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Original Article

Magnetic Resonance Study of White Matter Lesions: A Cross-sectional Study

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Abstract

Background: Magnetic resonance imaging (MRI) is an important noninvasive imaging modality with very high sensitivity for detecting white matter lesions. The objectives are to evaluate the role of MRI in white matter diseases, to establish an accurate diagnosis. To assess the severity and extent of the underlying lesion in various conditions and to demonstrate the different patterns of abnormal myelination in white matter diseases.

Materials and Methods: A total of 50 patients who were clinically suspected of white matter diseases underwent MR imaging in using 1.5T PHILIPS Achieva machine. Various MR sequences were used in imaging.

Results: Among the 50 patients, 26% had leukodystrophies, 16% had acute disseminated encephalomyelitis (16%) and 16% had vacuolating leukoencephalopathies, 12% had hypomyelination, 8% had multiple sclerosis and microvascular ischemic changes (SVIC), 4% each of Leigh's disease, human immunodeficiency virus encephalopathy, nonspecific demyelination, and 2% had progressive multifocal leukoencephalopathy. Patients in the pediatric age group constituted 82% of the total study group with slight male predominance.

Conclusion: MR imaging has become the primary imaging modality in patients with white matter diseases and plays an important role in the identification, localization, and characterization of underlying white matter abnormalities in affected patients. Its multiplanar imaging capability and excellent gray-white matter resolution make MRI very sensitive in detecting subtle white matter lesions. In our study, we observed that Fluid attenuated inversion recovery sequence has better sensitivity for white matter lesions especially those in periventricular locations. Specificity of MR imaging can be improved by MR Spectroscopy studies.

Key words: Leukodystrophies, Leukoencephalopathy, Magnetic resonance imaging, White matter diseases

INTRODUCTION

Only recently with the advances in imaging technology have white matter diseases of central nervous system been extensively studied and understood. Computed tomography (CT) and magnetic resonance imaging (MRI) are the imaging modalities which are currently available for the investigation of these diseases and it has been proven beyond doubt that MRI is far superior to CT and the imaging modality of the choice in these diseases.

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Functional MRI of brain was introduced by Oqawa *et al*., in 1992, and developed by Rosen *et al*. In 2003, Paul Lauterbur and Sir Peter Mansfield awarded jointly Nobel Prize for their discoveries concerning MRI.[1] MRI has a very high sensitivity for detecting white matter lesions due to its excellent gray-white matter resolution. Multiplanar imaging is possible only with MRI, which helps in the detection and localization of lesions. It is also found to be ideal in posterior fossa imaging and allows simultaneous imaging of extracerebral sites such as spinal cord and optic nerve. MRI thus is very helpful in defining the pathogenesis and in the early diagnosis of disease and in monitoring the treatment. Recent advances such as MR spectroscopy (MRS), diffusion imaging, and magnetization transfer imaging have revolutionized the role of MRI in increasing the specificity of diagnosis in many of these conditions. By correlating the clinical features and biochemical analysis with the MRI findings, we can come to a diagnosis in the majority of cases. Many of the diseases if detected early

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are reversible, and thus, play the major role of MRI in the early diagnosis so that the treatable conditions among them can be detected early and cured.

Ashikaga *et al*. evaluated cases of HSE with conventional SE and fluid attenuated inversion recovery (FLAIR) sequences and opined that the lesion conspicuity in the gray and white matter was found to be greatest with FLAIR sequence.[2]

This study is selected because of the important role of MRI in investigating white matter diseases and also to evaluate the data obtained from cranial MRI in these diseases.

The objectives of the study were to evaluate the role of MRI in white matter diseases, to assess the severity and extent of the underlying lesion in various conditions of white matter diseases and to demonstrate the different patterns of abnormal myelination in white matter diseases.

MATERIALS AND METHODS

The source of data for the study is patients from Navodaya Medical College Hospital and Research Centre. All patients referred to the Department of Radio Diagnosis with clinical history suspicious of white matter diseases and the patients with incidental finding of white matter diseases/lesions in all age groups in a period of 1-year from October 2015 to September 2016 were included in the study. The patients with clinical suspicion of post-traumatic white matter injury, intracranial tumors and metastatic disease, history of claustrophobia and history of metallic implants insertion, cardiac pacemakers, and metallic foreign body *in situ* were excluded from the study. A total of 50 patients were included.

Imaging was done with 1.5 Tesla PHILIPS Achieva machine using sense head coils. Sequences like localizer sequence conventional spin echo, sagittal FLAIR, short time inversion recovery, T1 FS, axial and sagittal T1-images, axial, sagittal and coronal T2-images, proton density images, diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) map and axial gray matter only and white matter only sequences were used for imaging purpose. Other optional sequences like diffusion tensor imaging and fiber tracking; IV contrast study; time of flight (TOF) angiography were included in the study as and when required. MRI findings were correlated with biochemical parameters (for metabolites) where feasible to increase the accuracy of diagnosis.

A circulatory head coil was used for excitation and signal reception. Standard head coil was used for the acquisition of images. The MR imaging protocol is part of our routine protocol and comprises T2-weighted (T2W) (axial and coronal), T1-weighted (T1W) (axial), FLAIR (axial), DWI (axial), gradient recalled echo (axial), susceptibility weighted imaging (axial) and magnetic resonance angiography (TOF-circle of Willis and neck). The total imaging time with 1.5 T-whole body MR imager was approximately 20 min 53 s. After localizer images were obtained, the axial images were titled parallel to the corpus callosum. The sagittal images were planned on the axial sections. The coronal images were planned on the sagittal/axial sections. For PD + T2W axial imaging, we used a turbo spin echo sequence with a repetition time (TR) of 3380 ms; echo time (TE) of 14,86 ms; 5 mm section thickness; 30 mm intersection gap; 150 flip angle; 230 nm field of view; 512 × 512 matrix and imaging time of 3 min 7 s. For T1W axial imaging, we used a turbo spin echo sequence with a repetition time (TR) of 500 ms, echo time (TE) of 8.1 ms, 5 mm section thickness, 30 mm intersection gap, 90 flip angle; 230 mm field of view, 512 × 512 matrix and imaging time of 3 min 55 s. For FLAIR axial imaging, we used a turbo spin echo sequence with a repetition time (TR) of 9000 ms, echo time (TE) of 87 ms, 5 mm section thickness, 30 mm intersection gap, 150 flip angle, 230 mm field of view, 512 × 512 matrix and imaging time of 4 min 32 s. For T2W coronal imaging, we used a turbo spin echo sequence with a repetition time (TR) of 3850 ms, echo time (TE) of 114 ms, 5 mm section thickness, 30 mm intersection gap, 150 flip angle, 210 mm field of view, 512 × 512 matrix and imaging time of 2 min 39 s. Diffusion-weighted axial imaging was performed with a single shot echo planar sequence with three diffusion weighted b values of 0.500 and 1000 s/mm^2. The imaging parameters were a repetition time (TR) of 4200 ms, echo time (TE) of 109 ms, 5 mm section thickness, 30 mm intersection gap, 90 flip angle, 230 mm field of view, 512 × 512 matrix and imaging time of 52 s. ADC maps were produced from diffusion weighted images. For gradient axial imaging, we used a gradient echo sequence with a repetition time (TR) of 860 ms, echo time (TE) of 26 ms, 5 mm section thickness, 30 mm intersection gap, 20 flip angle, 230 mm field of view, 512 × 512 matrix and imaging time of 2 min 58 s. About 3 ml of gadolinium based contrast was used for selected cases. SV PRESS 144, SV PRESS 31 Single voxel spectroscopy; 2D PRESS 144 multi voxel spectroscopy was performed at TE of 144 ms and 31 ms, TR was at 2000 ms. In single voxel studies, the voxel is placed on the lesion so that it covers the maximum area of the solid tumoral area. In multivoxel spectroscopy, the voxel was extended to cover perilesional area in selective cases of high-grade tumors, avoiding areas of cysts or necrosis and with minimal contamination from the surrounding non-tumoral tissue. Volume of interest size ranged between 1.5 cm × 1.5 cm × 1.5 cm (3.4 ml) and 2 cm × 2 cm × 2 cm (8 ml). We used PRESS and T1 FFE postcontrast sequence as localization sequence with

5 mm thickness. Spectroscopy was avoided in small lesions close to the bone and sinuses. MultiHance (Gadobenate Dimeglumine) was used as contrast agents in dose of 0.1 mmol/kg body weight. Post-contrast coronal and axial T1W were taken and sagittal T1W were taken wherever necessary. Special sequence like FLAIR sequence was obtained in all cases. The images were analyzed for lesion number, size, location, signal intensity changes, mass effect, and contrast enhancement. Detailed clinical history was noted in patients admitted in our hospital and for referred cases from outside hospitals as per the pro forma.

All patients were followed up to reach a therapeutic/biopsy diagnosis. Findings of cerebrospinal fluid examination, muscle or nerve biopsy, electromyography, and electroencephalography were compared with the MRI features. The symptomatic response of the patient to medical therapy was noted which helped in the retrospective confirmation of the diagnosis, and thus the final outcome of the disease was recorded. Biochemical analysis could not be performed since the facility is not available in our institution. However, we could retrospectively confirm our diagnosis from patients who were referred to higher center and came back to our institute for follow-up treatment.

Data were entered into Microsoft Excel data sheet and analyzed using Epi Info 7 version software. Categorical data were represented in the form of frequencies and proportions. Bar diagrams and Pie chart were plotted to represent graphically wherever necessary.

RESULTS

In the study, it was observed that most common white matter disease was leukodystrophy (26%). Leukodystrophies includes adrenoleukodystrophy (ALD), Alexander disease, Canavan disease, Krabbes disease, metachromatic leukodystrophy (MLD), and nonspecific leukodystrophy. Second most common disease was acute disseminated encephalomyelitis (ADEM) (16%) and cavitating leukoencephalopathies (16%) (periventricular leukomalacia [PVL], megalencephalic leukoencephalopathy and cavitating leukoencephalopathy and vanishing white matter disease (WMD). Followed by hypomyelination (12%), multiple sclerosis and microvascular ischemic changes (SVIC) (8%) and others (Table 1).

The majority of the subjects were in the age group of <5 years, i.e., 70% and least no of subjects were observed in the age group >50 years 8%. Hence, the white matter diseases are common in children that in adults and elderly (Table 2).

In this study, the majority (n = 47) of the patients were found with supratentorial lesions and 19 were found to be having infratentorial lesions. Degree of myelination was adequate in nearly two-thirds of the individuals (n = 35) (Table 3).

According to the locations of the lesions, in nearly half of the cases, the lesions were in frontal region (n = 24), in 26 patients the lesions were seen in parietal area, 22 had lesions in temporal area, 19 in occipital region, deep gray matter lesions were seen in 17 cases, periventricular lesions in 36 cases and brainstem lesions were seen in 10 cases (Table 4).

In all the cases, hypointensity was noticed in T1W images and hyperintensity in the case of T2W images. Hyperintensity was observed in diffusion weighted images in case of 15 study participants, and ADC matching was present in the case of only 10 cases. Contrast enhancement was seen in 8 cases out of 16 in whom the contrast study was done (Table 5).

Table 1: MRI diagnosis in white matter diseases

MRI diagnosis	Frequency (%)
Leukodystrophy	13 (26)
ADEM	8 (16)
Cavitating leukoencephalopathies	8 (16)
Hypomyelination	6 (12)
Multiple sclerosis	4 (8)
SVIC	4 (8)
Leigh's disease	2 (4)
HIV encephalopathy	2 (4)
Nonspecific	2 (4)
PML	1 (2)
Total	50 (100)

MRI: Magnetic resonance imaging, ADEM: Acute disseminated encephalomyelitis, SVIC: Microvascular ischemic changes, HIV: Human immunodeficiency virus, PML: Progressive multifocal leukoencephalopathy

Table 2: Age distribution of subjects

Age (years)	Frequency (%)
<5	35 (70)
5-50	11 (22)
>50	4 (8)
Total	50 (100)

Table 3: Supratentorium and infratentorium lesions in white matter disease and degree of myelination

Lesions	Frequency (%)
Supratentorium	
Absent	3 (6)
Present	47 (94)
Infratentorium	
Absent	31 (62)
Present	19 (38)
Degree of myelination	
Inadequate	15 (30)
Adequate	35 (70)

Table 4: Location of lesions

Lesions	Frequency (%)
Frontal	
Absent	26 (52)
Present	24 (48)
Parietal	
Absent	24 (48)
Present	26 (52)
Temporal	
Absent	28 (56)
Present	22 (44)
Occipital	
Absent	31 (62)
Present	19 (38)
Deep gray matter	
Absent	33 (66)
Present	17 (34)
Periventricular	
Absent	14 (28)
Present	36 (72)
Subcortical U-fibers	
Absent	34 (68)
Present	16 (32)
Cerebellum	
Absent	37 (74)
Present	13 (26)
Brainstem	
Absent	40 (80)
Present	10 (20)

Table 5: MRI findings

MRI method and finding	Frequency (%)
T1W	
Hypointense	50 (100)
T2W	
Hyperintense	50 (100)
DWI	
Hypointense	35 (70)
Hyperintense	15 (30)
ADC Matching	
Absent	40 (80)
Present	10 (20)
Contrast enhancement	
Not performed	34 (68)
Present	8 (16)
Absent	8 (16)

DWI: Diffusion-weighted imaging, ADC: Apparent diffusion coefficient

In the study, only 11 patients were taken for MRS peak and it was observed that in leukodystrophy MRS peak was observed in choline, N-acetyl aspartate, Myo inositol and lactate. In leukoencephalopathies, MRS peak was observed only in lactate. ADEM had peak for choline (Table 6).

Among 13 leukodystrophy conditions various diagnosis in the study were MLD (38.4%), ALD (23.1%), Canavan disease (15.4%), 7.7% Krabbes disease, Alexander disease, and nonspecific leukodystrophy (Table 7).

Among 8 leukoencephalopathies 50% was PVL, 25% was megalencephalic leukoencephalopathy and 12.5% was cavitating leukoencephalopathy, vanishing WMD, respectively (Table 8).

In ADEM, the lesion was more common in Frontal region (75%), followed by parietal lesion in 62.5%. Lesion was observed in all the regions in varying proportions (Table 9).

DISCUSSION

This study was directed to evaluate the role of MRI in patients presenting with global developmental delay and clinically suspected of white matter disease.

About 50 patients clinically suspected of white matter disorders were submitted for MRI scan of brain. In the study, it was observed that most common white matter disease was leukodystrophy (26%). The second most common disease was ADEM (16%) and cavitating leukoencephalopathies (16%) followed by hypomyelination (12%), multiple sclerosis and SVIC (8%) and others. In our study, five out of 13 cases of leukodystrophies suffered from MLD (38.4%), among which 4 were males (80%) and 1 was female (20%). Alves *et al*.[3] reported that late infantile constitutes 70% of all cases and this is the commonest variety. MR imaging of these patients revealed bilaterally symmetrical and confluent lesions in all cases among which 100% incidence of periventricular white matter involvement, 80% of patients showing frontal white matter involvement and 60% showing parietal, temporal and cerebellar white matter involvement. Kim *et al*.[4] studied 7 patients of MLD, of which all of them showed bilateral, symmetrical and confluent high signal intensities on T2W imaging. They reported 100% incidence of periventricular white matter and centrum semi ovale. One of our patients, aged 25 months, showed involvement of subcortical U-fibers. This is in correlation with the study by Kim *et al*.[4] who reported that a follow-up MRI of a 26-month-old patient showed demyelinating process progressed to the subcortical U-fibers. In a study of three patients of MLD by Humera *et al*.,[5] all of which were male patients showed 100% involvement of periventricular white matter showing high signal intensities on T2W and FLAIR images (Figure 1).

In our study, three out of 13 cases of leukodystrophies suffered from ALD (23.1%), among which two were males (66.67%) and one was female (33.33%). Age of patients ranged from 9 months to 6 years. Snyder *et al*.[6] reported that childhood onset ALD (4-8 years) is the most common type. White matter abnormalities usually appear in the occipital regions initially, with early involvement of the splenium of the corpus callosum and posterior limbs of internal capsules.[7] The progression pattern of the disease is centrifugal and posteroanterior.[8] This results

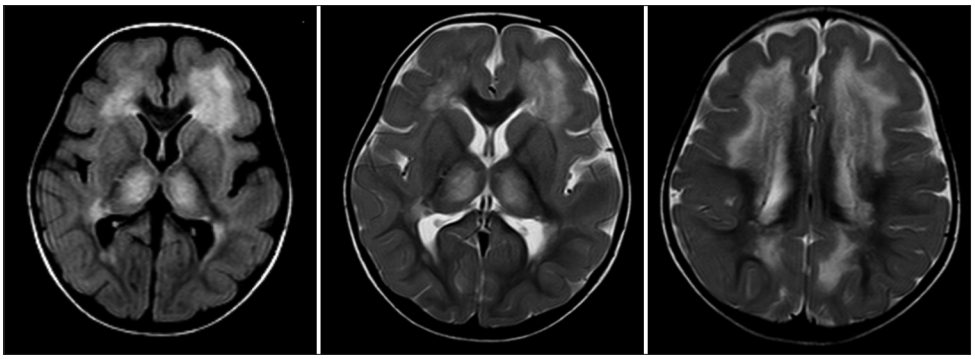

Figure 1: Metachromatic leukodystrophy: Axial T2 and fluid attenuated inversion recovery hyperintensities are seen in bifrontal, temporal and parietal periventricular white matter with sparing of subcortical U-fibers. The hyperintensities is more marked in bifrontal lobes and cerebellum. Oval hyperintense foci with central cystic area are seen involving bilateral thalami

Table 6: MRS peak among the white matter diseases

White matter diseases	MRS peak					Total
	Not performed	N-acetyl choline	Choline	Myo inositol	Lactate	
Leukodystrophy	5	2	3	2	1	13
ADEM	7	0	1	0	0	8
Leukoencephalopathies	6	0	0	0	2	8
Hypomyelination	6	0	0	0	0	6
Multiple sclerosis	4	0	0	0	0	4
SVIC	4	0	0	0	0	4
Leigh's disease	2	0	0	0	0	2
HIV encephalopathy	2	0	0	0	0	2
Nonspecific	2	0	0	0	0	2
PML	1	0	0	0	0	1
Total	39	2	4	2	3	50

ADEM: Acute disseminated encephalomyelitis, MRS: Magnetic resonance spectroscopy, PML: Progressive multifocal leukoencephalopathy, HIV: Human immunodeficiency virus, SVIC: Microvascular ischemic changes

Table 7: Various types of leukodystrophy white matter diseases

Leukodystrophies	Frequency (%) (*n*=13)
Leukodystrophy	
MLD	5 (38.4)
ALD	3 (23.1)
Canavan disease	2 (15.4)
Krabbes disease	1 (7.7)
Alexander disease	1 (7.7)
Nonspecific leukodystrophy	1 (7.7)
Total	13

MLD: Metachromatic leukodystrophy, ALD: Adrenoleukodystrophy

Table 8: Various types of leukoencephalopathies in white matter disease

Leukoencephalopathies	Frequency (%) (*n*=8)
PVL	4 (50)
Megaloencephalic leukoencephalopathy	2 (25)
Cavitating leukoencephalopathy	1 (12.5)
Vanishing WMD	1 (12.5)
Total	8

PVL: Periventricular leukomalacia, WMD: White matter disease

in the most characteristic imaging feature of the disease. Our study shows no involvement of frontal white matter in all three patients. All patients showed temporal and occipital white matter abnormalities (100%) and 66.67% showing periventricular and brain stem involvement. All patients showed enhancement on contrast study (100%) and also choline peak in MRS. In a study of three patients of ALD by Ahsan *et al.*,[5] it was reported 66.67% involvement of periventricular white matter specifically in trigonal area showing high signal intensities on T2W and FLAIR images. Melhem *et al.*[9] in a study of 43 patients of ALD reported 21 patients (49%) showing contrast enhancement.

MR imaging findings of multiple sclerosis patients were bilateral periventricular hyperintensities on T2W and FLAIR images. Deep gray matter hyperintensities were also identified in all patients. One patient showed hyperintense lesions in bilateral frontal, parietal and occipital lobes, each lesion measuring approximately 5-10 mm in size. In a study by Tas *et al.*[10] showed that gadolinium-enhancement

Table 9: Location of lesion in ADEM (*n*=8)

Location of lesions	ADEM	
	Present	Percentage
Frontal lesion	6	75
Parietal lesion	5	62.5
Temporal lesion	4	50
Occipital lesion	2	25
Deep gray matter lesion	1	12.5
Periventricular lesion	3	37.5
Subcortical U-fibers lesion	4	50
Cerebellum lesion	4	50
Brainstem lesion	4	50

ADEM: Acute disseminated encephalomyelitis

was more specific for diagnosing multiple sclerosis than abnormalities revealed on T2W imaging. In a study of 42 patients by Barkhof *et al*.,[11] Gadolinium enhancement was identified as the most predictive MRI parameter. When compared these results with our study where we have found similar diagnostic criteria for MS on MRI such as 9 or more T2 hyperintense lesions, periventricular lesions and gadolinium enhancement in all the 4 patients of our study who were clinically suspected strongly for multiple sclerosis. Thus, our study results correlated with these studies (Figure 2).

In our study, seven patients suffering from ADEM were obtained among which four were females and three were male patients ranging from 2 to 16 years of age. Mikaeloff *et al*.[12] reported in their study that 66% pf their patients suffering from ADEM showed juxtacortical hyperintensities on MR imaging. Marchioni *et al*.,[13] in their study, reported that 42% of their patients with ADEM showed gadolinium enhancement whereas Lin *et al*.[14] reported that 40 % of their patients suffering from ADEM showed gadolinium enhancement. These findings are in correlation with our study (Figure 3).

Our study revealed one male patient of 2 years of age found to be a case of alexander disease (Figure 4). MR imaging study of this patient showed hyperintensities on T2W images predominantly in the frontal white matter with extension into the parietal and temporal white matter, periventricular regions and subcortical U-fibers. The lesions show contrast enhancement. MRS revealed a rise in Lactate peak. In a study by van der Knaap *et al*.,[15] five MR imaging criteria were defined: Extensive cerebral white matter changes with frontal predominance, a periventricular rim with high signal on T1W images and low signal on T2W images, abnormalities of basal ganglia and thalami, brain stem abnormalities, and contrast enhancement of particular gray and white matter structures. Four of the five criteria had to be met for an MR imaging-based diagnosis. Our study meets four criteria mentioned in their study, thus correlating with the study.

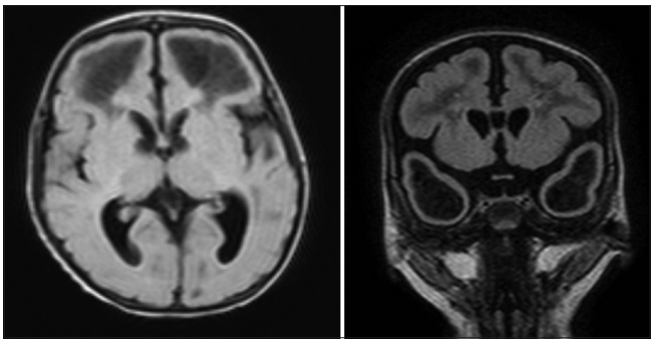

Figure 2: Periventricular leukomalacia: Axial and coronal fluid attenuated inversion recovery images show bilateral symmetric periventricular cystic lesions measuring 8-12 mm are seen in adjacent to frontal/body of lateral ventricles and also anterior to temporal horns. In addition smaller size lesions are seen in bilateral posterior periventricular white matter. Mild volume loss is seen in bilateral periventricular brain parenchyma

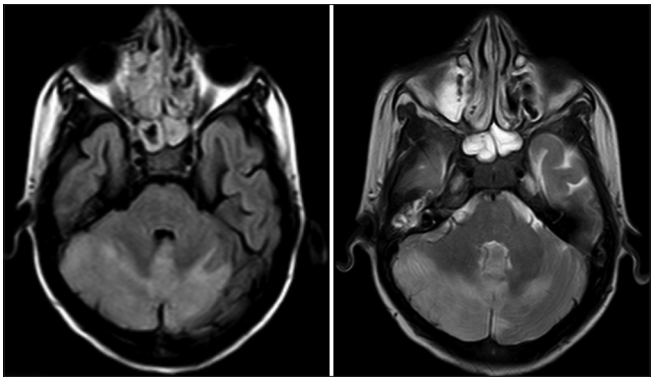

Figure 3: Acute disseminating encephalomyelitis: Diffuse ill-defined abnormal signal intensities are seen predominantly involving the cortices of both cerebellar hemispheres (R>L) and cerebellar vermis, appearing homogenously hyperintense on T2 and fluid attenuated inversion recovery, hypointense on T1-weighted. There is minimal mass effect seen in the form of effacement of the involved sulci

In this study, two patients out of 13 cases of leukodystrophies were reported to have Canavan disease (Figure 5). One of our two patients showed diffuse bilateral symmetric cerebral and cerebellar white matter high signal intensities on T2 and FLAIR images and no lobar predominance. Another patient showed involvement of bilateral deep gray matter and subcortical U-fibers. Both the patients were subjected to MRS study which revealed and marked rise in the N-acetylaspartate (NAA) peak. Michel and Given[16] reported a case of Canavan disease in which there was diffuse, bilateral, and symmetric increased T2 signal intensity throughout the cerebral white matter. These findings were noted to a lesser degree in the cerebellar white matter, thalamus, globi pallidi, and dorsal brainstem. The white matter abnormality specifically involved the subcortical white matter. There was no lobar predominance of white matter abnormalities. Single-voxel point-resolved spatially localized MRS revealed a marked increase in both

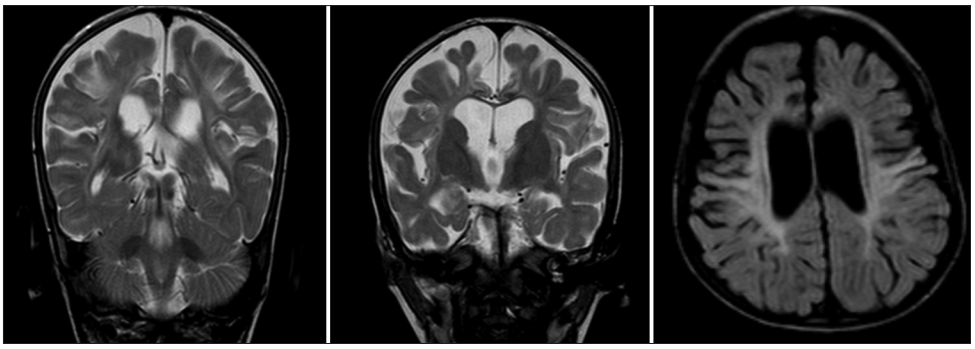

Figure 4: Alexander disease: Axial T2 and fluid attenuated inversion recovery cortical hyperintensities are seen in bilateral high frontal lobe and also in posterior periventricular white matter

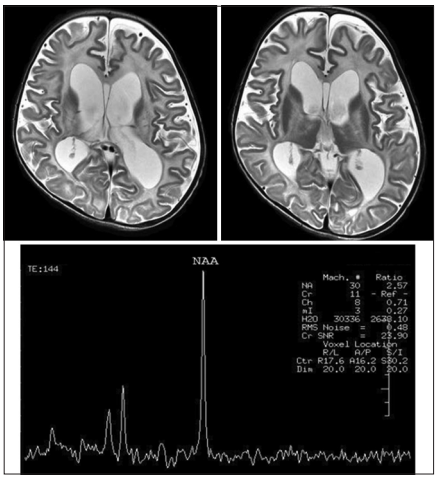

Figure 5: Canavan disease: Axial T2-weighted image shows high signal in white matter typically a diffuse bilateral involvement of sub cortical U-fibers. Magnetic resonance spectroscopy - markedly elevated N-acetylaspartate (NAA) and NAA:creatine ratio

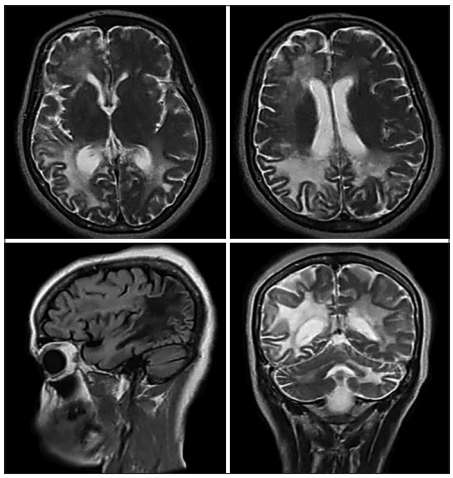

Figure 6: Progressive multifocal leukoencephalopathy: Axial T2-weighted images show multifocal bilateral parietal, occipital, periventricular, subcortical U-fibers, deep gray matter hyperintensities. Sagittal T1-weighted image shows posterior hypointensities. Coronal T2-weighted image shows occipital and bilateral cerebellar white matter involvement

the NAA peak and the ratio of NAA to creatinine. The choline peak was not elevated. Our MR findings are in correlation with their study.

One case of progressive multifocal leukoencephalopathy (PML) was encountered in our study (Figure 6). The patient was 33-year-old male patient with human immunodeficiency virus (HIV) seropositive status. MR imaging findings revealed multifocal bilateral parietal, occipital, periventricular, subcortical U-fibers, deep gray matter and bilateral cerebellar white matter hyperintensities on T2W and FLAIR images. Ahsan *et al.*[5] reported, in their study, that one male patient suffered from PML. He was 30-year-old and tested positive for HIV. MRI showed abnormal signal intensity in deep white matter of brain in right frontal regions, parietooccipital regions, putamen, and midbrain. The signals were isointense on T1 and hyperintense on T2W images. It is observed that these findings are in correlation with our study.

CONCLUSION

In general, patients in the pediatric age group constituted 82% of the total study group, and there was slight male predominance. Leukodystrophies constituted 26% of total cases, the commonest being MLD. All patients of acute disseminating encephalomyelitis had a prior history of viral fever. The most common symptom noted was altered sensorium and site of involvement was cerebral white matter, asymmetric multifocal pattern being the most common. The patient with PML was HIV positive and showed focal white matter lesions without mass effect or enhancement. In leukodystrophies, we had 13 cases of which five were MLD, three were ALD, two cases of Canavan disease and one each case of Krabbes disease, Alexander disease and nonspecific leukodystrophy. In leukoencephalopathies, we had 8 cases of which four were PVL, two were megalencephalic leukoencephalopathy and one each case of cavitating leukoencephalopathy and vanishing WMD. All patients were

from the pediatric age group, and male predominance was noted. All cases of hypomyelination showed periventricular white matter involvement. Elderly patients showed punctate and confluent hyperintensities involving supratentorial and infratentorial brain white matter lesions, attributing to degenerative changes of small vessels multiplanar imaging capability and excellent gray - white matter resolution makes MRI very sensitive in detecting subtle white matter lesions. FLAIR sequence has better sensitivity for white matter lesions especially those in periventricular locations. Post-contrast studies help in differentiating acute from chronic lesions and thus monitoring the progression of the disease. Allows simultaneous imaging of orbit especially in cases of multiple sclerosis. Helps in early diagnosis of mild and atypical cases so that treatment can be started early in curable disease. Ideal for posterior fossa and spinal cord imaging. Noninvasive imaging modality which can be done on outpatient basis and with no radiation hazards. Specificity of MR imaging can be improved by MRS studies. Only limitation is that though sensitive and not as specific in aiding in the specific diagnosis of diseases. However, the specificity can be improved by clinical correlation and laboratory tests.

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