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CLINICAL AND RADIOLOGICAL DIFFERENCES BETWEEN MYELIN OLIGODENDROCYTE GLYCOPROTEIN ASSOCIATED DISEASE (MOGAD) AND AQUAPORIN4 NEUROMYELITIS OPTICA SPECTRUM DISORDERS (AQP4 NMOSD)

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ABSTRACT

OBJECTIVE: To assess the Clinical and Radiological differences between Myelin Oligodendrocyte Glycoprotein Associated Disease (MOGAD) and Aquaporin4 Neuromyelitis Optica Spectrum Disorders (AQP4 NMOSD).

METHODOLOGY: The Department of Neurology at the Jinnah Postgraduate Medical Centre (JPMC), Karachi, conducted this prospective cross-sectional study on 55 patients of age 13-55 years from either of gender, who had either anti-MOG or anti-AQP4 antibodies positive and fulfilled NMOSD criteria. Patients excluded from the study included those diagnosed with multiple sclerosis, tuberculosis, systemic lupus erythematosus (SLE), sarcoidosis, and vascular disorders and patients lost to follow-up. Differences in MOGAD and Anti-AQP4-IgG+ NMOSD were stated. The data was interpreted using SPSS version 26.

RESULTS: The mean age of the participants was 33.15±10.42 years. The gender distribution showed the predominance of females, constituting 67.3% of the sample, with males accounting for the remaining 32.7%. In patients who were ANTI MOG AB positive (n=11) and ANTI AQP-4 AB positive (n=29), the clinical features and symptoms were; cervical myelitis was present in 33.3% & 66.7% patients (p=0.0001), dorsal myelitis in 70.0% & 30.0% (p=0.089), optic neuritis in 57.1% & 42.9% (p=0.05), bilateral lower limb weakness in 33.3% & 66.7% (p=0.144) and urinary retention in 50.0% each (p=0.670) whereas the radiological features of patients were; fundoscopy was found normal in 25.9% & 74.1% (p=0.002), CSF findings were as; normal in 7.1% & 92.9% (p=0.0001), raised protein in 14.3% & 85.7% (p=0.032), raised protein, mononuclear pleocytosis in 42.9% & 57.1% (p=0.450), MRI brain was normal in 17.8% & 82.2% (p=0.0001), MRI orbit was normal in 28.6% & 71.4% (p=0.0001) and MRI spine was normal in 33.3% & 66.7% (p=0.205).
CONCLUSION : AQP4+ NMOSD had predominantly cervical myelitis and optic neuritis. On radiographs, Anti-AQP4+ cases had longitudinally extensive transverse myelitis in the cervical region while Anti-MOG+ patients had greater conus involvement and dorsal myelitis. MOGAD patients presented at a younger age as compared to AQP4+. Patients with Anti-AQP4+ showed higher residual disability compared to MOGAD patients. For accurate diagnosis, treatment, and prognosis, these distinctions are crucial. Clinical outcome and quality of life should be improved through research of these continued disorders.

KEYWORDS: Anti Aquaporin-4, MOGAD, Myelin Oligodendrocyte Glycoprotein, Myelitis, NMOSD, Optic Neuritis

INTRODUCTION

Neuromyelitis Optica Spectrum disorders (NMOSD) are autoimmune inflammatory demyelinating conditions affecting central nervous system, characterized by recurrent attacks of severe optic neuritis and/or myelitis as well as diencephalic, brainstem and symptomatic cerebral syndromes [1]. Discovery of antibodies against Myelin Oligodendrocyte Glycoprotein (anti-MOG IgG) and Aquaporin-4 (anti-AQP4 IgG) has led to distinction of NMOSD into two distinct disorders namely anti-AQP4 Immunoglobulin G positive Neuromyelitis Optica Spectrum Disorders (AQP4 NMOSD) and Myelin Oligodendrocyte Glycoprotein Associated Disorder (MOGAD) [2,3]. AQP4 are particularly present in astrocytic processes at the blood-brain barrier, while MOG is present on oligodendrocyte cell surfaces and on myelin sheath outermost surface [4].

About two third patients diagnosed with NMOSD have anti-AQP4 IgG, while rest of one third anti-AQP4 negative patients have anti-MOG IgG [5]. Prevalence of AQP4 NMOSD is reported to be 3.5/ 100,000 in East Asia and in contrast with MOGAD, it is more frequently associated with relapsing course and poor recovery [6]. Magnetic resonance imaging is useful tool to distinguish between CNS inflammatory demyelinating disorders, especially NMOSD and Multiple Sclerosis [7]. Multiple studies have assessed the clinico-radiological among both diseases.

It is evident from literature that AQP4 NMOSD and MOGAD though previously categorized under NMOSD, are two distinct diseases, with different clinical course and distinct pattern of MRI lesions [2,3,5,8,9].

NMOSD is an inflammatory disorder with particular predilection for optic nerve and spinal cord, resulting in optic neuritis and myelitis. Various studies have attempted to demonstrate the differences between clinico-radiological features of MOGAD and AQP4 NMOSD, with contrasting findings regarding female preponderance, presenting phenotype and MRI findings. No study, to our knowledge, has been performed in local population. This study aims to address these questions. Findings of this study will help in better understanding of the conditions.

METHODOLOGY

The Department of Neurology at the Jinnah Postgraduate Medical Centre (JPMC), Karachi, conducted this prospective cross-sectional study on 55 patients of age 13-55 years from either of gender, who had either anti-MOG or anti-AQP4 antibodies positive and fulfilled NMOSD criteria. Patients excluded from the study included those diagnosed with multiple sclerosis, tuberculosis, systemic lupus erythematosus (SLE), sarcoidosis, and vascular disorders and patients lost to follow-up. Differences in MOGAD and Anti-AQP4-IgG+ NMOSD were stated. The data was interpreted using SPSS version 26.

Upon obtaining consent, baseline demographic and clinical data (age, gender, residence, phenotype) were recorded using a predefined form. Following the diagnosis of NMOSD, serological tests for anti-AQP4 IgG and anti-MOG IgG were conducted via cell-based assays. Patients were classified into the AQP4 NMOSD group or MOGAD group based on the results. All patients underwent 3T MRI of the brain, optic nerve, and spinal cord using T1W, T2W, FLAIR, and gadolinium-enhanced sequences, with radiological features documented.

All participants received a standard regimen of intravenous methylprednisolone (1 gram daily for 5 days), followed by a tapering course of oral prednisolone starting at 60 mg daily. If there was no or minimal clinical improvement within 7 days, plasma exchange was initiated, consisting of 5 exchanges over 5 to 7 days. Patients were followed up one month after the onset of the episode, or sooner if symptomatic, with outcome variables including relapse rates, motor, and visual disabilities recorded. All data was analyzed using the Statistical Package for Social Sciences version 26. Descriptive statistics was calculated for demographic variables. Statistical test of significance was applied to compare the MOG antibody and AQP-4 antibody at 5% level of significance.

RESULTS

In this study among the 55 participants, the majority fell within the 13-30 years age group (50.9%), followed by 31-40 years (21.8%), 41-50 years (20.0%), and over 50 years (7.3%). Females comprised 67.3% of the sample, while males accounted for 32.7%. Comorbid conditions included diabetes mellitus (5.5%), hypertension (3.6%), ischemic heart disease (1.8%), hepatitis C (1.8%), pulmonary TB (1.8%), and postpartum (3.6%). Previous attacks were reported as unilateral optic neuritis (1.8%), bilateral optic neuritis (10.9%), dorsal myelitis (9.1%) and cervical myelitis (7.3%). Most participants had a GCS score of 15/15 (94.5%). Disease course was predominantly monophasic (60.0%), followed by relapsing-remitting (34.5%) and chronic progressive (5.5%). Viral prodrome was noted in 38.2% of cases. All participants had no family history of similar conditions. Visual evoked potentials (VEP) were normal in 78.2% and abnormal in 21.8%. Treatment regimens primarily involved pulse therapy (58.2%), with combinations of pulse therapy and other treatments including azathioprine (3.6%), other immunomodulators (3.6%), plasma exchange (5.5%), and rituximab (1.8%). Some received a combination of pulse therapy with immunomodulators (16.4%) or plasma exchange (5.5%). (TABLE 1)

The clinical features and symptoms varied between patients who were ANTI MOG AB positive (n=11) and those who were ANTI AQP-4 AB positive (n=29). Symptomatic cerebral syndrome was present in 100% of anti-AQP-4 positive patients, but none in anti-MOG positive patients. Acute disseminated encephalomyelitis (ADEM) was found exclusively in anti-MOG positive patients (100%), while area postrema syndrome and acute brainstem syndrome were solely observed in anti-AQP-4 positive patients (100%). Cervical myelitis was significantly higher in anti-AQP-4 positive patients (66.7%) compared to anti-MOG positive patients (33.3%) (p=0.0001). Dorsal myelitis was more common in anti-MOG positive patients (70.0%) than in anti-AQP-4 positive patients (30.0%)

(p=0.089). FLAMES syndrome was only seen in anti-MOG positive patients (100%). Optic neuritis was observed in both groups, with a higher prevalence in anti-MOG positive patients (57.1%) than in anti-AQP-4 positive patients (42.9%). Opticospinal syndrome was only seen in anti-AQP-4 positive patients (100%) (p=0.050). Transverse myelitis was equally distributed between both groups (50%).

Regarding clinical symptoms, all four limb weakness was exclusively present in anti-AQP-4 positive patients (100%) (p=0.0001). Bilateral lower limb weakness was more prevalent in anti-AQP-4 positive patients (66.7%) compared to anti-MOG positive patients (33.3%) (p=0.144). Urinary incontinence was seen only in anti-AQP-4 positive patients (100%). Urinary retention was equally observed in both groups (50%). Bilateral vision loss was slightly higher in anti-AQP-4 positive patients (60.0%) compared to anti-MOG positive patients (40.0%). Unilateral vision loss was equally distributed between both groups. Fever was more common in anti-MOG positive patients (80.0%) than in anti-AQP-4 positive patients (20.0%) (p=0.103). Other symptoms were slightly more prevalent in anti-MOG positive patients (54.5%) compared to anti-AQP-4 positive patients (45.5%). (TABLE 2)

The age distribution of patients with Myelin Oligodendrocyte Glycoprotein Associated Disorder (MOGAD) and Aquaporin4 Neuromyelitis Optica Spectrum Disorders (AQP4 NMOSD) shows notable differences. Among patients aged 13-30 years, a significant proportion (55.2%) tested positive for Anti-AQP4 antibodies, compared to 54.5% who tested positive for Anti-MOG antibodies. In the 31-40 year age group, 27.3% of patients were Anti-MOG positive, while 20.7% were Anti-AQP4 positive. For the 41-55 year age group, 18.2% of patients tested positive for Anti-MOG antibodies, whereas 13.8% tested positive for Anti-AQP4 antibodies. Interestingly, there were no Anti-MOG positive patients over the age of 50, whereas 10.3% of the Anti-AQP4 positive patients were in this age group. (Figure 1)

In a comparison of radiological features between anti-MOG antibody positive (n=11) and anti-AQP-4 antibody positive (n=29) patients, significant differences were noted. Fundoscopy showed that anti-AQP-4 positive patients had a higher rate of normal findings (74.1% vs. 25.9% in anti-MOG positive patients, p=0.002). Anti-MOG positive patients showed bilateral optic atrophy (100%) and disc edema, while anti-AQP-4 positive patients had more instances of bilateral disc edema (83.3%, p=0.040) and unilateral optic atrophy (100%, p=0.050).

CSF analysis revealed normal results in 92.9% of anti-AQP-4 positive patients compared to 7.1% of anti-MOG positive patients (p=0.0001). Raised protein levels were more common in anti-AQP-4 positive patients (85.7%, p=0.032).

MRI brain scans showed normal results in 82.2% of anti-AQP-4 positive patients versus 17.8% of anti-MOG positive patients (p=0.0001). Various brain abnormalities, including specific hyperintense signals, were exclusive to either anti-AQP-4 or anti-MOG positive patients.

MRI orbit scans indicated that 71.4% of anti-AQP-4 positive patients had normal findings compared to 28.6% of anti-MOG positive patients (p=0.0001). Hyperintense optic nerve signals were exclusive to anti-AQP-4 positive patients.

In MRI spine analysis, longitudinally extensive transverse myelitis in the cervical region was present only in anti-AQP-4 positive patients (p=0.024). Other spinal abnormalities varied between the two groups. (TABLE 3)

Table 1. Demographic Characteristics of Study Population

Demographic Data		**Frequency**	**Percentage**
Age Group	13 – 30 Years	28	50.9%
	31 – 40 Years	12	21.8%
	41 – 50 Years	11	20.0%
	> 50 Years	4	7.3%
Gender	Male	18	32.7%
	Female	37	67.3%
Comorbid	Diabetes Mellitus	3	5.5%
	Hypertension	2	3.6%
	Ischemic Heart Disease	1	1.8%
	Hepatitis C	1	1.8%
	Pulmonary TB	1	1.8%
	Postpartum	2	3.6%
Previous Attack	Unilateral Optic Neuritis	1	1.8%
	Bilateral Optic Neuritis	6	10.9%
	Dorsal Myelitis	5	9.1%
	Cervical Myelitis	4	7.3%
GCS	13/15	2	3.6%
	14/15	1	1.8%
	15/15	52	94.5%
Disease Course	Chronic Progressive	3	5.5%
	Monophasic	33	60.0%
	Relapsing Remitting	19	34.5%
Viral Prodrome	Yes	21	38.2%
	No	34	61.8%
Family History	None	55	100.0%
VEP	Normal	43	78.2%
	Abnormal	12	21.8%
Treatment	Pulse therapy	32	58.2%
	pulse therapy, azathioprine	2	3.6%
	Pulse therapy, Immunomodulators	2	3.6%
	Pulse therapy, Immunomodulators, Plasma exchange	2	3.6%
	Pulse Therapy, Plasma exchange	3	5.5%
	pulse therapy, rituximab	1	1.8%
	Pulse therapy+ Immunomodulators	9	16.4%
	Pulse therapy+ Plasma exchange	3	5.5%
	Pulsetherapy+ Immunomodulators	1	1.8%

Table 2. Clinical Features of MOG antibody and AQP-4 antibody positive patients

Clinical Features n (%)	**ANTI MOG AB Positive (n=11)**	**ANTI AQP-4 AB Positive (n=29)**	**P-value**
Clinical Diagnosis			
Symptomatic Cerebral Syndrome	0 (0.0%)	1 (100.0%)	>0.05
ADEM	1 (100.0%)	0 (0.0%)	>0.05
Area Postrema Syndrome	0 (0.0%)	1 (100.0%)	>0.05
Acute Brainstem Syndrome	0 (0.0%)	1 (100.0%)	>0.05
Cervical Myelitis	1 (33.3%)	14 (66.7%)	0.0001
Dorsal Myelitis	7 (70.0%)	3 (30.0%)	0.089
FLAMES Syndrome	1 (100.0%)	0 (0.0%)	>0.05
Optic Neuritis	4 (57.1%)	3 (42.9%)	>0.05
Opticospinal Syndrome	0 (0.0%)	3 (100.0%)	0.050
Transverse Myelitis	1 (50.0%)	1 (50.0%)	0.833
Clinical Symptoms			
All four Limb Weakness	0 (0.0%)	14 (100.0%)	0.0001
Bilateral Lower Limb Weakness	4 (33.3%)	8 (66.7%)	0.144
Urinary Incontinence	0 (0.0%)	4 (100.0%)	0.871
Urinary Retention	4 (50.0%)	4 (50.0%)	0.670
Bilateral Vision Loss	2 (40.0%)	3 (60.0%)	>0.05
Unilateral Vision Loss	2 (40.0%)	3 (60.0%)	>0.05
Fever	4 (80.0%)	1 (20.0%)	0.103
Others	6 (54.5%)	5 (45.5%)	>0.05

Figure 1: Comparison of age group between MOG AB & AQP-4 antibody positive patients

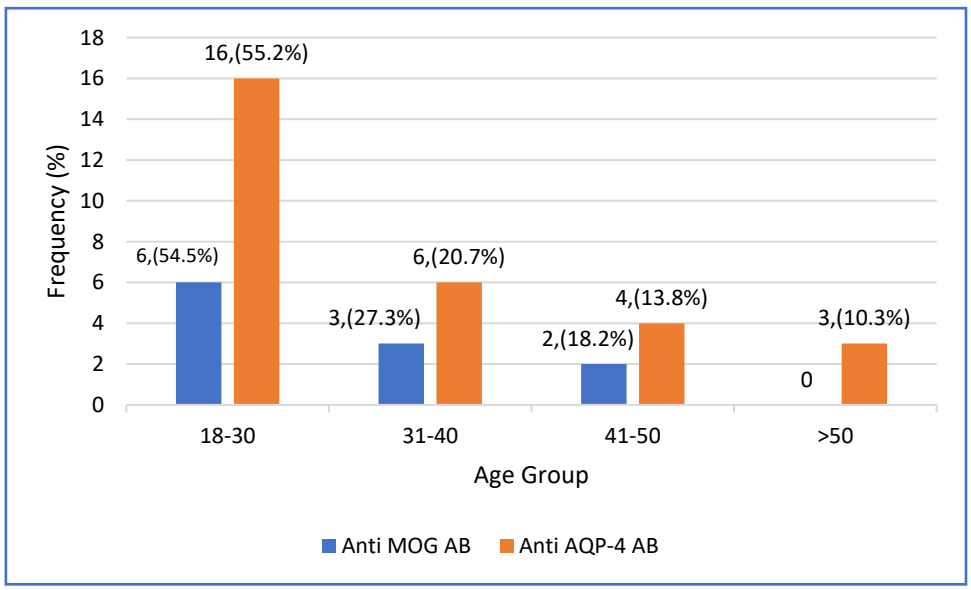

Table 3. Radiological features of MOG antibody and AQP-4 antibody positive patients

Radiological Features n (%)	**ANTI MOG AB Positive (n=11)**	**ANTI AQP-4 AB Positive (n=29)**	**P-value**
Fundoscopy			
Normal	7 (25.9%)	20 (74.1%)	0.002
Bilateral optic atrophy	2 (100.0%)	0 (0.0%)	0.167
Bilateral disc edema	1 (16.7%)	5 (83.3%)	0.040
Unilateral papillitis	1 (50.0%)	1 (50.0%)	0.833
Unilateral optic atrophy	0 (0.0%)	3 (100.0%)	0.050
CSF Findings			
Normal	1 (7.1%)	13 (92.9%)	0.0001
Normal Protein, Mononuclear Pleocytosis	2 (100.0%)	0 (0.0%)	0.200
Raised Protein	1 (14.3%)	6 (85.7%)	0.032
Raised Protein, Mononuclear Pleocytosis	6 (42.9%)	8 (57.1%)	0.450
MRI BRAIN			
Normal	5 (17.8%)	23 (82.2%)	0.0001
Bilateral hyperintense signals in frontal and parietal lobes, corpus callosum	0 (0.0%)	1 (100.%)	>0.05
Bilateral hyperintense signals in temporoparietal lobes	0 (0.0%)	1 (100.%)	>0.05
Bilateral symmetrical fluffy hyperintense signals in temporoparietal lobes	1 (100.0%)	0 (0.0%)	>0.05
Focal subcortical hyperintense signal on T2 and FLAIR in unilateral temporal area	1 (100.0%)	0 (0.0%)	>0.05
Hyperintense signals in bilateral frontoparietal lobes with gyral swelling	1 (100.0%)	0 (0.0%)	>0.05
Hyperintense signals in brainstem in periependymal regions with postcontrast enhancement	0 (0.0%)	1 (100.0%)	>0.05
Hyperintense signals in cerebellum and brainstem periependymal regions with contrast enhancement	0 (0.0%)	1 (100.0%)	>0.05
Hyperintense signals in periaqueductal area with post contrast enhancement	0 (0.0%)	1 (100.0%)	>0.05
Hyperintense signals on T2 in subcortical bilateral parietal, parasagittal regions, pons and cerebellar peduncles	1 (100.0%)	0 (0.0%)	>0.05

Hyperintense signals on T2W and FLAIR involving Bilateral periependymal region and brainstem	0 (0.0%)	1 (100.0%)	>0.05
Mild relative gyral thickening seen in unilateral temporoparietal region on FLAIR images suggestive of focal encephalitis	1 (100.0%)	0 (0.0%)	>0.05
Unilateral poorly demarcated white matter signal in unilateral temporal region	1 (100.0%)	0 (0.0%)	>0.05
MRI ORBIT			
Normal	8 (28.6%)	23 (71.4%)	0.0001
Bilateral hyperintense signals in optic nerve	1 (33.3%)	2 (66.7%)	>0.05
Hyperintense signal in unilateral optic nerve with swelling	0 (0.0%)	0 (0.0%)	-
Hyperintense signals in unilateral optic nerve on T2 and FLAIR	0 (0.0%)	1 (100.0%)	>0.05
Hyperintense signals in unilateral optic nerve on T2 and FLAIR	1 (50.0%)	1 (50.0%)	0.833
Patchy T2 signal abnormality in bilateral optic nerves	0 (0.0%)	1 (50.0%)	>0.05
Patchy T2 signal abnormality in unilateral optic nerve	0 (0.0%)	0 (0.0%)	-
Swelling of retroorbital segment of unilateral optic nerve with perineural enhancement	1 (100.0%)	0 (0.0%)	>0.05
Unilateral hyperintense signals in optic nerve	0 (0.0%)	1 (100.0%)	>0.05
MRI SPINE			
Normal	3 (33.3%)	6 (66.7%)	0.205
Dorsal cord thinning and atrophy	0 (0.0%)	1 (100.0%)	>0.05
Dorsal cord thinning and atrophy,conus involvement	1 (100.0%)	0 (0.0%)	>0.05
Longitudinally extensive transverse myelitis in cervical region	0 (0.0%)	4 (100.0%)	0.024
Longitudinally extensive transverse myelitis, cord expansion in cervical region	2 (16.7%)	10 (83.3%)	>0.05
Longitudinally extensive transverse myelitis, cord expansion in dorsal region	0 (0.0%)	1 (100.0%)	>0.05
Longitudinally extensive transverse myelitis, cord expansion, in dorsal region	1 (100.0%)	0 (0.0%)	>0.05
Longitudinally extensive transverse myelitis, cord expansion, post contrast enhancement in dorsal	2 (66.7%)	1 (33.3%)	>0.05

Longitudinally extensive transverse myelitis, cord swelling in cervical region	0 (0.0%)	1 (100.0%)	>0.05
Longitudinally extensive transverse myelitis, dorsal cord	2 (40.0%)	3 (60.0%)	>0.05
Longitudinally extensive transverse myelitis,cervical region	0 (0.0%)	1 (100.0%)	>0.05
Longitudinally extentensive transvere myelitis in cervicodorsal region	0 (0.0%)	1 (100.0%)	>0.05
Patchy longitudinally extensive signal in dorsal cord	0 (0.0%)	0 (0.0%)	-

DISCUSSION

Myelin Oligodendrocyte Glycoprotein Associated Disease (MOGAD) and Aquaporin4 Neuromyelitis Optica Spectrum Disorders (AQP4 NMOSD) are 2 differents autoimmune inflammatory diseases of the Central Nervous Systemizzes [10]. While the two entities show some clinical and radiological similarities, they also exhibit distinct differences that set them apart in both the clinical practice as well as research [11].

MOGAD presents predominantly in children and younger adults with manifestations ranging from bilateral optic neuritis, ADEM-like episodes or pivoting to myelitis/brainstem involvement [12]. In contrast to the quick course of illness in most children with AQP4 NMOSD,12 adults suffer severe optic neuritis and longitudinally extensive transverse myelitis (LETM) at disease onset often resulting in significant disability[13].

On radiological assessment, while MOGAD lesions are generally smaller and more often involve the cortical or juxtacortical areas (with tumefactive disease seen on occasion) [14]. AQP4 NMOSD, in comparison with nmo+ONM and ON spectrum disease, shares the below distinctive characteristics: Pathognomonic of longitudinally extensive lesions associated with optic nerve and spinal cord involvement which frequently span three or more vertebral segments particularly in the cervical/ thoracic regions [15].

MOGAD frequently correlates with MOG antibodies, while AQP4 NMOSD is characterized notably by AQP4 antibodies. These consequential biomarkers are pivotal for medical diagnosis and treatment decisions [16].

Therapeutic tactics diverge substantially: MOGAD commonly responds well to corticosteroids and may necessitate immunosuppression for relapse deterrence, whereas AQP4 NMOSD demands aggressive immunosuppressive remedies owing to gravity and frequent relapses [17]. Strategies targeting B-cells, such as rituximab, are remarkably useful in AQP4 NMOSD [18].

Our study findings stated that In patients who were ANTI-MOG AB positive (n=11) and ANTI AQP-4 AB positive (n=29), the clinical features and symptoms were; cervical myelitis was present in 33.3% & 66.7% patients (p=0.0001), dorsal myelitis in 70.0% & 30.0% (p=0.089), optic neuritis in 57.1% & 42.9% (p=0.05), bilateral lower limb weakness in 33.3% & 66.7% (p=0.144) and urinary retention in 50.0% each (p=0.670) whereas the radiological features of patients were; fundoscopy was found normal in 25.9% & 74.1% (p=0.002), CSF findings were as; normal in 7.1% & 92.9% (p=0.0001), raised protein in 14.3% & 85.7% (p=0.032), raised protein, mononuclear pleocytosis in 42.9% & 57.1% (p=0.450), MRI brain was normal in 17.8% & 82.2% (p=0.0001), MRI orbit was normal in 28.6% & 71.4% (p=0.0001) and MRI spine was normal in 33.3% & 66.7% (p=0.205).

Rempe T, et al reported that compared to AQP4 NMOSD, optic neuritis in MOGAD was more frequently associated with bilateral optic nerve involvement (54.5% vs 13.9%). On MRI, more frequent involvement of conus medullaris was observed in cases of myelitis in MOGAD (36.4% vs 4.75%) [2]. Similar findings of the more frequent bilateral optic nerve and conus medullaris involvement were reported by Sato DK, et al [3].

Jain RS, et al reported that myelitis was the most common manifestation (72%) in AQP4 NMOSD, while the second most common manifestation was optic neuritis (20%). In MOGAD, the most common manifestation was also myelitis (59%) followed by optic neuritis (18%). AQP4 NMOSD had more female preponderance (80% vs 41%). Bilateral optic neuritis was present in 12% of patients in the AQP4 NMOSD group and 9% of patients in the MOGAD group. There were no significant

differences in the pattern of involvement of the optic nerve (20% vs 18%), cortex (bilateral 0% vs 9%), basal ganglia (bilateral 0% vs 4.5%), thalamus (bilateral 4% vs 9%) and spinal cord (cervical to conus 0% vs 9%) on MRI in AQP4 NMOSD vs MOGAD [5].

Ojha PT, et al in their study reported that the presenting phenotype was bilateral optic neuritis in 43% of MOGAD patients compared to 4% AQP4 NMOSD patients. In comparison, in AQP4 NMOSD patients most common presenting phenotype was transverse myelitis seen in 55% of patients compared to 5% in the MOGAD group. There was no significant difference between both groups (MOGAD vs AQP4 NMOSD) in terms of female preponderance (52% vs 67%). On MRI, in MOGAD more frequent involvement of the cortex was seen (19% vs 0%), while in AQP4 NMOSD more frequent involvement of the medulla was seen (100% vs 17%) [8].

Kitley J, et al in their study, reported that motor disability (58% vs 0%) and visual disability (33% vs 0%) were seen in AQP4 NMOSD compared to MOGAD. 40% patients of AQP4 NMOSD relapsed in follow-up compared to none in the MOGAD group [9].

Comprehending these clinical, radiological, and immunological variances is fundamental for precise diagnosis, therapeutic variety, and prognostic prediction in MOGAD and AQP4 NMOSD. Continuing exploration into their underlying pathophysiological mechanisms pledges to refine diagnostic standards further and optimize remedial interventions, thereby bettering outcomes for patients troubled by these challenging neurological disorders.

CONCLUSION

AQP4+ NMOSD had predominantly cervical myelitis and optic neuritis. On radiographs, Anti-AQP4+ cases had longitudinally extensive transverse myelitis in the cervical region while Anti-MOG+ patients had greater conus involvement and dorsal myelitis. MOGAD patients presented at a younger age as compared to AQP4+. Patients with Anti-AQP4+ showed higher residual disability compared to MOGAD patients. For accurate diagnosis, treatment, and prognosis, these distinctions are crucial. Clinical outcome and quality of life should be improved through research of these continued disorders.

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