifocal Leukoencephalopathy: -

A 29-year-old with complaints of first focal epileptic seizure and mild cognitive impairment. (The diagnosis of progressive multifocal leukoencephalopathy was established by histological analysis and detection of JC-virus desoxyribonucleic acid in brain biopsy specimens).



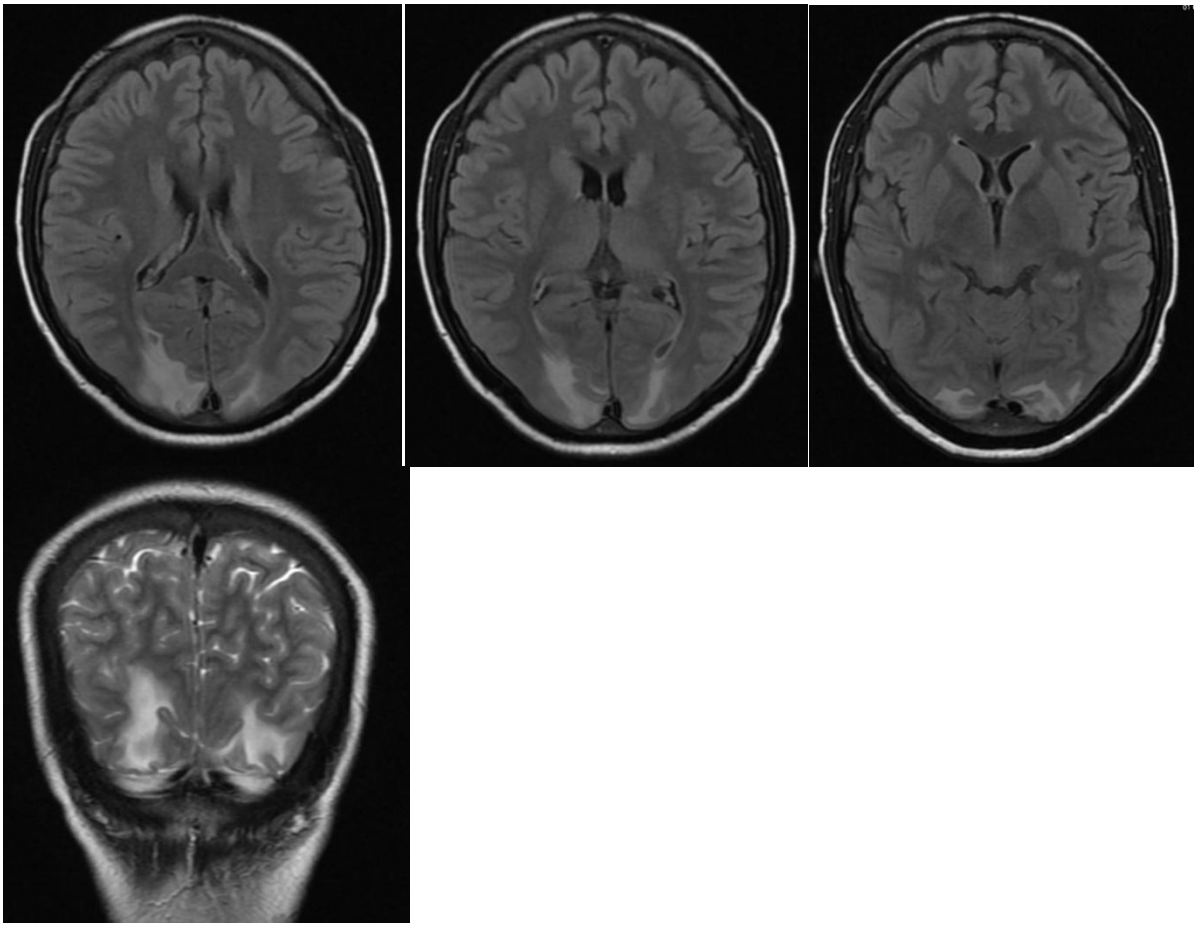


- **T2W and FLAIR hyperintensity is noted involving bilateral basal ganglia, left insular cortex, bilateral high frontal region, cortical - subcortical region of bilateral high frontal and right parietal region, cerebellum on left side and medulla on both sides.**

CASE 4

Case of Posterior Reversible Encephalopathy Syndrome : -

26 year old male with acute onset of headache, altered level of consciousness, visual alterations, seizures, nausea, and vomiting.

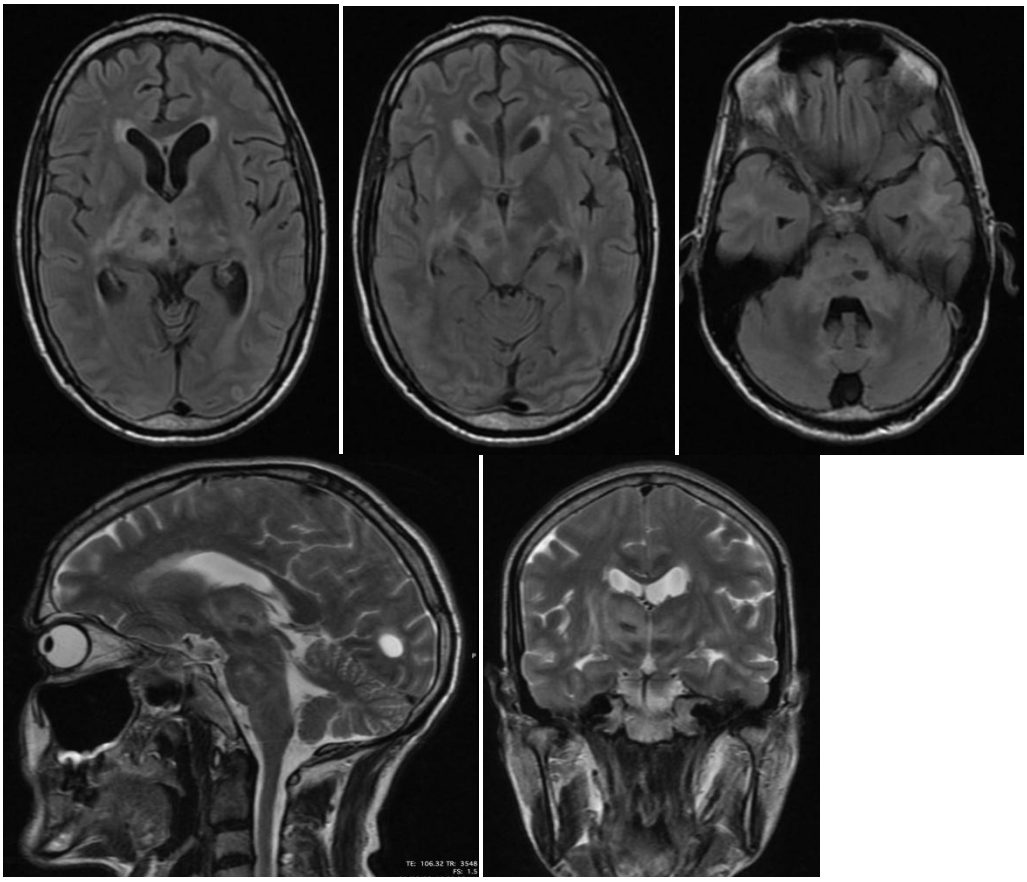


- **Bilateral symmetrical FLAIR and T2W hyperintensity noted involving both occipital region.**
- **(On DWI sequences: No evidence of restriction was noted)**

CASE 5

Case of Metabolic (Uremic) Encephalopathy : -

A 57-year-old male on chronic hemodialysis for 4 years because of diabetic nephropathy with rapid-onset gait disturbance, severe dysarthria, and consciousness disturbance.

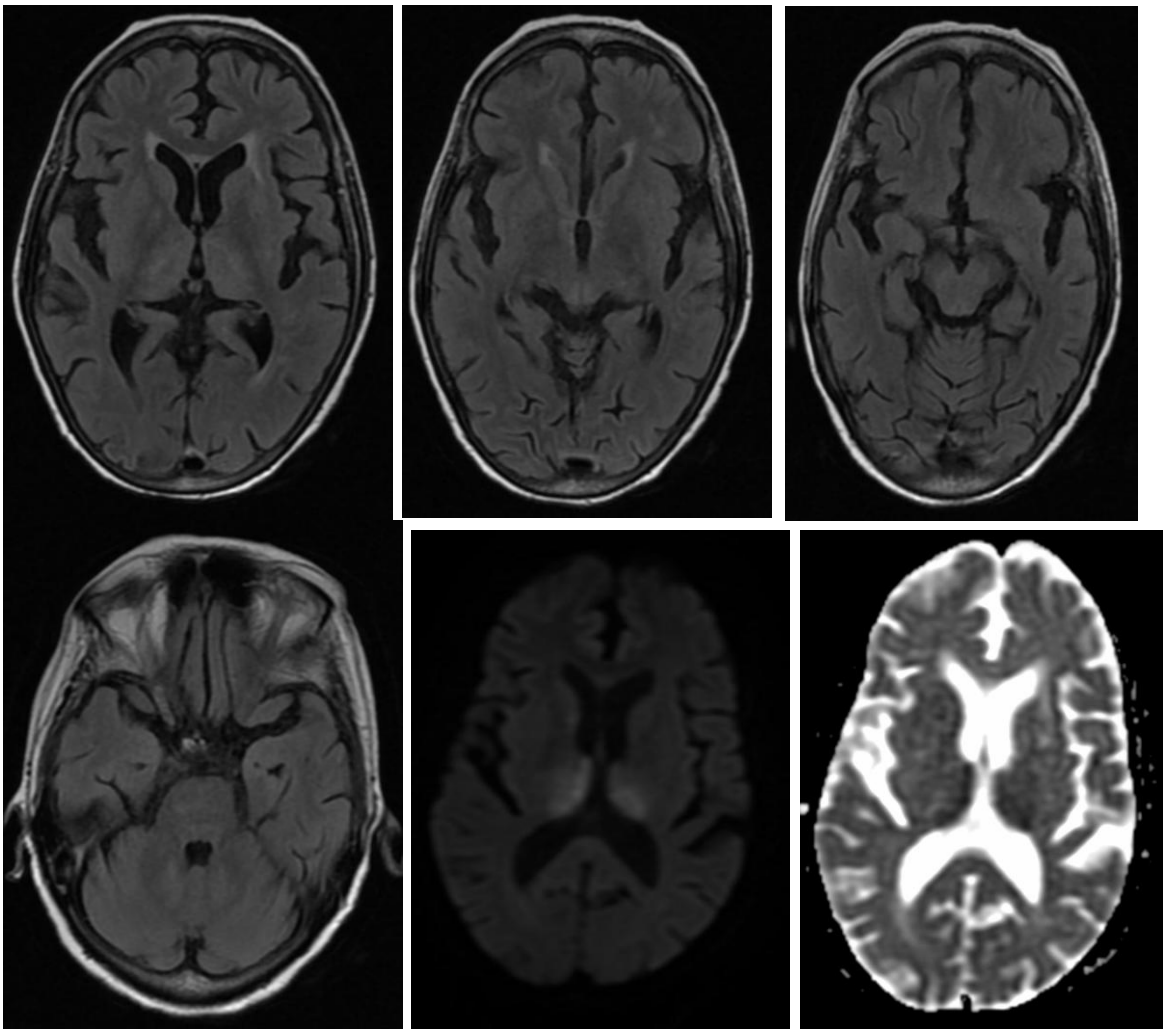


- **Bilateral symmetrical T2W - FLAIR hyperintensities noted involving bilateral thalami , centrum semiovale , cortical and subcortical white matter , temporal lobes , posterior lobes of cerebellar hemisphere and cerebellar follia.**
- **Similar characteristic patterns are seen involving brainstem ,periaqeductal white matter and posterior limb of right internal capsule.**

CASE 6

Case of Extrapontine Myelinosis:

A young alcoholic male patient with a history of loose stools and vomitings. There was documentation of hyponatraemia (Na-112 mEq/L) which was treated with intravenous normal saline. Next day, sodium was documented to be 135 mEq/L. On the third day, the patient had worsening of sensorium.

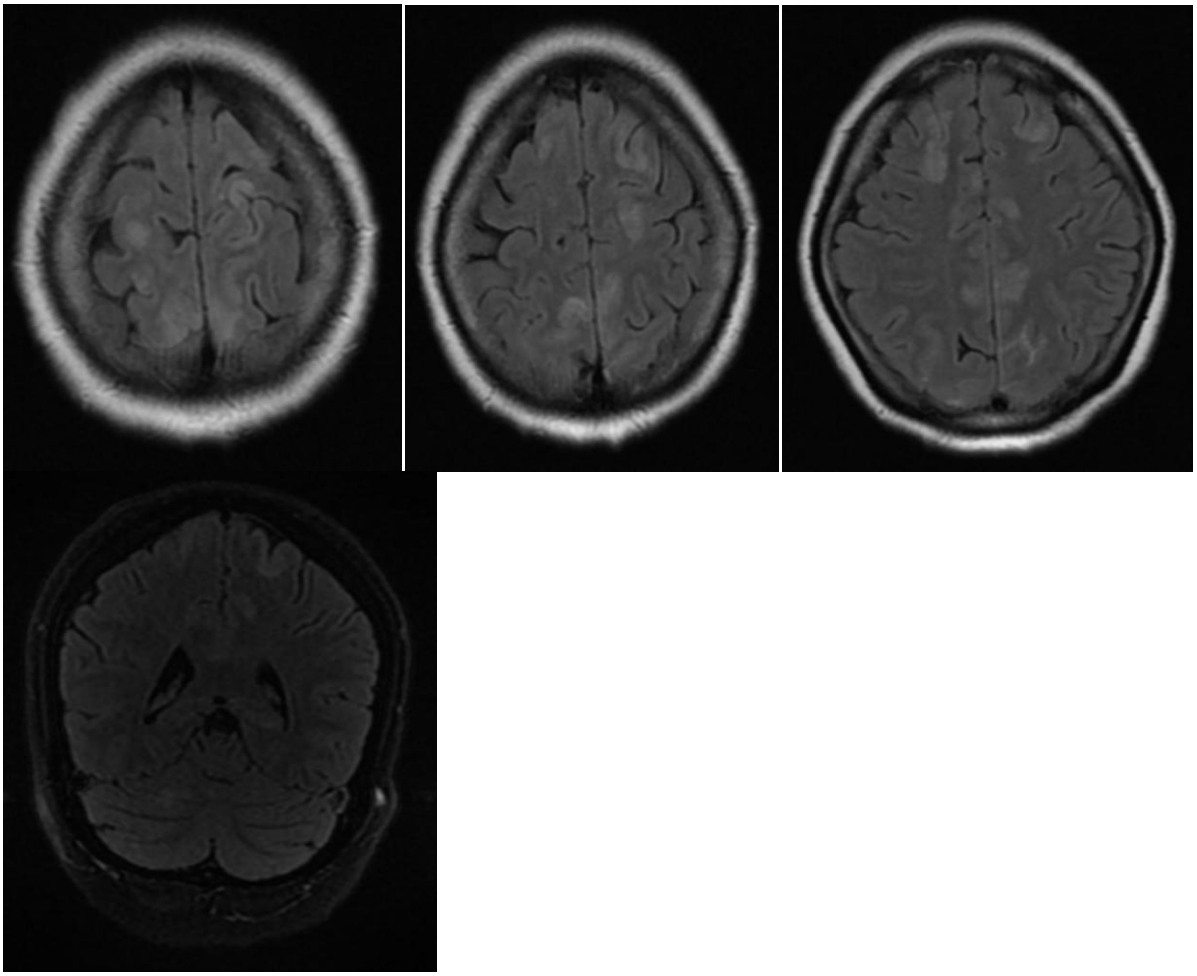


- **Areas of bilateral symmetrical restricted diffusion noted involving bilateral thalamus, midbrain and cerebellum which shows low values on ADC images.**
- **These areas appear hyperintense on T2W and FLAIR images.**

CASE 7

Case of Chemotherapy induced toxic encephalopathy:

A 40-year-old lady received her first cycle of Cisplatin for palliative treatment of lung carcinoma, and presented several days later with signs of encephalopathy.

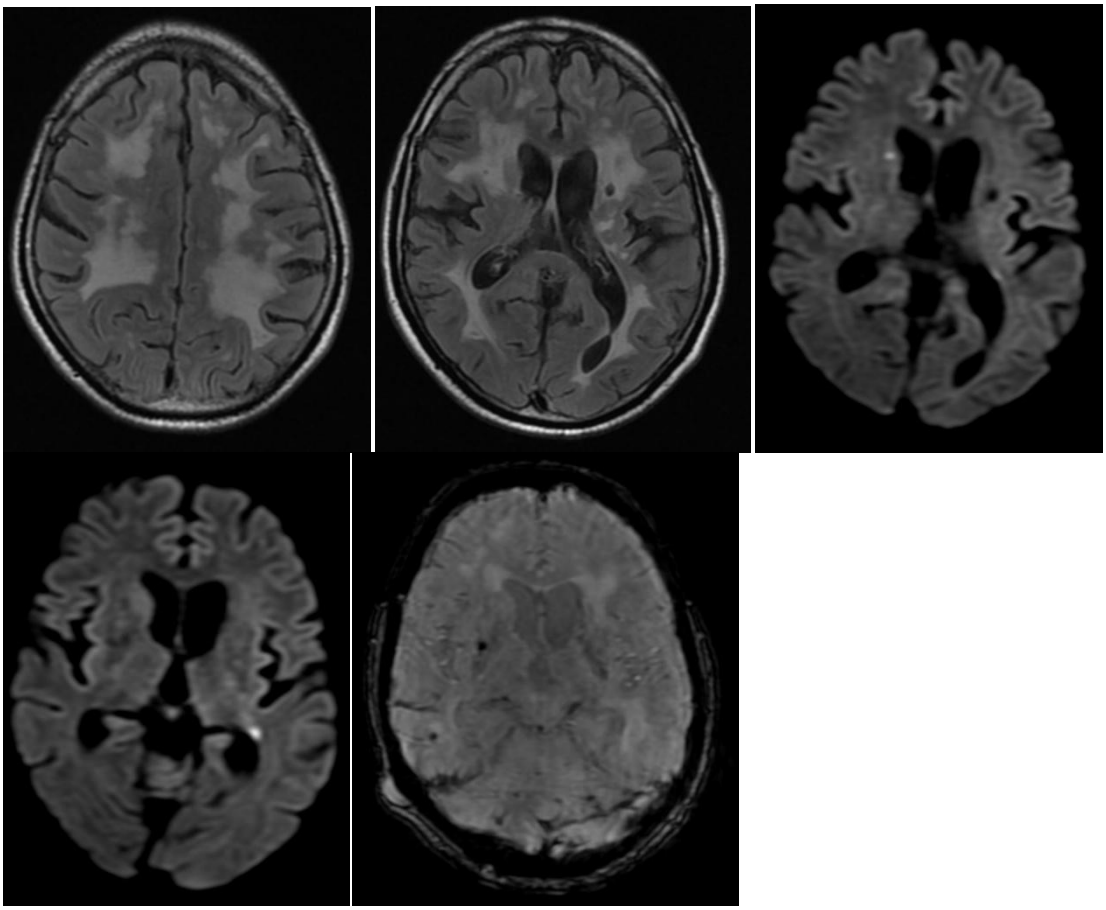


Diffuse bilaterally symmetrical T2 and FLAIR hyperintense signals in the subcortical and deep white matter, involving bilateral fronto-parietal lobes.

(No restriction on DW and ADC images was seen. No blooming on SWI seen. No post contrast enhancement was seen).

CASE 8

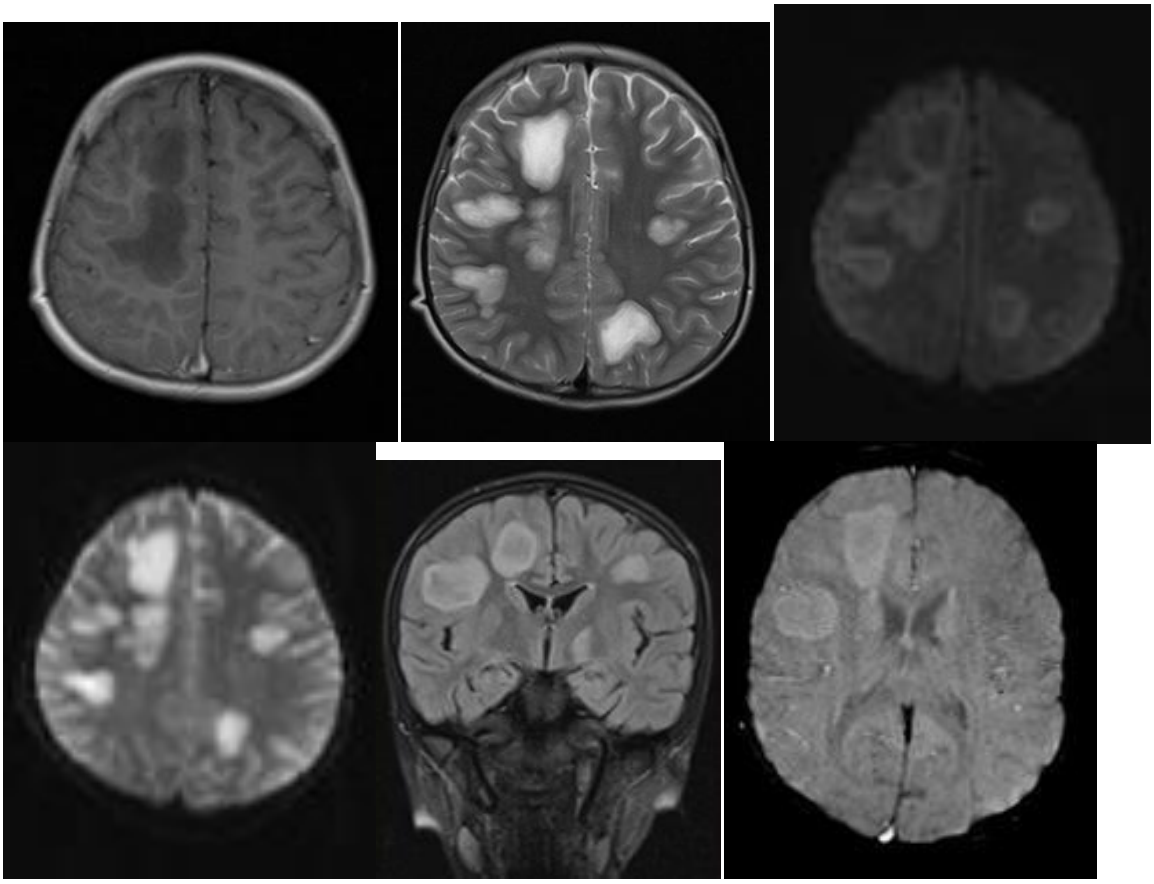
Case of CADASIL- (Cerebral Autosomal Dominant Arteriopathy with Sub-cortical Infarcts and Leukoencephalopathy):
A 55-year-old man with history of severe hypertension and diabetes with few episodes of transient ischemic attacks.



- **Multiple discrete and confluent hyperintensities in periventricular, subcortical white matter and subcortical U fibers more predominantly in bilateral posterior parietooccipital lobe and in bilateral anterior temporal lobes**
- **Chronic infarcts in bilateral corona radiata and gangliocapsular region.**
- **Multiple microbleeds in bilateral gangliocapsular and pons.**

CASE 9

Case of Acute Disseminated Encephalomyelitis (ADEM):
A 14-year-old boy with complaints of slurring of speech and difficulty in swallowing and fever for 2 days.



Numerous white matter lesions with high T2/FLAIR signal, and peripheral incomplete (open-ring) enhancement, and restricted diffusion along the enhancing rim.

Table 1: Frequency of demyelinating disorders diagnosed on MRI

MRI diagnosis	Frequency	Percentage	Gender	
			Male	Female
Multiple sclerosis	6	30 %	2	4
Neuromyelitisoptica	1	5%	0	1
Progressive multifocal leukoencephalopathy	1	5%	1	0
PRES – posterior reversible encephalopathy syndrome	2	10%	1	1
Metabolic encephalopathy	2	10%	1	1
Extrapontinemyelinosis	2	10%	1	1
Toxic encephalopathy	1	5%	1	0
Vascular - CADASIL	2	10%	1	1
Acute disseminated encephalomyelitis (ADEM)	3	15%	2	1
	20	30 %	10	10

Table 2: Age distribution among the subjects having demyelinating disorders

MRI diagnosis	Age groups (in years)						
	0-10	11-20	21-30	31-40	41-50	>50	Total
Multiple sclerosis	0	0	0	5	2	0	7
Neuromyelitisoptica	0	0	0	0	1	0	1
Progressive multifocal	0	0	0	1	0	0	1

leukoencephalopathy							
PRES – posterior reversible encephalopathy syndrome	0	0	2	0	0	0	2
Metabolic (uremic) encephalopathy	0	0	1	0	1	0	2
Extrapontinemyelinosis	0	0	0	2	1	1	4
Toxic encephalopathy	0	0	0	1	0	0	1
Vascular - CADASIL	0	0	0	0	0	2	2
Acute disseminated encephalomyelitis (ADEM)	1	2	0	0	0	0	0
	1	2	3	9	5	3	20

Multiple sclerosis was most commonly encountered demyelinating disease in my study of 20 cases, common age of presentation was 31-40 years. It was followed by ADEM, PRES, Metabolic encephalopathy, CADASIL and extrapontinemyelinosis. MRI has important role in differentiating active from inactive disease phase based on enhancement pattern and diffusion restriction.Different pattern of abnormal myelination on T2W and FLAIR images in different demyelinating disease in adjunct with detailed clinical history helped to reach an accurate diagnosis.

IV. DISCUSSION

As treatment strategies differ among these diseases, precise diagnosis of demyelinating diseases is crucial, and magnetic resonance imaging (MRI) plays a pivotal role in the diagnosis. The most important roles of magnetic resonance imaging (MRI) in demyelinating diseases include: (i) diagnosis (ii) disease monitoring (iii) differentiating active from inactive disease and monitoring of side-effects from disease-modifying drugs.[(1)]

MULTIPLE SCLEROSIS:

Diagnosis of MS is based on neurological examination to determine the presence of certain clinical symptoms and signs and is supported by other tests, such as MRI, evoked potential tests in visual, sensory, or auditory pathways and cerebrospinal fluid (CSF) analysis. MRI is highly recommended in patients with symptoms and signs suggestive of MS due to the high sensitivity to detect typical brain and spinal cord lesions. MS is characterized by perivenular inflammation and demyelination, manifesting as periventricular, infratentorial, juxtacortical, and spinal cord lesions[(2)]. Periventricular white matter lesions are hyperintense on T2-weighted images, ovoid, perpendicular to the ventricle, with a perivenular topography (so-called "Dawson's fingers"), and appear dark on T1-weighted images ("black holes"). The corpus callosum lesions are localized at the calloso-septal interface. Generally, they are small in size, focal, and separated from each other, determining the typical subcallosal "dot-dash" appearance. Another classic MS location is the involvement of subcortical U-fibers, as isolated juxtacortical white matter hyperintensity on T2-weighted images: this type of lesion is relatively specific for MS; on the other hand, the lesions can also be localized in the cortex[(3)]. More than 90% of patients with clinically definite MS have spinal cord abnormalities, although isolated spinal cord lesions can occur in 25% of patients[(4)]. The cervical region is the most commonly affected segment of the spinal cord. Typically, the lesions are short (1–2 vertebral bodies) in craniocaudal extent, often multifocal and asymmetric, and affect less than half of the cross-sectional area of the cord [(5)]. The lesions can demonstrate contrast enhancement or cord swelling in active demyelination MS[(5)]. According to the McDonald 2010 criteria, the diagnosis of MS requires the evidence of dissemination in time and space of demyelinating lesions, including in patients with CIS [(6)]. The MRI dissemination in space is defined by the presence of a T2 lesion in at least two of the four classical sites of white matter disease (juxtacortical, periventricular, infratentorial regions, and spinal cord), whereas dissemination in time requires simultaneous presence of asymptomatic gadolinium-enhancing and non-enhancing lesions, or the appearance of a new lesion during the follow-up [(6)].

NEUROMYELITIS OPTICA:

Neuromyelitisoptica (NMO) is an inflammatory disease of the CNS that is characterized by severe attacks of optic neuritis (ON) and longitudinally extensive transverse myelitis (LETM). [(7)]

Overlap of imaging findings of these two entities (MS and NMO) is higher in Asian populations. Nonetheless, features that are helpful in favoring NMO over multiple sclerosis include[(8,9,10):]Brain- smooth confluent periependymaldistribution,fewer oval perivenular orientation of periventricular lesions (no Dawson's fingers),fewer juxtacortical lesions (U-fiber), more extensive involvement of the corpus callosum (especially its ependymal surface), larger, more confluent lesions, lack of open ring enhancement,corticospinal tract and diencephalic involvement. Spinal cord- more longitudinally extensive spinal cord lesion,preferential involvement of the central cord rather than the peripheral cord. Optic nerves -more longitudinally extensive optic neuritiswith preferential involvement of the posterior optic pathway.

ACUTE DISSEMINATED ENCEPHALOMYELITIS

ADEM is a demyelinating CNS disease which mainly affects the pediatric population [(11)].ADEMhas a monophasic and rapidly progressive course [(12)]. Brain lesions in ADEM are determined by perivenular inflammation leading to large areas of demyelination [(13)]. Typical ADEM MRI findings on T2-weighted and FLAIR images appear as bilateral, asymmetrical, multiple, confluent, poorly marginated, hyperintense areas with random distribution (leopard skin regional distribution) [(14)]. ADEM lesions typically involve both central white and deep grey matter[(15)]. Unlike multiple sclerosis, lesions in ADEM do not involve the calloso-septal interface, spare the periventricular white matter and do not present with Dawson's fingers lesions[(16)]. Spinal cord myelitis is seen approximately in one-third of patients, as spinal cord lesions extended for more than two vertebral segments [(17)].

PROGRESSIVE MULTIFOCAL LEUKOENCEPHALOPATHY

PML occurs almost exclusively in immunosuppressed patients, including those with AIDS (79%), hematologic malignancies (13%), organ transplants (5%), and autoimmune diseases on immunosuppressive therapy (3%)

PML can be conceptually organized into early and late stages. Early PML begins as single or multifocal round or oval white matter lesions (WMLs). The lesions are asymmetric in distribution and most commonly are located in the parietal and occipital lobes, as well as the corpus callosum. Involvement of the arcuate or U-fibers forms a sharp or scalloped border between the lesions and the cortex.In late PML with disease progression, the lesions become larger and more confluent and atrophy can be seen. The lesions tend to follow white matter tracts[(18)].

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)

PRESis a condition clinically characterized by headache, altered mental status, seizures, and visual loss and may be associated with systemic hypertension, preeclampsia/eclampsia, chemotherapy, immunosuppressive therapies in the setting of organ transplantation, and uremic encephalopathy.

Typical posterior reversible encephalopathy syndrome manifests as bilateral vasogenicedema within the occipital and parietal regions (70-90% of cases), perhaps relating to the posterior cerebral artery supply. However,it can be found in a non-posterior distribution, mainly in watershed areas, including within the frontal, inferior temporal, cerebellar, and brainstem regions[(19)]

OSMOTIC OR CENTRAL PONTINE MYELINOLYSIS

Osmotic or central pontine myelinolysis is classically described in alcoholics after rapid correction of hyponatremia. Clinical manifestations vary from minimal symptoms to a complete locked-in syndrome, coma, or death. MRI usually shows a centrally located lesion in the pons with sparing of its peripheral rim. Extrapontine structures such as the cerebral white matter, thalamus, and basal ganglia may be involved [(20)].The lesions appear hyperintense on T2-weighted and FLAIR imaging and may show increased signal on diffusion-weighted imaging that is presumably due to cytotoxic edema[(21)].

UREMIC ENCEPHALOPATHY

Uremic encephalopathy, a metabolic disorder that occurs in the context of both acute and chronic renal failure, is a complication resulting from the presence of endogenous uremic toxins in patients with severe renal failure. Uremic encephalopathy has three patterns of imaging findings: basal ganglia involvement (most common), cortical or subcortical involvement (PRES-like), and white matter involvement (caused by ATL). Imaging findings are unspecific, and the patient's clinical history and laboratory findings are indispensable for diagnosis[(22)]. The lentiform fork sign –(T2-weighted and FLAIR images by hyperintensity of the white matter that surrounds the lentiform nuclei delineating the lateral and medial boundaries of both putamina) can be identified in patients with uremic encephalopathy and may be indicative of underlying metabolic acidosis coexisting with uremia[(23)].

V. CONCLUSION

Magnetic resonance imaging (MRI) has become a very important tool in diagnosis and differentiation of different demyelinating disorders. Disease monitoring throughout its course, treatment response and prognosis are often based on the combination of clinical symptoms and neuroimaging findings. MRI is the most commonly used imaging modality as it offers high-resolution images in a noninvasive and safe method, without exposing patients to ionizing radiation.

Conflict of interest:

There is no conflict of interest.

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