Journal of Surgical Radiology

Site: https://journalsurgrad.com/

ISSN Print 2156-213X| ISSN Online: 2156-4566 Volume: 05 Issue: 06 Open Access

Research Article

Magnetic Resonance Imaging in the Evaluation of White Matter Disorders

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Article History
Received: 21.05.2026
Revised: 29.05.2026
Accepted: 09.06.2026
Published: 18.06.2026

Citations:
Chaudhry, B., Agrawal, S. K., Sukhani, P. K., Kalwaniya, P., Meena, H., & Waquar, S. (Year). Magnetic Resonance Imaging in the Evaluation of White Matter Disorders. J Surg Radiol, V5(6) 303-312

Abstract: ***Introduction***: White matter disorders constitute a heterogeneous group of neurological diseases affecting myelin integrity, resulting in cognitive, motor, sensory, and neurobehavioral deficits. Magnetic resonance imaging (MRI) is the gold standard for detection and characterization of white matter lesions (WMLs). This study aimed to evaluate the spectrum of MRI findings in patients with clinically suspected white matter disorders and correlate imaging features with clinical presentation.**Aim :** To assess the role of MRI in the diagnosis of white matter diseases in clinically suspected patients**Methods:** This hospital-based cross-sectional observational study included 50 consecutive patients referred for MRI brain evaluation due to suspected white matter pathology. Clinical data, demographic characteristics, and vascular risk factors were recorded. MRI sequences included T1-weighted, T2-weighted, FLAIR, diffusion-weighted imaging (DWI), and contrast-enhanced T1 sequences when indicated. Lesion characteristics, distribution, pattern, and severity were assessed, and the Fazekas scale was used for grading WML severity. MRI findings were correlated with final clinical diagnosis to determine diagnostic accuracy.**Results**: The mean age was 46.8±17.2 years, with a male predominance (56%). Vascular etiology, predominantly small vessel ischemic disease, was the most common cause (44%), followed by demyelinating disorders (32%). Cognitive impairment (36%), motor weakness (32%), and seizures (28%) were the most frequent clinical presentations. MRI revealed T2 hyperintensity in all patients, FLAIR hyperintensity in 96%, T1 hypointensity in 76%, restricted diffusion in 28%, and contrast enhancement in 24%. Periventricular (60%) and deep white matter (52%) were the most commonly affected regions. MRI diagnosis agreed with clinical diagnosis in 80% of cases, with sensitivity of 95.5% and diagnostic accuracy of 90%.**Conclusion:** MRI is a highly sensitive and reliable modality for evaluation and etiological classification of white matter disorders. Vascular lesions predominate in older adults, while demyelinating disorders are more common in younger patients. MRI provides crucial information for diagnosis, management, and prognostication of WMLs.

Keywords: White matter lesions, MRI, small vessel disease, demyelinating disorders, Fazekas scale, cognitive impairment

INTRODUCTION

White matter of the central nervous system (CNS) forms an extensive network of myelinated and unmyelinated nerve fibers that enables rapid communication between cortical and subcortical regions. It constitutes nearly half of total brain volume and is essential for cognitive, sensory, motor, and behavioral functions[1]. White matter disorders, commonly referred to as Leukoencephalopathies, comprise a broad spectrum of diseases characterized by abnormalities in myelin formation, maintenance, or its destruction. Since myelin is critical for efficient saltatory conduction and axonal protection, damage to white matter can result in significant neurological dysfunction.

Myelin is produced by oligodendrocytes and consists of water, lipids, cholesterol, phospholipids, galactolipids, and proteins[2]. Its structural integrity depends on the coordinated interaction of oligodendrocytes, axons, astrocytes, microglia, and precursor cells. Disruption at any point in this complex system due to genetic, inflammatory, metabolic, vascular, infectious, toxic, or traumatic causes may lead to white matter injury.

White matter disorders may broadly be classified into demyelinating, dysmyelinating, and hypomyelinating disorders. Demyelinating disorders involve destruction of previously normal myelin and include multiple sclerosis (MS), acute disseminated encephalomyelitis (ADEM), neuromyelitis optica spectrum disorders (NMOSD), and myelin oligodendrocyte glycoprotein antibody-associated disease[3]. Dysmyelinating disorders are caused by inherited defects in myelin metabolism, resulting in structurally abnormal myelin.

Classical leukodystrophies such as metachromatic leukodystrophy, adrenoleukodystrophy, Krabbe disease, and Canavan disease fall within this group[4–7]. Hypomyelinating disorders are characterized by deficient formation of otherwise normal myelin and include Pelizaeus–Merzbacher disease, 4H leukodystrophy, Salla disease, and hypomyelination with atrophy of the basal ganglia and cerebellum.

White matter lesions (WMLs) can arise from both vascular and non-vascular causes. Vascular causes commonly include chronic hypoperfusion, cerebral small vessel disease, vasculitis, atherosclerosis, migraine-related vascular changes, cerebral amyloid angiopathy, and Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL)[8]. These lesions typically appear as periventricular or deep white matter hyperintensities on magnetic resonance imaging (MRI). Risk factors such as hypertension, diabetes mellitus, dyslipidemia, smoking, hyperhomocysteinemia, systemic inflammation, and aging significantly contribute to lesion burden [8].Non-vascular causes include inflammatory demyelination, metabolic and toxic encephalopathies, infectious conditions such as HIV- associated leukoencephalopathy and progressive multifocal leukoencephalopathy, traumatic diffuse axonal injury, autoimmune disorders, neoplastic infiltration, and inherited leukodystrophies[4,9]. Depending on the underlying pathology, these disorders may produce focal, multifocal, symmetric, or confluent white matter abnormalities.Clinical manifestations of white matter disorders are highly variable and depend on lesion location, burden, etiology, and age of onset. Mild punctate lesions may remain asymptomatic, especially in older individuals, whereas extensive WMLs are associated with cognitive decline, executive dysfunction, gait disturbances, urinary symptoms, depression, dementia, and disability[9–11]. Several studies have shown that WMLs are independent predictors of stroke, dementia, disability, and mortality[10–12]. In demyelinating disorders such as MS, symptoms commonly include sensory disturbances, visual loss, motor weakness, bladder dysfunction, fatigue, and cognitive impairment[3].MRI has revolutionized the evaluation of white matter disorders because of its superior soft tissue contrast, multiplanar capability, and high sensitivity for detecting microstructural abnormalities[13]. T1-weighted images provide information regarding myelin integrity and cerebral atrophy, while T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences highlight edema, gliosis, and demyelination. Diffusion-weighted imaging (DWI) is particularly useful in detecting acute ischemia and cytotoxic edema, whereas diffusion tensor imaging (DTI) helps evaluate tract integrity. Susceptibility-weighted imaging (SWI) is useful for detecting microhemorrhages and mineralization, and contrast-enhanced MRI helps identify active inflammation.Certain MRI patterns are highly suggestive of specific diseases. Dawson's fingers are characteristic of MS, the tigroid pattern is often seen in metachromatic leukodystrophy, and confluent deep white matter involvement is commonly associated with CADASIL[13,14]. MRI is also useful for differentiating acute from chronic lesions, monitoring disease progression, assessing response to treatment, and guiding further biochemical or genetic investigations. Despite advances in MRI, interpretation of white matter abnormalities remains challenging because of the significant overlap in imaging features among different diseases. A structured MRI-based approach focusing on lesion distribution, symmetry, enhancement, diffusion restriction, and associated gray matter or spinal cord involvement can improve diagnostic accuracy[14]. Therefore, evaluation of MRI findings is essential for early diagnosis, timely treatment, and improved patient outcomes in white matter disorders.

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of Radiodiagnosis at Mahatma Gandhi Medical College and Hospital with the objective of evaluating the diagnostic role of magnetic resonance imaging (MRI) in clinically suspected cases of white matter disorders.The study was carried out over a period from April 2024 to September 2025. All clinically suspected patients of white matter diseases referred from the Departments of Medicine and Neurology, including both outpatient and inpatient services, for MRI evaluation during the study period were screened for eligibility.All consecutive patients fulfilling the inclusion criteria were enrolled in the study. Since this was an observational diagnostic study, no prior fixed sample size calculation was performed, and the sample size was determined by the number of eligible patients presenting during the study period.Prior approval for the study was obtained from the Institutional Ethics Committee before commencement of data collection.

Written informed consent was obtained from all patients or their legally authorized guardians after explaining the purpose of the study, MRI procedure, and the use of contrast media wherever required.Patients of all age groups with clinically suspected white matter disorders referred for MRI brain examination were included in the study.

Patients who refused consent, had contraindications to MRI such as cardiac pacemakers, intracranial aneurysm clips, cochlear implants, or metallic foreign bodies, had severe claustrophobia, or had a known history of severe hypersensitivity to contrast media were excluded from the study.MRI examinations were performed using either 1.5 Tesla or 3 Tesla MRI scanners available in the Department of Radiodiagnosis. All patients were examined in the supine position using a standard head coil.The routine MRI protocol included axial, sagittal,

and coronal sequences comprising T1-weighted imaging, T2-weighted imaging, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and post-contrast T1-weighted imaging wherever indicated. Contrast-enhanced MRI was selectively performed in patients with suspected inflammatory, infective, neoplastic, or active demyelinating lesions. Prior to contrast administration, renal function status and history of allergy to contrast agents were assessed.For each patient, demographic and clinical details were recorded in a structured proforma. Demographic variables included age, sex, and occupation. Clinical presentation was documented in detail and included symptoms such as seizures, aphasia, dementia, unilateral visual blurring, sensory disturbances, motor weakness, urinary incontinence, speech and swallowing difficulties, numbness, tingling sensations, and cognitive dysfunction.Relevant past medical histor y and vascular risk factors were also documented, including hypertension, diabetes mellitus, hyperlipidemia, hyperhomocysteinemia, and elevated C-reactive protein levels.All MRI scans were systematically evaluated with emphasis on lesion location, morphology, distribution, and signal characteristics. Lesion location was categorized as periventricular, deep white matter, subcortical, or infratentorial. Lesion distribution was assessed as focal or diffuse and symmetric or asymmetric. Signal characteristics on T1-weighted, T2-weighted, FLAIR, and DWI sequences were analyzed. The presence of diffusion restriction, contrast enhancement, gray matter involvement, mass effect, and cerebral atrophy was also noted.White matter lesions were graded using the Fazekas scale for periventricular and deep white matter involvement wherever applicable. MRI findings were interpreted in correlation with the clinical presentation to arrive at the most likely diagnosis.All MRI images were independently reviewed by experienced radiologists to reduce observer bias and improve diagnostic accuracy.

Statistical analysis was performed using SPSS version 25.. Categorical variables were expressed as frequency and percentage, while continuous variables were expressed as mean±standard deviation or median with interquartile range wherever appropriate. Chi-square test or Fisher's exact test was used for comparison of categorical variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 50 patients with clinically suspected white matter disorders were included in the study.The age distribution of the study population showed a predominance of middle-aged and elderly individuals. The highest proportion of patients belonged to the 41–60 years age group (36.0%), followed by patients aged more than 60 years (24.0%). The mean age of the study population was 46.8±17.2 years, with a median age of 48 years (IQR: 32–62 years). Males constituted 56.0% of the study population, while females accounted for 44.0%, indicating a slight male predominance (Table 1, Figure 1).

Table 1. Demographic Characteristics of the Study Population (n = 50)

Variable	Number of Patients n (%)
≤20 years	6 (12.0%)
21–40 years	14 (28.0%)
41–60 years	18 (36.0%)
>60 years	12 (24.0%)
Male	28 (56.0%)
Female	22 (44.0%)
Mean age ± SD	46.8 ± 17.2 years
Median age (IQR)	48 (32–62) years

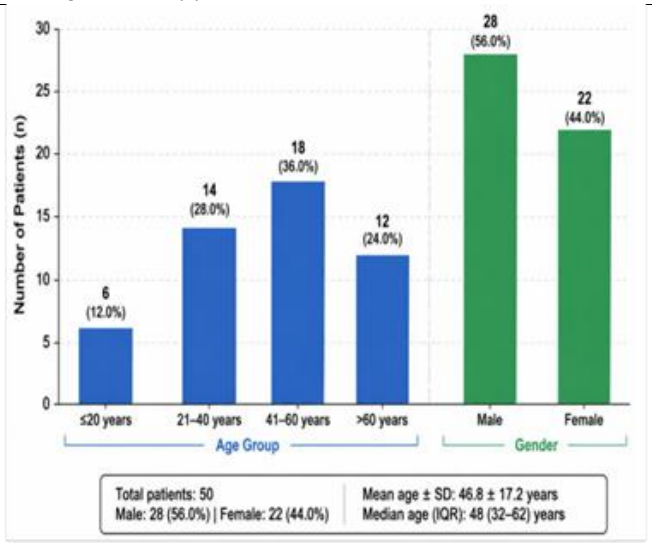


Figure 1. Patient Distribution by Age Group and Gender

Cognitive impairment or dementia was the most common presenting symptom, observed in 36.0% of patients, followed by motor weakness (32.0%) and seizures (28.0%). Sensory disturbances and gait imbalance were present in nearly one-fourth of patients, while visual disturbances and urinary incontinence were less common. Hypertension was the most common associated clinical risk factor, present in 48.0% of patients, followed by diabetes mellitus (36.0%) and hyperlipidemia (32.0%) (Table 2, Figure 2).

Table 2. Clinical Presentation and Risk Factors in the Study Population (n = 50)

Variable	Number of Patients n (%)
Cognitive impairment / dementia	18 (36.0%)
Motor weakness	16 (32.0%)
Seizures	14 (28.0%)
Sensory disturbances	12 (24.0%)
Gait imbalance / ataxia	11 (22.0%)
Visual disturbances	10 (20.0%)
Urinary incontinence	7 (14.0%)
Hypertension	24 (48.0%)
Diabetes mellitus	18 (36.0%)
Hyperlipidemia	16 (32.0%)
Hyperhomocysteinemia	12 (24.0%)
Elevated hs-CRP	10 (20.0%)

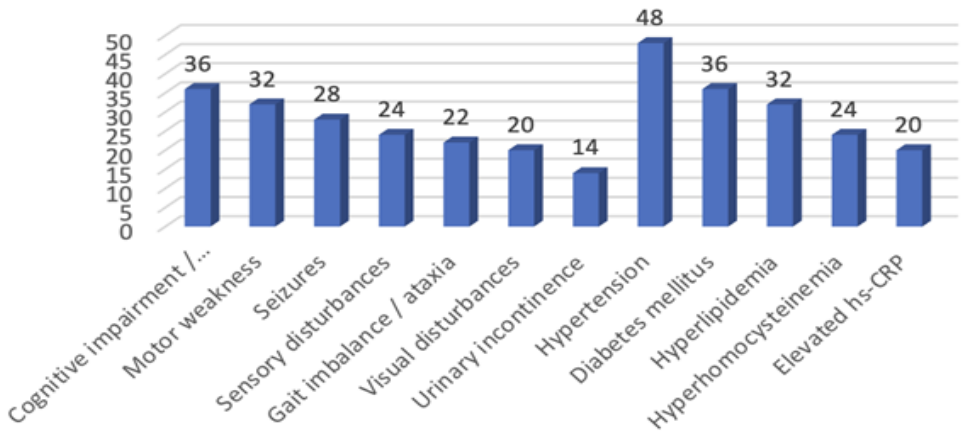


Figure 2. Clinical Presentation and Risk Factors in the Study Population (n = 50)

Vascular etiology, predominantly small vessel ischemic disease, was the most common cause of white matter lesions, accounting for 44.0% of cases. Demyelinating disorders, including multiple sclerosis and acute disseminated encephalomyelitis, formed the second largest group (32.0%). Infectious or inflammatory causes accounted for 12.0% of cases, while metabolic, toxic, and genetic causes together contributed to 12.0% of cases. Small vessel ischemic disease was the most frequent final MRI diagnosis (44.0%), followed by multiple sclerosis (28.0%) (Table 3, Figure 3).

Table 3. Etiological Classification and Final MRI Diagnosis of White Matter Disorders (n = 50)

Variable	Number of Patients n (%)
Vascular etiology	22 (44.0%)
Demyelinating disorders	16 (32.0%)
Infectious / inflammatory	6 (12.0%)
Metabolic / toxic	4 (8.0%)
Genetic / leukodystrophy	2 (4.0%)
Small vessel ischemic disease	22 (44.0%)
Multiple sclerosis	14 (28.0%)
ADEM	4 (8.0%)
Infectious / inflammatory leukoencephalopathy	5 (10.0%)
Metabolic / toxic white matter disorder	3 (6.0%)
Leukodystrophy	2 (4.0%)

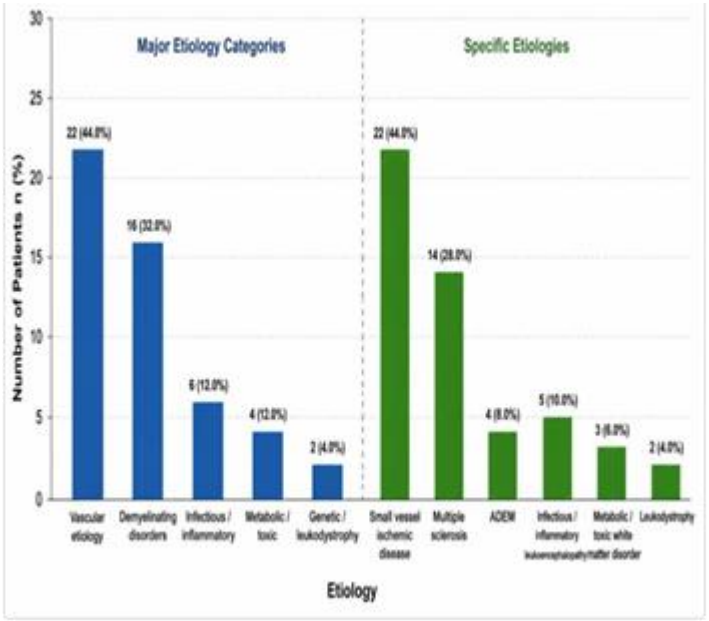


Figure 3. Etiology of White Matter Pathologies

On MRI, all patients demonstrated hyperintense lesions on T2-weighted images, while FLAIR hyperintensity was observed in 96.0% of patients. T1 -weighted hypointense lesions were present in 76.0%, restricted diffusion in 28.0%, and post-contrast enhancement in 24.0% of cases. Periventricular white matter involvement was the most common lesion location, seen in 60.0%

of patients, followed by deep white matter involvement (52.0%) and subcortical lesions (40.0%). Corpus callosum involvement was noted in 24.0% of patients and brainstem or cerebellar involvement in 16.0%. Confluent lesions were the most common lesion pattern, observed in 40.0% of cases, and asymmetric involvement was more common than

symmetric involvement (60.0% vs. 40.0%). According to Fazekas grading, Grade 2 lesions were the most common, seen in 40.0% of patients (Table 4).

Table 4. MRI Characteristics and Lesion Distribution in White Matter Disorders (n = 50)

Variable	Number of Patients n (%)
T1W hypointense lesions	38 (76.0%)
T2W hyperintense lesions	50 (100.0%)
FLAIR hyperintense lesions	48 (96.0%)
Restricted diffusion	14 (28.0%)
Contrast enhancement	12 (24.0%)
Periventricular lesions	30 (60.0%)
Deep white matter lesions	26 (52.0%)
Subcortical lesions	20 (40.0%)
Corpus callosum involvement	12 (24.0%)
Brainstem / cerebellar involvement	8 (16.0%)
Punctate lesions	16 (32.0%)
Patchy lesions	14 (28.0%)
Confluent lesions	20 (40.0%)
Symmetric involvement	20 (40.0%)
Asymmetric involvement	30 (60.0%)
Fazekas Grade 1	16 (32.0%)
Fazekas Grade 2	20 (40.0%)
Fazekas Grade 3	14 (28.0%)

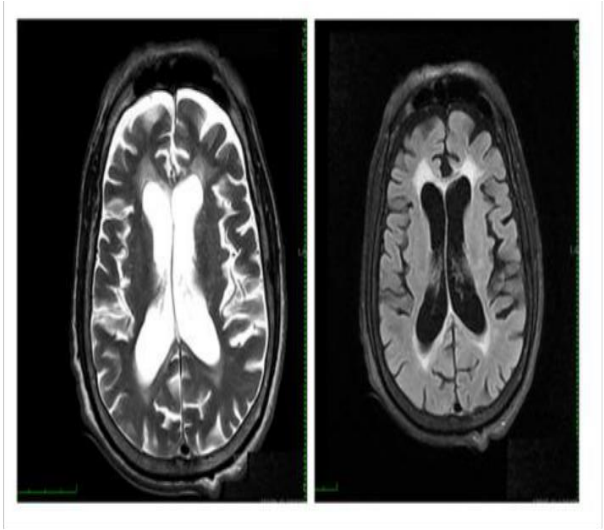
A statistically significant association was observed between age group and etiology of white matter lesions ($p = 0.002$). Vascular lesions were more common in patients aged more than 40 years, whereas demyelinating disorders predominated in younger patients aged 40 years or less. Periventricular lesions were significantly more common in vascular etiologies compared to non - vascular causes (81.8% vs. 42.9%, $p = 0.01$). In contrast, corpus callosum involvement, contrast enhancement, and restricted diffusion were significantly more frequent in non-vascular disorders, particularly demyelinating diseases. MRI diagnosis was concordant with the final clinical diagnosis in 80.0% of patients. MRI demonstrated excellent sensitivity (95.5%) and high diagnostic accuracy (90.0%) for detecting white matter disorders, although specificity was moderate (50.0%) (Table 5, Figure 4).

Table 5. Association of MRI Findings with Etiology and Diagnostic Performance of MRI

Variable	Findings
Vascular lesions in patients >40 years	18/30 (60.0%)
Demyelinating lesions in patients≤40 years	12/20 (60.0%)
p-value for age and etiology association	0.002
Periventricular lesions in vascular etiology	18/22 (81.8%)
Periventricular lesions in non-vascular etiology	12/28 (42.9%)
p-value	0.01
Corpus callosum involvement in vascular etiology	2/22 (9.1%)
Corpus callosum involvement in non-vascular etiology	10/28 (35.7%)
p-value	0.004
Sensitivity of MRI	95.5%
Specificity of MRI	50.0%
Positive Predictive Value	93.3%
Negative Predictive Value	60.0%
Diagnostic Accuracy	90.0%

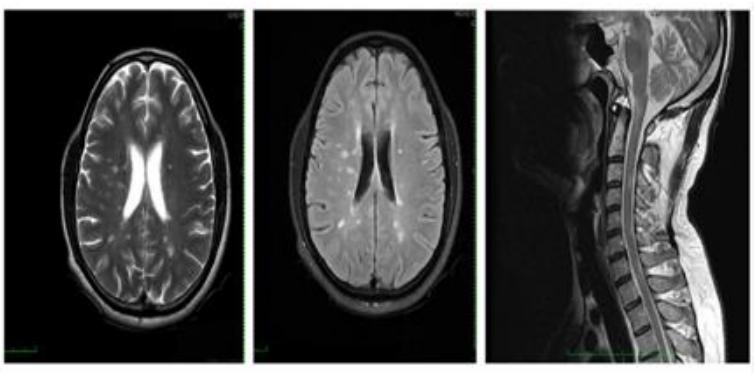
Figure 4. Diagnostic Performance of MRI

Case of ISCHEMIC DEMYELINATION



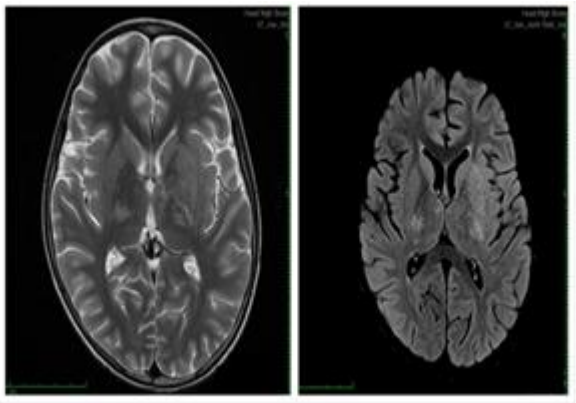
Axial T2 and Flair images showing confluent peri ventricular white matter hyperintensities

Case of MULTIPLE SCLEROSIS



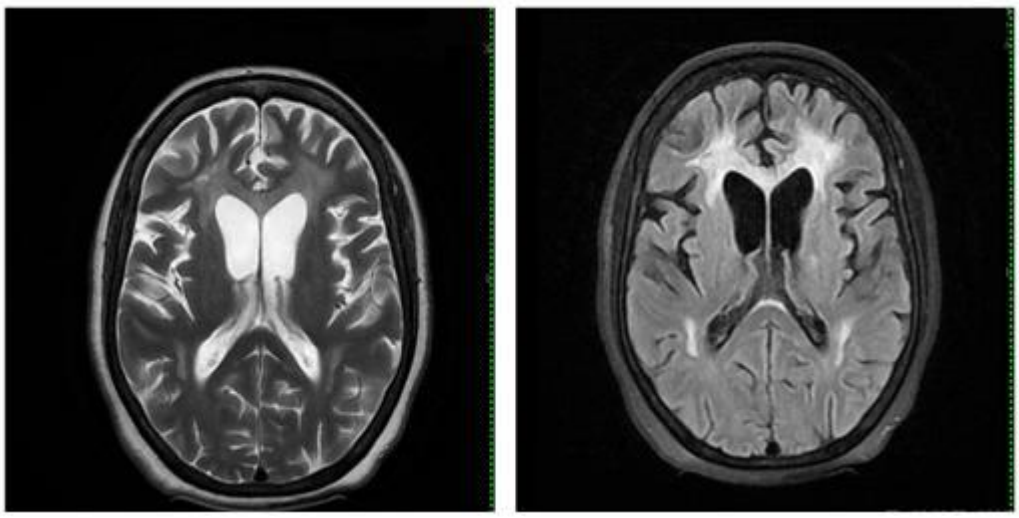
Axial T2 and Flair images showing sub cortical and peri ventricular hyperintensities Sagittal T2 image of cervical spine showing patchy areas of white matter hyper intensities in cervical cord

Case of ADEM



Axial T2 and Flair images showing Confluent and patchy hyperintensities involving bilateral basal ganglia Left>Right

Case of METACHROMATIC LEUKODYSTROPHY



Axial T2 and Flair images showing confluent T2 hyperintensities adjoining the frontal and occipital horns

DISCUSSION

In this hospital-based cross-sectional study involving 50 patients with clinically suspected white matter disorders, comprehensive MRI evaluation allowed detailed assessment of lesion characteristics, distribution, pattern, severity, and etiological classification, which were th en correlated with clinical findings. The mean age of the study population was 46.8±17.2 years, with a median age of 48 years, and the highest proportion of patients belonged to the 41–60 years age group (36.0%). This predominance of middle-aged and elderly patients indicates that white matter disorders are more commonly identified in adulthood, likely due to cumulative vascular and degenerative changes[15,16]. These findings are consistent with Barkovich et al. (2000)[15], who reported that adult white matter disorders are predominantly acquired, and with Schiffmann et al. (2009)[14], emphasizing the relevance of MRI-based diagnostic algorithms in adult populations. Males constituted 56% of patients, indicating a slight male predominance, which aligns with previous studies by Rana et al. (2018)[18] and Gowdar et al. (2015)[17], reporting54–58% male prevalence in similar cohorts. This is contrasted by demyelinating disorders such as multiple sclerosis, which show female predominance[19,20]. The sex distribution in our study reflects the mixed etiology, with vascular and metabolic lesions being more frequent.Cognitive impairment was the most common presenting symptom (36.0%), followed by motor weakness (32.0%) and seizures (28.0%), with sensory disturbances (24.0%), gait imbalance (22.0%), visual disturbances (20.0%), and urinary incontinence (14.0%). These findings suggest predominant involvement of cortical and long motor–sensory pathways. Similar symptom distribution was reported by Barkovich et al. (2000)[15], Sharma et al. (2025)[8], and Debette et al.

(2010)[9], emphasizing the strong association between white matter hyperintensities and cognitive decline, gait disturbances, and seizures. Etiologically, vascular causes, mainly small vessel ischemic disease, accounted for 44.0% of cases, followed by demyelinating disorders (32.0%), infectious/inflammatory (12.0%), metabolic/toxic (8.0%), and genetic/leukodystrophy (4.0%) These results correspond with prior adult studies by Weidauer et al. (2020)[16] and Sharma et al. (2025)[8], highlighting small vessel ischemic disease as the leading cause of adult white matter lesions. Demyelinating disorders were more frequent in younger patients (≤40 years), reflecting an age-dependent etiological pattern consistent with Lakhkar et al. (2002)[19] and Rana et al. (2018)[18].MRI findings demonstrated universal T2 hyperintensity (100%), FLAIR hyperintensity in 96%, T1 hypointensity in 76%, restricted diffusion in 28%, and contrast enhancement in 24%(Table 2). Periventricular involvement was most common (60%), followed by deep white matter (52%), subcortical (40%), corpus callosum (24%), and brainstem/cerebellum (16%). Vascular lesions were predominantly periventricular, whereas non-vascular lesions showed higher corpus callosum involvement, greater contrast enhancement, and diffusion restriction (Table 4), supporting prior observations by Schiffmann et al. (2009)[14], Lakhkar et al. (2002)[19], and Hesselink et al. (2006)[21].Pattern analysis showed that confluent lesions were most frequent (40%), with asymmetric involvement in 60% of patients. Moderate severity (Fazekas grade 2) was the most common (40%), indicating established but not end-stage pathology. These results mirror reports by Weidauer et al. (2020) [16], Sharma et al. (2025)[8], and Rana et al. (2018)[18], highlighting the association of lesion morphology with etiology and chronicity. MRI-based diagnosis demonstrated 80.0% concordance with the final clinical diagnosis, particularly high for small vessel ischemic disease (81.8%) and multiple sclerosis (78.6%) (Table 5). Sensitivity, specificity, positive predictive value, negative predictive value, and diagnostic accuracy were 95.5%, 50.0%, 93.3%, 60.0%, and 90.0%, respectively. These metrics are comparable with published data by Datar et al. (2018)[20], Barkovich et al. (2000)[15], and Malani et al. (2023)[22], underscoring MRI's excellent sensitivity but moderate specificity due to overlapping imaging features across etiologies.

CONCLUSION

Overall findings of this study indicate that MRI is a highly sensitive and reliable tool for the evaluation of white matter disorders. Vascular etiologies, particularly small vessel ischemic disease, were the most common, followed by demyelinating disorders. MRI effectively characterized lesion distribution, pattern, and severity, and showed strong concordance with clinical diagnosis. Age and vascular risk factors were significant determinants of lesion type and distribution. Early MRI assessment facilitates accurate diagnosis, guides management, and aids prognostication in patients with white matter abnormalities.

LIMITATIONS OF THE STUDY

1. The sample size was relatively small and derived from a single tertiary-care center, which may limit the generalizability of the findings.
2. The cross-sectional observational design precludes establishing causal relationships between risk factors and white matter lesions.
3. Long-term clinical outcomes and progression of lesions were not evaluated, limiting insight into the prognostic significance of the findings.

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