Section: Neurology

Original Research Article

MRI IN WHITE MATTER DISEASES OF BRAIN A PRACTICAL APPROACH

R. Vikash Babu[1], M. Rajya Lakshmi[2]

[1]Assistant Professor, Konasema Institute of Medical Sciences, Amalapuram, Andhra Pradesh, India
[2]Assistant Professor, Konasema Institute of Medical Sciences, Amalapuram, Andhra Pradesh, India

Received : 27/07/2025
Received in revised form : 16/09/2025
Accepted : 02/10/2025

Keywords:
White matter, Brain, MS, DAI, PML. CADASIL, ODM

Corresponding Author:
Dr. R. Vikash Babu,
Email: ravikash114@gmail.com

DOI: 10.47009/jamp.2025.7.5.151

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (5); 785-795

ABSTRACT

Background: The aim is to analyze the practical approach of MRI in white matter diseases of adult brain. The objectives are to study the distribution and nature of MRI findings in white matter diseases ofadult brain, to establish an accurate diagnosis and to narrow down the differentialdiagnosis in various white matter diseases. **Materials and Methods:** The present study entitled "Practical approach of MRI in white matterdiseases of brain" was conducted in the Department of Radiology & General medicine, Konasema insistute of medical sciences, amalapuram for a period of 24 months from September 2022 toAugust 2024, after being approved by the Institutional Ethics Committee.It was an observational study involving 50 cases, who were above 14 years ofage with clinical suspicion / diagnosis of white matter lesion, referred to the Department of Radiology for MRI irrespective of sex. Inclusion Criteria is patients with clinical suspicion / diagnosis of white matter lesion referred to MRI with age more than 14 years and of both sexes. Both out patients and in patients were included in the study. Exclusion Criteria is Patients with MRI non-compatible implants intheirbody in any form (pacemaker, orthopedic implants etc. Patients with claustrophobia. Unstable patients on life support mechanism. Patients not willing to give the consent. All the patients with age related vascular causes were not included in the study.**Result:** The present study has been carried out for a period of two years among 50adult patients aged 15 years and above who were referred for MRI to the department of Radiodiagnosis, Konasemainsistute of medical sciences, Amalapuram with clinical suspicion or diagnosis of white matter disease. 62% of the study population belonged to the age group of 15 to 34 years. There after a decreasing trend of the white matter lesions was observed with increase in age. Out of 50, only 2 cases (4%) belonged to age 65 years and above. Out of 50 cases studied, 27 (54%) were females and 23 (46%) were males. Out of the total 50 cases studied, majority of thecases were of ADEM (28%) followed by PRES (20%). 5 cases each of ODM, MS and DAI were seen (10% each). 3 cases (6%) of CADASIL, 2 cases (4%) each of TDM, PML, CTX and MBD were observed. **Conclusion:** MRI due to its excellent gray-white matter resolution is very sensitive in detecting subtle white matter changes.The present study concludes that MRI, in correlation with DWI, MRS,MRcontrast in required cases is an ideal modality in early diagnosis of white matter diseases and aids in the early institution of therapy so that the curable conditions among them can be treated.

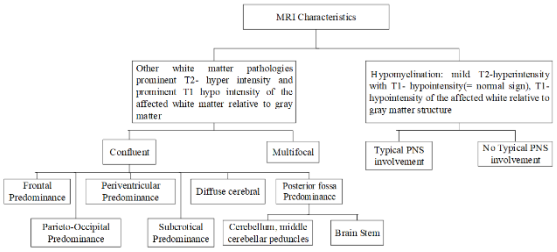
INTRODUCTION

Demyelinating disorders are a heterogeneous group of diseases described as "central white matter disease", in which myelin loss exceeds axonal loss. The result of demyelinating diseases is the thinning or even focal disappearance of the myelin sheath of axons. Such changes will affect signal propagation in affected axons; depending on their location, this canlead to a host of neurologic and psychiatric symptoms.[1]Primary demyelinating disorders, infectious, neoplastic, post-traumatic and metabolic disorders are the most common. When white matter disease is encountered on an imaging study, it is useful to first characterize the white matter involvement as multifocal, confluent / diffuse, or selective (geographic).This approach, combined with the clinical information regarding patient

ISSN (O): 2687-5365; ISSN (P): 2753-6556

demographics, clinical history and physical findings, helps the imager limit the differential diagnosis.If the white matter abnormalities are confluent, the next most helpful MRI discriminator concerns with the identification of predominant localization of the abnormalities. The major preferential localizations are frontal, parieto-occipital, temporal, periventricular, subcortical, diffuse cerebral, and posterior fossa.Special MRI features are typically seen in a number of specific disorders and have a significant diagnostic value.[2]

MRI Characteristics of White matter diseases



The advent of MR has revolutionized the concept of understanding of white matter diseases. MRI is considered far superior to CT and is the imaging modality of choice in white matter diseases. Further, with the advent of multi-echo sequences of MR, even subtle lesions of demyelination can be detected.A correct diagnosis could be made in majority of the patients based on MR findings and clinical history alone. MR, in conjunction with clinical findings, plays a significant role in establishing the diagnosis and in the further follow up of patients with white matterdiseases.[3]

MATERIALS AND METHODS

The present study entitled "Practical approach of MRI in white matter diseases of brain" was conducted from September 2018 to August 2020 after being approved by the Institutional EthicsCommittee.It was an observational study involving 50 cases who were above 14 years of age with clinical suspicion / diagnosis of white matter lesion, referred to the Department of Radiology for MRI, irrespective of sex.

Inclusion Criteria

- Patients with clinical suspicion / diagnosis of white matter lesion referred to MRI with age more than 14 years and of bothsexes.
- Both out patients and in patients were included in thestudy.

Exclusion Criteria

- Patients with MRI non-compatible implants in their body in any form (pace maker, orthopedic implant setc.
- Patients with claustrophobia.
- Unstable patients on life support mechanism.
- Patients not willing to give the consent
- All the patients with age related vascular causes were not included in the study.

Examination Technique

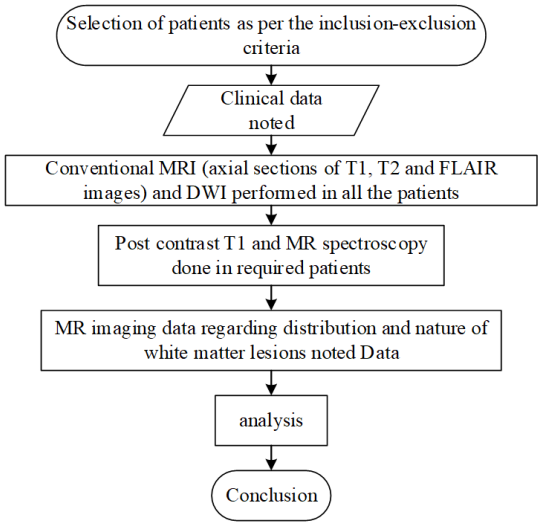
All the MRI sequences were obtained on 1.5 Tesla MRI machine 'GE Signa' 1.5T Signa Excite system (General Electric Medical Systems, Milwaukee, USA). A dedicated eight channel high resolution head coil was used.

Method: After obtaining informed consent, general data regarding age, sex, symptoms, history of present illness, past and personal history, smoking habit, alcohol consumption, etc. were noted.Axial sections of T1, T2 & FLAIR images of MRI were obtained from all the patients. Diffusion weighted MR sequence was also performed in all the patients. Post contrast T1 images and MR spectroscopy were obtained in required patients only.MR imaging data of each patient regarding distribution and nature of the white matter lesions viz., region of involvement, signal characterization, presence or absence of diffusion restriction, presence or absence of contrast enhancement, levels of metabolites in MR spectroscopy, etc. werenoted.

Data Analysis: Statistical analysis of the data was performed by using Microsoft Excel 2007. Data was represented in the form of frequencies and percentages with the help of tables, bar diagrams and pie diagrams.

Reference Citing: Vancouver system of listing and citing of references is used. As per this system, the references are numbered and listed consecutively in the order in which they are first cited in the text.

Brief Procedure



Flow chart of the brief procedure of the study

RESULTS

Thepresentstudyhasbeencarriedoutforaperiodoftwoyearsamong50adult patients aged 15 years and above who were referred for MRI to the department of Radiodiagnosis, Konasema institute of medical sciences, amalapuram with clinical suspicion or diagnosis of white matter disease. 62% of the study

ISSN (O): 2687-5365; ISSN (P): 2753-6556

population belonged to the age group of 15 to 34 years. Thereafter, a trend of the white matter lesions was observed with increase in age. Out of 50, only 2 cases (4%) belonged to the age range of 65 years and above. Out of 50 cases studied, 27 (54%) were females and 23 (46%) were males.

Table 1: Prevalence of white matter lesions (n=50)

Disease	No. of cases	Percentage
ADEM	14	28%
PRES	10	20%
ODM	5	10%
MS	5	10%
DAI	5	10%
CADASIL	3	6%
TDM	2	4%
PML	2	4%
CTX	2	4%
MBD	2	4%
Total	50	100

Out of the total 50 cases studied, majority of the cases were of ADEM (28%) followed by PRES (20%). 5 cases each of ODM, MS and DAI were seen (10% each). 3 cases (6%) of CADASIL, 2 cases (4%) each of TDM, PML, CTX and MBD were observed. In most of the white matter diseases, younger age group is commonly involved except in ODM and CTX. All the 5 cases in ODM belonged to the age group of 45 years and above. Both the cases of observed CTX belonged to middle age (45 to 54 years). There was not much of a difference in sex distribution of overall white matter lesions in the considered study population, 27 females (54%) and 23 males (46%). Among individual diseases, female preponderance was observed in PRES (M:F = 1: 9) and MS (M:F=1:4), while male preponderance was observed in DAI (all the 5 cases were males).

Out of 14 cases of ADEM, 9 cases (64%) belonged to 15 to 34 years age group. Thereafter with increase in age, there was a decrease in the number of cases of ADEM. Out of 14 cases of ADEM, 6 were females (42.9%) and 8 were males (57.1%).

Table 2: Age & Sex Distribution of ADEM Lesions (n=14)

Age In years	FEMALE	MALE	TOTAL
15 to24	2 (50.0%)	2 (50%)	4 (28.6%)
25 to34	3 (60%)	2 (40%)	5 (35.7%)
35 to44	1 (50%)	1 (50%)	2 (14.3%)
45 to54		2 100%)	2 (14.3%)
55 to 64		1 100%)	1 (7.1%)
≥ 65		0	0
TOTAL	6 (42.9%)	8 (57.1%)	14 (100%)

On MRI of the brain, patients with ADEM showed T1 hypointense and T2/FLAIR hyperintense lesions. The predominant site of involvement was the subcortical white matter (85.7%), followed by the brainstem (35.7%) and basal ganglia (35.7%). The thalamus and periventricular white matter were affected in 28.5% of cases each, while the cerebellum was involved in 14.3% of cases. On post-contrast imaging, 4 out of 14 cases (28.5%) demonstrated discrete enhancing lesions.

In cases of Posterior Reversible Encephalopathy Syndrome (PRES), all 10 patients were below 35 years of age. Of these, 9 were females, all with a history of preeclampsia, who presented with high blood pressure. The single male patient was a known case of hypertension and presented with headache, elevated blood pressure, and vision loss.

Table 3: Site of Lesions In ADEM

Site of Lesions	No. Of cases	Percentage (%)
Cerebral White matter (predominantly subcrotical)	12	85.7%
Brain stem	5	35.7%
Basal ganglia	5	35.7%
Thalamus	4	28.5%
Periventricular white matter	4	28.5%
Cerebellum	2	14.3%

Table 4: Site of Lesions in PRES

Site Of Les Ion	No. Of Cases	Percentage (%)
Occipital/parietal	8	80%
Frontal lobe	4	40%
Inferior temporal	4	40%
Cerebellum	4	40%
Brain stem	2	20%

ISSN (O): 2687-5365; ISSN (P): 2753-6556

MR imaging showed T1 (4 cases-isointense, 6 cases-hypointense), T2 and FLAIR hyperintense lesions. The common site involved in PRES was occipital or parietal region (80%). Frontal lobe, inferior temporal region and cerebellum were involved in 40 % cases each. In 20% of the cases, brain stem was involved. On DWI 2 cases, (20%) showeda few areas of restricted diffusion in subcortical regions of both cerebral hemispheres, which appeared isointense on ADC map.All the 5 cases of ODM observed were of 45 years and above. 2 females and3 males. All these cases were end stage renal disease patients on hemodialysis.Pons was involvedin 100% of the cases of ODM. Thalamus and midbrain were involved in 40% of the cases

each.Allthecases(5)ofmultiplesclerosisinourstudysho wedlesionsincorpucallosum.Periventricularregionwa sinvolvedin80%(4)ofthecases.Corticalwhitematter,b rainstem,spinalcordandopticnerveinvolvementwasse eninonecase(20%) each.In 4 cases (80%) lesions have ovoid configuration with the major axes perpendicular to the ventricular surface (Dawson's fingers) which are associated with the inflammatory changes around the long axis of the medullary vein that create the dilated perivenular space.

Diffuse Axonal Injury

Out of 5 cases of DAI, two cases were grade III, two were grade II and one case was grade I. All were males of 25-34 years age group with mean age of 29.2years. All these 5 cases presented with history of polytrauma. Involvement of corpus callosum was seen in 4 out of 5 cases (80%). Brain stem was involved in two cases (40%). Thalamus and basal ganglia were involved in one case (20%) each.

CADASIL

All the 3 cases of CADASIL belonged to 35 to 44 years age group. 2 were females, 1 male. On MR Imaging T2 and FLAIR hyperintense areas were observed in external capsule and anterotemporal white matter in all the 3 (100%) cases. Periventricular and subcortical region was also involved in all the 3 cases (100%). Corana radiata was involved in 2 cases (66.7%), brainstem in 2 cases (66.7%). All the three cases showed subcortical infarcts.

Tumefactive Demyelination

Two cases of Tumefactive demyelination were reported in our study. Both were females (28 and 50 years). Both patients presented with gradual progressive loss of vision with one case having complete vision loss at the time of presentation.MR in both patients showed symmetrical T1hypointense, T2 FLAIR hyperintense lesion involving the periventricular and subcortical whitematter of bilateral posterior temporal, occipital, parietal lobes extending across the splenium of corpus callosum. The lesions were showing no mass effect over the adjacent ventricular horns. On contrast administration, both the cases showed irregular incomplete ring enhancement with open side of the ring towards the cortex.MRS in both the cases showed elevated lactate, reduced NAA, increased choline, elevated glutamate, reduced NAA/Ch ratio. DWI showed restricted diffusion in the periphery of the lesion with hypointense signal in center which appeared as hyperintense center with iso to hypointense periphery on ADC maps.

Progressive Multifocal Leukoencephalopathy: The 2 cases of PML were HIV positive males (32 and 27years). MRI showed well defined lesions appearing hypointense on T1, hyperintense on T2 and FLAIR involving the subcortical white matter of bilateral frontal and right parietal lobes with no mass effect. Basal ganglia and thalamus were involved in one case. On contrast administration no obvious enhancing lesions andno evidence of cortical atrophy were noted. MRS showed reduced NAA, elevated choline, normal creatinine and reduced NAA/Ch ratio. In addition, our patient's CSF was positive for JC virus which supported ourdiagnosis.

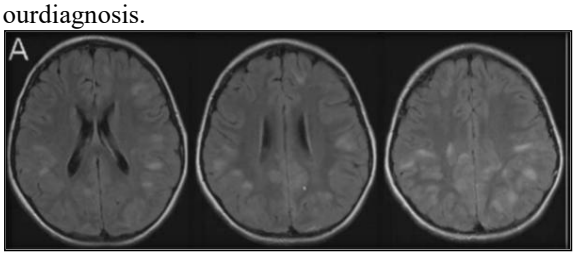


Figure 1: MR Image of ADEM

T2 FLAIR axial images were acquired during the symptomatic phase ADEM.

Typical poorly demarcated lesions with white matter and gray matter involvement.

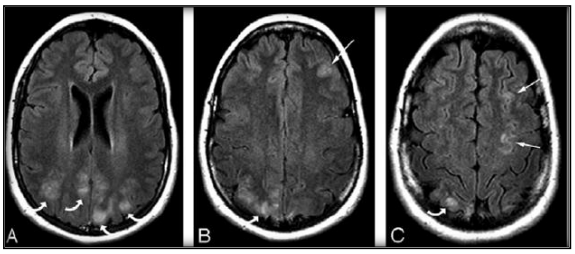


Figure 2: MR imaging of PRESS:Demonstrates patchy vasogenic edema in the parietal region (curved arrows) bilaterally along with linear involvement alongthe superior frontal sulcus on the left.

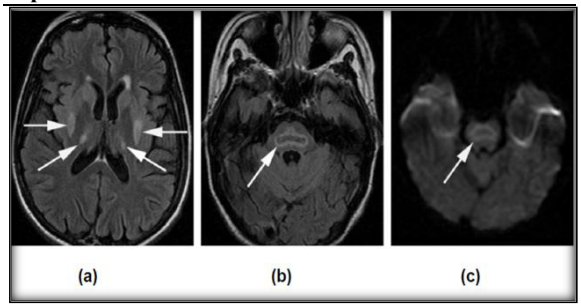


Figure 3: MR Image of Osmotic Demyelination:T2 prolongation in the basal ganglia and lateral thalami (arrows in a) Trident-shaped area of T2 prolongation surrounding an area of encephalomalacia in the central pons (arrow in b).c, Diffusion-weighted MR image show a trident-shaped rimlike area of hyperintensity surrounding the central pons.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Cerebellar Tendon Xanthomatosis: Both the cases of CTX were females belonging to 45 to 54 years age group. Both of them showed symmetrical T2 hyperintensities in bilateral cerebellar hemispheres involving dentate nucleus. Cerebellar foliae are prominent with dilated fourth ventricle. Both the patients presented with cataract and achillis tendon xanthomas.

Marchiafava Bignami Disease: The two cases of MBD observed in our study were chronic alcoholic males of age 36 years and 22 years old. T2 and FLAIR hyperintensities with diffusion restriction noted in body and splenium of corpus callosum in both the cases. With the administration of thiamine, both cases showed improvement in clinicalsymptoms.

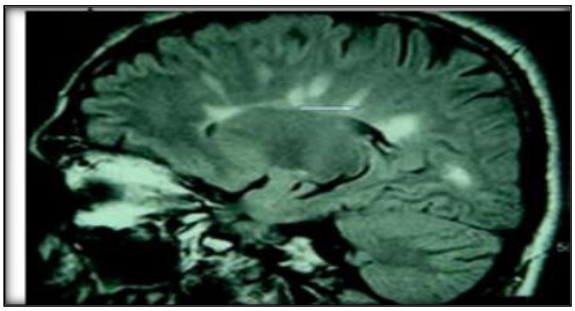


Figure 4: MR Image of Multiple SclerosisFLAIR image shows MS plaques extending up through corpus callosum –Dawson's fingures.

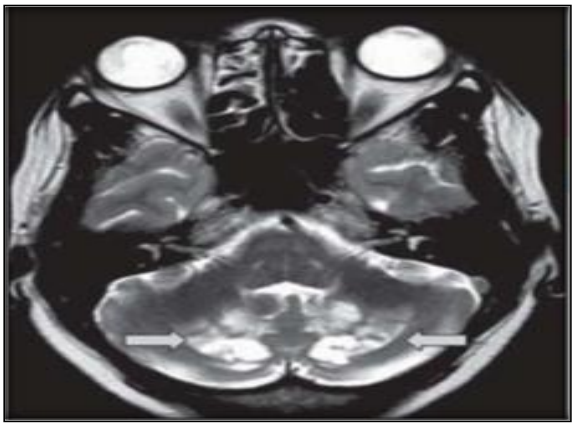


Figure 5: MR brain Image of Cerebrotendinousxanthomatosis T2W axial MRI image of the brain shows bilateral symmetrical hyperintensities (arrows) involving the dentate nuclei and the deep cerebellar white matter.A few hypointense foci (arrowheads) are seen within the hyperintensities.

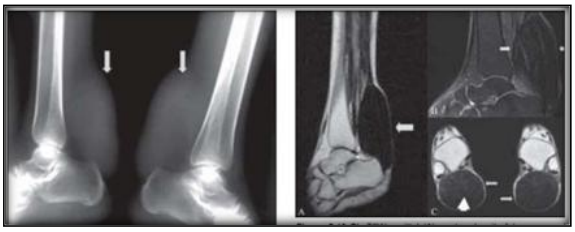


Figure 6: X-Ray and MR ankle image of Cerebrotendinousxanthomatosis Plain lateral radiograph of both ankles showing bilateral symmetrical swellings (arrows) in the posterior aspect of the lower legs,superior to the calcaneum T2W sagittal (A), proton density fat-suppressed sagittal (B) and T1W axial (C) MRI images of the ankles and Achilles tendons show enlarged bilateral Achilles tendon.

DISCUSSION

The present study has been carried out for a period of two years among 50 adult patients aged 15 years with clinical suspicion or diagnosis of white matter disease. 62% of the study population belonged to the age group of 15 to 34 years. There after a decreasing trend of the white matter lesions was observed with increase in age. Out of 50, only 2 cases (4%) belonged to age 65 years and above. Out of 50 cases studied, 27 (54%) were females and 23 (46%) were males. Out of the total 50 cases studied, majority of the cases were of ADEM (28%) followed by PRES (20%). 5 cases each of ODM, MS and DAI were seen (10% each). 3 cases (6%) of CADASIL, 2 cases (4%) each of TDM, PML, CTX and MBD wereobserved.

Acute Disseminated Encephalo Myelitis (Adem): In our study, 14 cases (8 males, 6 females) were diagnosed with ADEM. Allthe 14 cases have history of fever prior to the onset of clinical symptoms. In addition, 4 patients presented with altered sensorium, 2 patients with double vision, 2 patients with3 episodes of seizures. The age group varied through a wide range of 19 to 52 years with male: female ratio of2:1.5.

However,Kesselring J et al,[4] in their study had noted a male female ratio of 1.4:1. In our series, all the cases were adults with mean age of 31 yrs. According to literature, ADEM can occur in all ages, although most reported cases are in children and young adults. However, study done by Schwartz S et al on occurrence ofADEMin adult patients consisting of 40 cases showed the mean age as 33.5 years which is comparable with our result.In our study on MR imaging, T1 hypointense, T2 and FLAIR hyperintense lesions were noted, of which majority were located in subcortical white matter of both cerebral hemispheres (12/14 cases-85.7%), followed by brain stem (5/14cases- 35.7%), cerebellar white matter (2/14 cases-14.3%). Brain stem and /or cerebellum involvement in 43% of cases.[5]

On comparison,R.C. Dale et al5showed that involvement of the deep and subcortical white

ISSN (O): 2687-5365; ISSN (P): 2753-6556

matter was nearly universal (91%), brainstem and /or cerebellum were involved in 87% of cases. The thalami and /or basal ganglia were involved in 69% of cases. Periventricular region was involved in 44%None of the patients showed involvement of spinal cord.In Mikealoff et al study,[6] cerebellum and /or brain stem were involved in 68%, thalamus and/ or basal ganglia were involved in 63%, juxtacortical region was involved in 66%of cases.In Linn et al study,[7] cerebellum and brain stem were involved in 77%, thalamus and basal ganglia were involved in 62%, periventricular region was involved in 60%, cortical region was involved in 43%of cases.In our study,Thalamic involvement was seen in 4 out of 14 cases (28.5%) and basal ganglia was involved in 5 out of 14 cases (35.7%), thalamus and /or basal ganglia in 50%ofcases.Our findings were consistent with the study done by Baum PA etal,[8]which showed that thalamic involvement is reported to be rare in multiple sclerosis, and may prove useful in distinguishing between ADEM and the initial presentation of multiple sclerosis.On contrast administration, out of 14 cases,4cases (28.5%) showed discrete enhancing lesions with one case showing incomplete ring type of configuration.These findings were comparable with the observations done by Van der knapp et al,[9] and Nathan P Young et a,[10] which showed the white and/or gray matter lesions may enhance, but usually not all lesions enhance, and contrary to what is usually stated, their experience is that in many cases enhancement is at most subtle or is not present atall.

Table 5: ADEM- Comparison of Present Study with Existing Studies

Finding	RC daleetal	Mikealoffet al	Linn etal	Prevalenceinpresentstudy
Sub cortical whitematter	91%	66%	43%	85.3%
Tand /orBG	69%	63%	62%	50%
BS and /or CB	87%	68%	77%	43%
Periventricular	44%	-	60%	28.5%

*T-Thalamus,BG-Basal ganglia, BS-Brain stem, CB –Cerebellum.

Posterior Reversible Encephalopathy Syndrome (Hypertensive Encephalopathy)

Inour study, we reported 10 cases of PRES of which 9 were female and 1 male with a sex ratio of male to female 1: 9. Of the 10 cases, 9 were known case of preeclampsia and presented with high blood pressures. One male who was a known case of hypertensive presented with head ache, high B.P and visionloss.MR imaging showed T1 (4 cases-isointense, 6 cases-hypointense) T2 FLAIR hyperintense lesions affecting the subcortical white matter of bilateral parietooccipital lobes (8/10 cases i.e. 80%), temporal lobes (4/10 cases i.e. 40%), frontal lobes (4/10 cases i.e.40%), brainstem (2/10 cases i.e. 20%) and cerebellum (4/10 cases i.e. 40%).These findings were comparable with study done by Donmez FY et al,[11]who reported that the most commonly involved localizations in PRES were parietal lobe in 84.8%, occipital lobe in 72.7%, frontal lobe in 51.5%, temporal lobe in 33.3%, and cerebellum in 33.3%. Chou MC et al also suggested that involvement of anterior circulation region, brainstem, cerebellum, deep cerebral white matter, and thalamus are common in PRES.[12-19] Our findings are also comparable with the study done by W.S. Bartynskietal,[18] in which out of 136 patients, Vasogenic edema was consistently present in the parietal or occipital regions (98%), but other locations were common including the frontal lobes (68%), inferior temporal lobes (40%), and cerebellar hemispheres (30%). Involvement of the basal ganglia (14%), brain stem (13%), and deep white matter (18%) including the splenium (10%) was notrare.On DWI usually PRES does not show restricted diffusion, however in our series 2 case, 20% showed few areas of restricted diffusion in subcortical regions of both cerebral hemispheres which appeared isointense on ADC map.This atypical finding is comparable with study done by McKinney AM et al,[12]who reported that restricted diffusion was the second most common atypical presentation of PRES in their study, accounting for 17.3%. The findings were also consistent with C J Stevens et al who stated that restricted diffusion as an associated finding has been described in PRES and has been shown to be potentially reversible.

Table 6: Posterior Reversible Encephalopathy Syndrome Comparison of Present Study with Existing Studies

Finding	Donmez FY etal study	W.S. Bartynskiet al	Prevalenceinpresentstudy
Sub cortical whitematter of bilateral parietooccipital regions	Parietal(84.8%) & occipital(72.7%)	98%	80%
Temporal lobes	33.3%	40%	40%
Frontal	51%	68%	40%
Cerebellum	33.3%	32%	40%

Osmotic Demyelination Syndrome

In the present study, 5 cases of osmotic demyelination syndrome were observed. All these five were end stage renal disease patients on hemodialysis. All the 5 (100%) cases showed T1 hypo to isointense and FLAIR hyperintensity on central pons. This is in favor of osmotic demyelination syndrome with demyelination or

ISSN (O): 2687-5365; ISSN (P): 2753-6556

transient edema in pons. On comparison, N. CaglaTarhan et al,[13] study showed hyperintensity in central pons in 65% (11 out of 17) patients.

In our study, thalamus and midbrain were involved in two cases(40%) each.Threecases(60%) show diffusion restriction on DWI. These findings are almost comparable to the findings of Jonathan Graff-Radford etal,[14] in which pons was involved in all 22 patients (100%), followed by the thalamus (n=8 [36%]), midbrain (n=6 [27%]), cortical gray matter (n=3 [14%]), hippocampus (n=3 [14%]), caudate (n=2 [9%]), putamen (n=2 [9%]), and middle cerebral peduncle (n=2 [9%]). Of 19 cases in which diffusion-weighted sequences were obtained, 10 (53%) demonstrated evidence of restricted diffusion.In RameshaNekkareKallakatta etal,[15] study of 25 patients with osmotic demyelination, Pons was involved in 19 patients (76%) Caudate 18 (72%)Putamen19 (76%) Thalamus 5 (20%)Midbrain 4 (16%) Cortical grey mater (frontal and insular) 3(12%) Extratemporal cortical whitemater 3 (12%) Hippocampus 2 (8%) Cerebellum 2 (8%), Medulla 1 (4%) and Subthalamic nuclei 1 (4%).

Table 7: Osmotic demyelination syndrome: comparision of present study with existing studies

Finding	Jonathan Graff-Radfordetal	RameshaNekkareKallakatta etal	Prevalence in present study
Pons	100%	76%	100%
Thalamus	36%	20%	40%
Mid brain	27%	16%	40%
Diffusion restriction	53%		60%

MULTIPLESCLEROSIS

In our study,cases of multiple sclerosis noted, were between 21-44 years of age. Mean age of onset is 31.4years. A distinct female preponderance was noted in our study group, with 4 females and 1 male (male: female = 1:4). All the above results were comparable with the studies done by BN Lakhkar et al and Gangopadhyay G et al.In our study, MR imaging showed multiple discrete lesions appearing hyperintense on T2 and FLAIR images. Corpus callosum was involved in all five cases which was consistent with a prospective study done by Yulin Ge et al,[11] who found that most of MS patients demonstrated confluent / focal lesions involving the calloso-septal interface and concluded that callosal involvement was specific for MS and also useful in assessing the relapse rates.But in BN Lakhkar et al study, out of 15 patients only one patient had callosal atrophy. Involvement of periventricular white matter was seen in 4 out of 5 cases (80%). Brainstem, spinal cord and optic nerve were involved in one case (20%) each. These findings were similar to the results of studydone by BN Lakhkar et al,[3] which showed periventricular involvement in 80%, brain stem in 40%, spinal cord and optic nerve in 20% each.

InJoost C. J. Bot, MD etal,[17] study done on 25 cases of multiple sclerosis, all the 25 cases showed periventricular Involvement. Juxtacortical lesions were present in 80% of patients. Infratentorial lesions were present in 84% of patients. Deep white matter lesions were present in 96% of patients. Enhancing focal lesions were observed 32% ofpatients.While in our study, in four cases(80%),lesions have ovoid configuration with the major axes perpendicular to the ventricular surface (Dawson's fingers) which are associated with the inflammatory changes around the long axis of the medullary vein that create the dilated perivenular space which is consistent with the findings of BN Lakhkar et al and Yulin Ge et al.[3,18]

Diffuse Axonal Injury

In our study, 5 cases of DAI were observed, of which two cases were grade III, two were grade II and one case was grade I. All were males of 25-34 years age group with mean age of 29.2years. These findings are comparable to Pamela W. Schaefer et al, in which mean age is 25.2 years with a male and female ratio of 2.25. In present study, involvement of corpus callosum was seen in 4 out of 5 cases (80%). Brain stem was involved in two cases (40%). Thalamus and/or basal ganglia were involved in two cases (40%). These findings arecomparablewith Pamela W. Schaefer et al,[19] in which 12 (46%) patients had lesions in the brainstem, 12 (46%) had lesions within the basal ganglia and/or thalamus, and 16 (61%) had injuries of the corpuscallosum.Compared to Ezaki Y et al,[20] study done, among the 21 patients of DAI, grey white matter interface and central white matter involvement in 50%, Thalamus / basal ganglia involvement in 12% corpus callosum involvement in 9% and brainstem involvement in 18%.Compared to M Takaoka et al,[21]study done, among 21 patients of DAI, involvement of corpus callosum is seen in all patients and brain stem is involved in 8 cases(38%).

Table 8: Multiple sclerosis comparision of present study with existing studies

Finding	BN Lakhkar et al study	Joost C. J. Bot, MD etal	Prevalence in present study
Periventricular white matter (with Dawsons fingers)	80	100	80
Deep white matter (Brain stem)	40	96	20
Spinalcord	20	-	20
Opticnerve	20	-	20
Juxtracortical lesions	-	80	20

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 9: diffuse axonal injury comparision of present study with existing studies

Finding	Pamela W. Schaefer et al	Ezaki Y et al	M Takaoka et al study	Prevalence in present study
Corpus callosum	61	9%	100%	80%
Brain stem	46	-	38%	40%
Thalamus and/or basal ganglia	46	12%	-	40%
Grey white matter interface	-	50%	-	80%

CADASIL: In the present study, 3 cases (one male and 2 females) of CADASIL belonged to the age group of 35 to 44 years were observed. On MR Imaging, T2 and Flair hyperintense areas were observed in external capsule and anterotemporal white matter in all the 3 (100%) cases. Periventricular and subcortical region was also involved in all the 3 cases (100%). Other regions involved were corana radiata in 2 cases (66.7%), brainstem in 2 cases (66.7%). All the three cases showed subcortical infarcts.

On comparison, in Dorothee P. Auer et al study,[22] out of 28 patients on MR Imaging, all but two of the 28 patients showed involvement of temporopolar WM. Further, lesions were seen in the basal ganglia in 24 patients (85.7%), in the brainstem in 25 (89.3%), in the thalamus in 25 (89.3%), in the cerebellum in 13 (46.4%), and in the cortex in two (7.1%).Compared to Vandenboom etal,[23]study done on 24 patients of CADASIL,Subcortical lacunar infarcts in 9 patients (37 %),Lacunar infarcts 15 (62.5%),Microbleeds (n) 4,Anterior temporal lobe 23(95%),External capsule (n) 14 (58%) and Internal capsule (n) 15.

Table 10: CADASIL: comparision of present study with existing studies

FINDING	Dorothee P. Auer et alStudy	Vandenboom etal study	Prevalence in present study
Bilateral anterior temporal white matter	100%	95%	100%
External capsule	-	58	100%
Brain stem	89.3%		66%
Subcorticallacunar infarcts	-	37%	100%

Tumefactive Demyelination

We reported 2 cases of Tumefactive demyelination in our study series. Both cases were females (28 and 50 years) with average age of onset being 39 years which was comparable with D. Shah et al,[24] who stated that these lesions occur more frequently in women in the second and third decades of life, with an average age of onset of at least 37 years. Both patients presented with gradual progressive loss of vision with one case having complete vision loss at the time of presentation.MR in both patients showed symmetrical T1 hypointense, T2 FLAIR hyperintense lesion involving the periventricular and subcortical white matter of bilateral posterior temporal, occipital, parietal lobes extending across the splenium of corpus callosum. The lesions were showing no mass effect over the adjacent ventricular horns. On contrast administration, 2 cases showed irregular incomplete ring enhancement with open side of the ring towards the cortex.

In a study by Curtis A Given et al,[25]approximately half of tumefactive demyelinating lesions have pathologic contrast enhancement, usually in the form of ring enhancement and commonly the enhancement pattern will be in the form of an open ring, with the incomplete portion of the ring on the gray matter side of the lesion.Compared to DaeSik Kim et al,[26] who studied the enhancement patterns in the 15 patients with TDL reported incomplete rim enhancement was noted in 83%, Mixed T2 iso and hyper intensity of enhancing components in 83%, Absence ofmass effect was noted in 79%, absence of cortical involvement was noted in 76% MRS in both the cases showed elevated lactate, reduced NAA, increased choline, elevated glutamate, reduced NAA/Ch ratio. These findings were consistent with the study done by Curtis A Given et al,[25]who reported a characteristic spectrum consisting of elevated choline with suppressed levels of N-acetyl aspartate. Additionally,theremaybedetectablelevelsoflipidsandl actatecorrespondingtonecrosis and anaerobic metabolism mimicking a neoplastic process, However these lesions can be differentiated from neoplastic lesions with demonstarion of glutamate peak which was noted in our cases consistent with the findings of A. Cianfoni et al,[27]study, who reported that an abnormal elevation of the glutamate/glutamine (2.1–2.5 ppm) peaks is the more critical MR spectroscopy finding in tumefactive lesions in the brain, a finding that is not usually seen in the confounding aggressive intra-axial neoplasms.

DWI in both cases showed restricted diffusion in the periphery of the lesion with hypointense signal in center which appeared as hyperintense center with iso to hypointense periphery on ADC maps. These findings were consistent with the study done by C.H Toh et al,[28]who reported restricted diffusion in the lesion periphery was present in 7 of 8 tumefactive demyelinating lesions (87.5%) in their study group.

Progressive Multifocal Leukoencephalopathy

The present study included 2 cases of PML who are HIV positive males (32 and 27 years).Krupp LB et

ISSN (O): 2687-5365; ISSN (P): 2753-6556

al,[29]reported that PML has a stronger association with AIDS than with any immunosuppressive disease and 55% to 85% of recent PML cases are attributable to AIDS.

The younger age of onset was consistent with the study done by Giesen VHJ et al,[30]on HIV patients with PML which stated that the mean age of onset was 29 years.They also opined that the mean age of onset in HIV positive cases were significantly lower than HIV sero negative PML and male patients prevailed.

Table 11: Tumefactive Demyelination - Comparison with Existing Studies

Finding	CurtisA Given et al study	C.H Toh et al study	DaeSik Kim et al	Prevalence in present study
Incompleterim enhancement	50		83%	100
Absence of Mass effect	-	-	83%	100
Diffusion	-	87.5%	-	100

MR showed well defined lesions appearing hypointense on T1, hyperintense on T2 and FLAIR involving the subcortical white matter of bilateral frontal and right parietal lobes with no mass effect. On contrast administration, no obvious enhancing lesions were noted and no evidence of cortical atrophy. These findings correlated with the diagnostic criteria proposed by Giesen VHJ et al.[30]

MRS showed reduced NAA, elevated choline, normal creatinine and reduced NAA/Ch ratio. These findings were comparable with Iranzo A et al,[31]and Chang Let al,[32] which showed PML lesions to be characterized by significantly reduced NAA, significantly increased Cho compared with control group values. DWI showed restricted diffusion in the lesion involving subcortical white matter of right frontal lobe which appeared isointense on ADC map. These findings are comparable withBerguiM et al,[33]study which showed newer lesions and the advancing edge of large lesions had normal-to-low ADC and gave high signal on DWI. High signal on DWI and low ADC mark the regions of active infection and cell swelling, distinguishing them from areas of reparativegliosis.The MR, MRS and DWI findings in correlation with patient's HIV status were suggestive of progressive multifocal leukoencephalopathy. In addition, our patient's CSF was positive for JC virus which supported our diagnosis.

CerebrotendinoXanthomatosis

The present study includes 2 cases of CTX. Both the patients showed symmetrical T2 hyper intensities in bilateral cerebellar hemispheres involving dentate nucleus. Cerebellar foliae are prominent with dilated fourth ventricle. Symmetrical T2 hyperintensities are noted in parieto-occipital subcortical white matter& internal capsule.Both The Patient Presented With Cataract and achilles tendon xanthomas.

These findings are consistent with Frederik Barkhof et al,[34] study done on 24 CTX patientsin which cerebellum was affected in most patients (84%) Cerebellar involvement typically started in the dentate nucleus. The Achilles tendons were affected in seven patients .63% cases showed pyramidal tract involvement.

Marchiafava Bignami Disease

In our study, two cases of MBD were observed. Both were chronic alcoholic males of age 36 years and 22years old. T2 and FLAIR hyperintensities with diffusion restriction is noted in body and splenium of corpus callosum in both the cases. These findings are consistent with Lee SH etalMénégonetal.[35,36]

CONCLUSION

The present study has been carried out for a period of 2 years among 50 adult patients aged 15 years and above who were referred for MRI to the department of Radiodiagnosis, NRI Academy of Medical Sciences, Chinakakani with clinical suspicion or diagnosis of white matter disease.

- Majority (62%) of the study population belonged to the age group of 15 to 34 years.
- Prevalence of the overall white matter lesions does not show significant sex difference (54% females vs. 46% weremales).
- The commonest white matter disease in our study population was found to be ADEM (14 cases) followed by PRESS (10 cases) and then multiple sclerosis (5), osmotic demyelination (5) and Diffuse axon injury(5).
- • Most of the cases of ADEM presented with fever and neurological deficits and were constituted by adults with predominant lesions in subcortical white matter showing asymmetric distribution. Deep grey nuclei involvement was seen more commonly in ADEM than in MS. DWI showed restricted diffusion in 2cases.
- Cases of PRES showed female preponderance with history of post-partumseizues. Predominant lesions affecting the subcortical white matter of bilateral parieto-occipital lobes with normal metabolite levels were observed and normal diffusion was present in most of thecases.
- The 5 cases of ODM in our study group were adults on dialysis with electrolyte derangement. MRI showed pontine demyelination and involvement of thalamus andmidbrain.
- Cases of DAI were all males with history of polytrauma with predominant lesions in corpus callosum, grey white matter interface, deep whitematter.
- Multiple sclerosis in our study had a distinct female preponderance affecting patients in second to fourth decade with commonest site of

ISSN (O): 2687-5365; ISSN (P): 2753-6556

lesions being periventricular white matter and corpus callosum with various enhancing patterns. DWI showed areas of restricted diffusion in few enhancingplaques.

- The 3 cases of CADASIL observed in our study presented with dementia and focal neurological deficits. MRI showed bilateral symmetrical hyperintensities of temporal white matter, subcortical lacunar infarcts.
- The 2 cases of PML were HIV positive males. MRI showed involvement of the subcortical white matter. On contrast administration, no obvious enhancing lesions were noted. MRS showed reduced NAA, elevated choline, normal creatinine and reduced NAA/Chratio.
- The 2 cases of Tumefactive demyelination are females. MRI showed lesions involving the periventricular and subcortical white matter of bilateral posterior temporal, occipital lobes with incomplete ring enhancement. MRS showed elevated lactate, reduced NAA, increased choline, elevated glutamate, reduced NAA/Ch ratio. DWI in both cases showed restricteddiffusion.
- Both the cases of Cerebellar tendon xanthomatosis were females of same family with tendon xanthomas and MRI shows hyperintensity in cerebellum and dentatenucleus.
- 2 cases of marchia fava bignami disease in our study group were chronic alchoholic males with improvement of symptoms on thiamine administration. Both of themshowed involvement of hyperintensities in body and splenium of corpus callosum.

Conclusion

MRI due to its excellent gray-white matter resolution is very sensitive in detecting subtle white matter changes.The present study concludes that MRI, in correlation with DWI, MRS,MR contrast in required cases is an ideal modality in early diagnosis of white matter diseases and aids in the early institution of therapy so that the curable conditions among them can be treated.

REFERENCES

1. Grossman RI, Yousem DM (2003) Neuroradiology–therequisites, 2ndedn.Mosby, Philadelphia.
2. Blake A. Johnson. Practical approach to white matter diseases. Advanced MRI 2002- from head totoe.
3. Lakhkar BN, Aggarwal M, John JR. MRI in white matter diseases - Clinico radiological correlation. Indian J Radiol Imaging [serial online] 2002 [cited 2014 Oct 12];12:43-50.
4. Kesselring J, Miller DH, RobbSA, Kendall BE, Moseley IF, KingsleyDetal. Acutedisseminated encephalomyelitis — MR findings and the distinction from multiplesclerosis. Brain 1990; 113:291-302.
5. R.C.Dale et al. Acute disseminated encephalomyelitis, multiphasic disseminated encephalomyelitis and multiple sclerosis in children, Brain dec 2000; 123(12)2407-2422.
6. Mikaeloff Y etal Acute disseminated encephalomyelitis cohort study: prognostic factors for relapse European journal of paediatric neurology 2007;11(2) :90-95.
7. Lin CH etal acute disseminated encephalomyelitis: a follow up study in Taiwan.J.Neurol. Neurosurg psychiatry2007;78(2):162-167
8. Baum PA, Barkovich AJ, Koch TK, Berg BO (1994) Deep gray matter involvement inchildren with acute disseminated encephalomyelitis. AJNR Am J Neuroradiol1994.
9. Marjo S. van der Knaap, Jaap Valk-Textbook of Magnetic Resonance of Myelination and Myelin Disorders, ThirdEdition.
10. Nathan P. Young et al. Acute Disseminated Encephalomyelitis-Current Understanding and Controversies. Semin Neurol. 2008Feb;28(1):84-94.
11. Donmez FY et al. MRI features of posterior reversible encephalopathy syndrome in 33 patients. J Neuroimaging. 2010Jan;20(1):22-8.
12. McKinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, SantaCruz KS, Teksam M, Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings,AJR Am J Roentgenol. 2007 Oct; 189 (4):904-12.
13. N. CaglaTarhan A. MuhtesemAgildere U. Sibel Benli F. NurhanOzdemirCuneytAytekinUfuk Can. Osmotic Demyelination Syndrome in End-Stage Renal Disease After Recent Hemodialysis: MRI of the Brain. AJR 2004;182:809–816.
14. Jonathan Graff-Radford, MD et al Clinical and Radiologic Correlations of Central PontineMyelinolysis Syndrome. Mayo Clin2011;86(11):1063-1067.
15. Ramesh nekkare et al Clinical and functional outcome and factors predictive prognosis in osmotic demyelination. neurology, neurosurgery, psychiatry 2011;82:326-331.
16. Gangopadhyay G,DasSK,SardaP,SahaSP,GangopadhyayPK,Roy TN etal.Clinical profile of multiple sclerosis in Bengal. Neurology India 1999; 47:18-21.
17. Joost C. J. Bot, MD Differentiation of Multiple Sclerosis from Other Inflammatory Disorders and Cerebrovascular Disease: Value of Spinal MR Imaging1 Radiology 2002; volume223(1):46–56.
18. Yulin Ge et al. Multiple Sclerosis: The Role of MR Imaging, AJNR June 2006 27:1165-1176.
19. Pamela W. Schaefer et al. Diffusion-weighted MR Imaging in Closed Head Injury: High Correlation with Initial Glasgow Coma Scale Score and Score on ModifiedRankinScaleatDischarge.RadiologyOctober2004;233(1):58–66.
20. Y Ezakietal role of diffusion weighted magnetic resonance imaging in diffuse axonal injury acta radiol47(7),733-740.
21. M.Takaokaetal. Semiquantitative analysis of corpus callosum injury using magnetic resonance imaging indicates clinical severity in patients with diffuse axonal injury. Jneurol Neurosurgery psychiatry2002;73:289-293.
22. Dorothee P. Auer et al Differential Lesion Patterns in CADASIL and Sporadic Subcortical Arteriosclerotic Encephalopathy: MR Imaging Study with Statistical Parametric Group Comparison Radiology 2001;218:443–451.
23. Vanden boom R, Lesnikoberstein SA, Vanduien SG et al. Subcortical lacunar lesions: an MR imaging finding in patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy radiology2002:791-796.
24. Shah D, Patil A, Patil P, Bhatnagar S, Kulkarni V. Tumefactive demyelination mimicking neoplasm. Med J DY Patil Univ2014;7:806-8.
25. Curtis A. Given et al, The MRI Appearance of Tumefactive Demyelinating Lesions. AJR. January2004;182.
26. DaeSik Kim et al, Distinguishing Tumefactive Demyelinating Lesions from Glioma or Central Nervous System Lymphoma: Added Value of Unenhanced CT Compared with Conventional Contrast-enhanced MR Imaging Radiology: Volume 251: Number2—May2009.
27. A. Cianfoni et al. Metabolite Findings in Tumefactive Demyelinating Lesions Utilizing Short Echo Time Proton Magnetic Resonance Spectroscopy. AJNR February 2007; 28:272-277.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

28. C.H Toh Differentiation of Tumefactive Demyelinating Lesions from High- Grade Gliomas with the Use of Diffusion Tensor Imaging. AJNR 2012; 33: 846-851.
29. Krupp LB,Lipton RB, Swerdlow ML. Progressive multifocal leukoencephalopathy: Clinical and radiographic features. Ann Neurol 1985; 17:344-349.
30. Giesen VHJ, Jacob NE, Dorries K, Jablonowski H, Roick H, Arendt G. Diagnostic criteria and clinical procedures in HIV-1 associated progressive multifocal lukoencephalopathy. J Neurol Sci 1997; 47(1):63-72.
31. Iranzo A et al. Proton magnetic resonance spectroscopy pattern of progressive multifocal leukoencephalopathy. J NeurolNeurosurg Psychiatry. 1999 Apr; 66(4):520-3.
32. Chang L et al. Metabolite abnormalities in progressive multifocal leukoencephalopathy by proton magnetic resonance spectroscopy. Neurology. 1997Apr;48(4):836-45.
33. Bergui M et al. Progressive multifocal leukoencephalopathy: diffusion- weighted Imaging and pathological correlations. Neuroradiology. 2004 Jan;46(1):22-5. E pub 2003 Oct31.
34. Barkhof F, Verrips A, Wesseling P, Van Der Knaap MS. Cerebrotendinousxanthomatosis: The spectrum of imaging findings and the correlation with neuropathologic findings. Radiology2000;217:869-76.
35. LeeSH,KimSS,KimSH,etal. Acute marchiafava-Bignami disease with selective involvement of the precentral cortex and splenium. The neurologist 2011;17:213- 7.(PubMed).
36. MenegonP,SibinI,PacgaiC,etal.Marchiafava-Bignami disease: diffusion – Weighted MR in corpus callosum and cortical lesions. Neurology. 2005;65:475-7 (PubMed).

ISSN (O): 2687-5365; ISSN (P): 2753-6556