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Original Research Article

Prevalence of autoantibodies neuromyelitis optica (NMO igg) and myelin oligodendrocyte glycoprotein (MOG) in NMOSD and the relationship between them in the general population

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ABSTRACT

Background: Neuromyelitis optica spectrum disorder (NMOSD) and anti-myelin oligodendrocyte glycoprotein (anti-MOG) syndromes are inflammatory conditions of the central nervous system that often involve the optic nerves and spinal cord. They can be mistaken for MS due to their similar symptoms. Therefore, we investigated the relationship between NMO antibody (NMO-Ab) and anti-MOG antibody (MOG-Ab) and the positivity of these antibodies in the general Indian population.

Materials and Methods: This retrospective study analyzed 40186 patients for Neuromyelitis Optica Antibodies serum and Myelin Oligodendrocyte Glycoprotein antibodies serum. Additionally, 5762 patients were analyzed specifically for these antibodies in their cerebrospinal fluid. The study included patients of all ages, unaccounting for their clinical history, and was conducted between January 2019 and July 2023 at the Global Reference Lab.

Results: Overall, it was observed that MOG serum antibodies were more prevalent (18.59%) than NMO serum antibodies (8.12%). Females had a higher prevalence of NMO serum antibodies (13.23%) than males (2.16%), whereas the prevalence of MOG serum antibodies was similar in females (14.27%) and males (14.59%). The highest percentage of MOG serum positivity (31.40%) was observed in 1-12 years age group, and for NMO, it was 9.44% in the 19-30 years age group. The overlap in the positivity between NMO serum and CSF was 6.09% while for MOG serum and CSF, it was 3.86%. A concordance of 92.04% was observed for samples tested negative for NMO in serum as well as CSF. Only 2 cases of 259 cases tested for MOG antibodies showed negativity in serum but positivity in CSF.

Conclusion: The study highlights the prevalence of MOG and NMO antibodies in serum and CSF among patients with suspected or known autoimmune neurological disorders, with notable difference in positivity rates between genders for NMO antibodies. These findings underscore the importance of comprehensive antibody testing in the diagnosis and management of demyelinating diseases such as MOG-Ab NMOSD and AQP4-Ab NMOSD. Further research is warranted to explore the clinical implications of these antibodies and their role in guiding therapeutic interventions for affected individuals.

1. Introduction

Neuromyelitis Optica Spectrum Disorder (NMOSD), which includes NMO (also known as Devic's disease), is an autoimmune condition where the immune system targets components of the Central Nervous System (CNS), specifically the optic nerve, brainstem, and spinal cord. These inflammatory disorders are distinguished by episodes of immune-mediated demyelination and axonal injury, primarily affecting the optic nerves and the spinal cord. [1]

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In most cases of NMOSD, patients have a serum immunoglobulin autoantibody called NMO-Ab that targets the astrocyte aquaporin-4 (AQP4) water channel. This leads to recurrent attacks of severe optic neuritis or myelitis. Patients also commonly experience symptoms like pain, headache, depression, fatigue, and sleep disorders. The disease primarily affects young adults with a mean age of 39 years (18–21), and women are more frequently affected than men. In AQP4-seropositive patients, this gender disparity can reach a ratio of up to 10 women for every man affected.[2]

Up to 10% of NMOSD cases occur in children, with initial symptoms typically appearing between ages 10 and 14, but the disease can also affect children as young as 1 or 2 years old.[3]

The prevalence of NMO varies across different populations and geographic regions. According to numerous studies worldwide, NMO prevalence ranges from 0.34 to 10 cases per 100,000 in adults and from 0.06 to 0.22 cases per 100,000 in children.[4]

Several studies have provided estimates of the prevalence of neuromyelitis Optica (NMO) among different racial groups. The highest prevalence has been reported in the black population, with approximately 10 cases per 100,000 people. Asians have the next highest prevalence, at around 3.5 cases per 100,000 people, while the White/Caucasian population has a lower prevalence, with about 1 case per 100,000 people. When looking at specific East Asian populations, it's interesting to note that Japanese individuals have a higher prevalence rate of NMO, with approximately 3.42 cases per 100,000 people, followed by Koreans at 2.56 cases per 100,000 people.[5]

The diagnosis of NMOSD relies on clinical symptoms, a brain MRI that does not meet the criteria for multiple sclerosis (MS), and the presence of serum aquaporin-4 (AQP4) antibodies. AQP4 antibodies are detected in the sera of adult NMOSD patients in approximately 80% of cases.[6]

In certain individuals displaying NMOSD-like symptoms, the presence of antibodies against myelin-oligodendrocyte-glycoprotein (MOG) can be detected when aquaporin-4 antibodies (AQP4-Abs) are not found.[2]

MOG is a protein produced by oligodendrocytes and myelin-producing cells in the CNS. It is a potential autoantigen in Multiple Sclerosis (MS) and acute disseminated encephalomyelitis (ADEM). Different researchers found that NMO IgG was rarely detectable in MOG Ig patients, and anti-MOG Ig was not observed in most NMO IgG-seropositive NMO patients. This MOG Ig patient subpopulation had similar clinical features including a higher proportion of males, fewer relapses, and better recovery than AQP4-seropositive NMO patients despite variations in the ethnicity of the patient population.[7]

A positive MOG Ab has an overall specificity of 98.5% for MOG-associated disease diagnosis. Only 1.5% of healthy controls tested positive for MOG Ab. However, the sensitivity of MOG Ab ranges from 5.1% in MS to 36.4% in ADEM for different demyelinating conditions.[8]

The current International consensus 2015 on diagnostic criteria for NMOSD diseases differentiate between NMOSD with AQP4-Abs and without or unknown AQP4-Abs status and is followed for diagnosis of NMO spectrum disorders.[9]

The early diagnosis of NMOSD is crucial for proper management and improved prognosis and relies on both clinical presentation and laboratory detection of autoantibodies.

We examined the Prevalence of autoantibodies Neuromyelitis Optica (NMO IgG) and Myelin Oligodendrocyte Glycoprotein (MOG) in NMOSD and the relationship between them in the general population.

2. Materials and Methods

This retrospective study was conducted between January 2019 and July 2023 at the Global Reference Lab with approval obtained for the usage of Laboratory Information Management System (LIMS) data.

2.1. Inclusion criteria

In our study, a total of 40186 patients were examined for Neuromyelitis Optica Antibodies serum (NMO) and Myelin Oligodendrocyte Glycoprotein antibodies serum (MOG). Additionally, 5762 patients were analyzed specifically for NMO and MOG antibodies in their cerebrospinal fluid (CSF). The study included patients of all ages, including children and adults, regardless of their clinical history.

NMO and MOG samples of serum and CSF were analyzed using the Indirect Immunofluorescence test with a sample screening dilution of 1:10 on Euroimmun slides. Fluorescence was evaluated under a microscope, with a positive result indicating a specific fluorescence pattern.

2.2. Data analysis

MS Excel was used for data recording. Discrete variables are summarized in terms of frequencies and percentages. For comparison of categorical Variable, Chi square test was used. The statistical analysis was performed using "R Studio version 1.4.1103". A two-tailed p value of <0.05 was considered to be statistically significant.

3. Results

3.1. Overall distribution

A total of 40,186 patients were included in the analysis. Among them, 17,555 patients underwent testing for Myelin Oligodendrocyte Glycoprotein (MOG) antibodies in their serum, while 22,631 patients underwent testing for Neuromyelitis Optica (NMO) antibodies in their serum. Of these patients, 3,264 (18.59%) tested positive for MOG

antibodies, and 1,837 (8.12%) tested positive for NMO antibodies.

Furthermore, among the 5,762 patients whose cerebrospinal fluid (CSF) samples were analyzed, 2,208 were tested for MOG antibodies in their CSF, while 3,554 were tested for NMO antibodies in their CSF. Among them, 98 (4.44%) tested positive for MOG antibodies, and 188 (5.29%) tested positive for NMO antibodies. (Table 1)

3.2. Gender wise distribution

A total of 17,555 patients underwent testing for MOG serum, with 1,526 (14.59%) males and 1,738 (14.27%) females testing positive for MOG antibodies. Similarly, among the 22,631 patients tested for NMO serum, 226 (2.16%) males and 1,611 (13.23%) females tested positive for NMO.

Furthermore, out of 2,208 patients who underwent MOG CSF testing, 45 (4.24%) males and 53 (4.62%) females tested positive. Similarly, among the 3,554 patients who underwent NMO CSF testing, 25 (1.5%) males and 163 (8.63%) females tested positive for NMO. (Table 2)

3.3. Age-wise prevalence

The highest positivity for MOG serum, at 31.40% (634 cases), was observed in the 1-12 years age group, while for NMO, the highest positivity, at 9.44% (521 cases), was noted in the 19-30 years age group. Similarly, the highest positivity for MOG CSF was also observed in the 1-12 years age group, at 10.48% (37 cases), while the highest positivity for NMO CSF was noted in the above 60 years age group, at 6.72% (18 cases). (Table 3)

3.4. Comparison between NMO and MOG Serum and CSF

Out of 40186 patients tested, 17551 underwent serum testing for both NMO and MOG profiles. Among them, 13167 (75.02%) tested negative for both NMO and MOG, and 45 patients tested positive for both. Around 3217 (18.33%) tested negative for NMO serum but positive for MOG serum. (Table 4)

Out of 5762 tested patients, 2208 underwent testing for both NMO and MOG CSF profiles. Among them, 2016 patients (91.30%) tested negative for both NMO CSF and MOG CSF. On the other hand, 95 patients (4.30%) tested negative for NMO CSF, but their results came out positive for MOG CSF. (Table 5).

Relationship between NMO serum and CSF and MOG serum and CSF.

Out of 426 patients who underwent NMO Serum and NMO CSF testing, 393 (92.04%) were negative for both while 26 (6.09%) were positive for both. (Table 6)

Out of the 259 patients who underwent testing for MOG Antibodies in both serum and CSF, 202 (77.99%) tested negative for both, while 10 (3.86%) tested positive for both. (Table 7)

4. Discussion

There is limited data available on optic neuritis (ON) occurrence in the Indian population. Unlike studies conducted in the Western world, there is limited information on the natural course of ON, its correlation with central nervous system (CNS) neuroinflammatory conditions, and the prevalence of sero markers in Indian ON. However, the discovery of recent biomarkers such as NMO Ab and MOG Ab has improved our understanding of the demyelinating disease spectrum. According to studies, the highest prevalence of NMOSD has been reported in black populations, followed by Asians.

In our Indian study, we found that MOG serum antibodies had a higher prevalence at 18.59% compared to NMO serum at 8.12%. This finding is consistent with a study conducted in Chennai by Ambika S et al, which also reported a higher prevalence of MOG (28.08%) than NMO (9.85%) in the Indian population.[10]

In our study, we observed a higher prevalence of NMO antibodies in serum among females (13.23%) compared to males (2.16%). In contrast, there was little disparity between the prevalence of MOG antibodies in serum among females (14.27%) and males (14.59%). A comparable trend was noted for MOG and NMO antibodies in CSF. NMO CSF exhibited a higher positivity rate in females (8.63%) compared to males (1.5%), while the positivity rates for MOG antibody CSF were similar between females (4.62%) and males (4.24%). These findings are consistent with articles on worldwide incidence and prevalence by Victoria Papp et al., which reported the highest prevalence among females, being 2.3–7.6 times greater than that among males, in both Whites and Africans.[4] Our study found that the highest percentage of MOG serum positivity, at 31.40%, was among the age group of 1-12 years, while for NMO, the highest positivity, at 9.44%, was observed in the 19-30 years age group. Similarly, the highest percentage of MOG CSF positivity was also observed in the 1-12 years age group, at 10.48%. However, in 2023, Sara et al had conducted a study on 255 patients, to understand the relevance of MOG Ab in CSF, where it was found that, highest MOG-Ab CSF positivity was seen among adults, and presented more commonly with motor and sensory symptoms.[11] In comparison, in the current study the highest positivity for NMO CSF was noted in the age group above 60 years, at 6.72%. According to a study by Pittock SJ et al., the initial symptoms of this disease typically occur between the ages of 10 and 14. Moreover, children account for up to 10% of NMOSD cases, and the disease can affect even those as young as 1-2 years old.[3] Sara et al in their study further stated that, paired serum and CSF MOG-Ab positivity are common in MOGAD associated with more severe clinical

Table 1: Overall distribution

Test	Negative		Positive		
	N	%	N	%	Total
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies Serum	14291	81.41%	3264	18.59%	17555
NMO (Aquaporin 4) Neuromyelitis Optica Antibodies, Serum	20794	91.88%	1837	8.12%	22631
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies CSF	2110	95.56%	98	4.44%	2208
NMO (Aquaporin 4) Antibody, CSF	3366	94.71%	188	5.29%	3554

N, Number of participants; %, percentage

Table 2: Gender wise distribution

Test	Gender				p-value
	Female		Male		
	N	%	N	%	
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies Serum					
Negative	7571	62.17%	6720	64.26%	0.7802
Positive	1738	14.27%	1526	14.59%	
NMO (Aquaporin 4) Neuromyelitis Optica Antibodies, Serum					
Negative	10564	86.77%	10230	97.84%	< 0.0001
Positive	1611	13.23%	226	2.16%	
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies CSF					
Negative	1094	95.38%	1016	95.76%	0.6654
Positive	53	4.62%	45	4.24%	
NMO (Aquaporin 4) Antibody, CSF					
Negative	1726	91.37%	1640	98.50%	< 0.0001
Positive	163	8.63%	25	1.50%	

N, Number of participants; %, percentage; $p < 0.05$ is considered statistically significant.

Table 3: Age-wise prevalence

Test	Age Group							p-value
	<1 N (%)	1-12 N (%)	13-18 N (%)	19-30 N (%)	31-45 N (%)	46-60 N (%)	>60 N (%)	
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies Serum								
Negative	42 (79.25%)	1145 (64.36%)	1122 (76.38%)	3379 (80.76%)	4346 (83.33%)	2886 (86.33%)	1329(91.09%)	< 0.0001
Positive	11 (20.75%)	634 (35.64%)	347 (23.62%)	805 (19.24%)	873 (16.67%)	457 (13.67%)	130(8.91%)	
NMO (Aquaporin 4) Neuromyelitis Optica Antibodies, Serum								
Negative	54 (94.74%)	1958 (97.07%)	1714 (93.10%)	4999 (90.56%)	6246 (91.01%)	3390 (91.62%)	1774 (92.69%)	< 0.0001
Positive	3 (5.26%)	59 (2.93%)	127 (6.90%)	521 (9.44%)	617 (8.99%)	365 (8.38%)	140 (7.31%)	
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies CSF								
Negative	13 (100%)	316 (89.52%)	171 (95.53%)	484 (95.84%)	578 (96.66%)	379 (97.18%)	166 (99.40%)	< 0.0001
Positive	0 (0%)	37 (10.48%)	8 (4.47%)	21 (4.16%)	20 (3.34%)	11 (2.82%)	1 (0.60%)	
NMO (Aquaporin 4) Antibody, CSF								
Negative	20 (95.24%)	482 (98.57%)	279 (93.31%)	781 (94.10%)	959 (94.58%)	582 (94.17%)	250 (93.28%)	0.0065
Positive	1 (4.76%)	7 (1.43%)	20 (6.69%)	49 (5.90%)	55 (5.42%)	36 (5.83%)	18 (6.72%)	

N, Number of participants; %, percentage; $p < 0.05$ is considered statistically significant.

Table 4: Comparison between NMO and MOG serum

MOG, Serum	NMO, Serum			
	Negative		Positive	
	Frequency	Percentage	Frequency	Percentage
Negative	13167	75.02%	1122	6.39%
Positive	3217	18.33%	45	0.26%

Table 5: Comparison between NMO and MOG CSF

MOG, CSF	NMO, CSF			
	Negative		Positive	
	Frequency	Percentage	Frequency	Percentage
Negative	2016	91.30%	94	4.26%
Positive	95	4.30%	3	0.14%

Note % is calculated by keeping the denominator as the total cases who have done both NMO and MOG. For Serum (n=17551) and CSF(n=2208)

Table 6: Relationship between NMO serum and CSF

NMO Serum	NMO CSF			
	Negative		Positive	
	Frequency	Percentage	Frequency	Percentage
Negative	393	92.04%	0	0.00%
Positive	8	1.87%	26	6.09%

Table 7: Relationship between MOG serum and CSF

MOG Antibodies Serum	MOG Antibodies CSF			
	Negative		Positive	
	Frequency	Percentage	Frequency	Percentage
Negative	202	77.99%	2	0.77%
Positive	45	17.37%	10	3.86%

presentation.[11]

Our research found that 75.02% of the tested patients had negative results for both NMO and MOG antibodies. Only 0.26% of patients tested positive for both NMO and MOG antibodies. On the other hand, 18.33% of the patients tested negative for NMO Serum but positive for MOG serum, while 6.39% were positive for NMO serum but negative for MOG serum. Studies have shown that the presence of serum MOG antibodies alongside negative NMO serum results has significant diagnostic implications, particularly in distinguishing between MOG antibody-associated disease (MOGAD) and other neurological disorders. This scenario suggests a unique clinical profile that warrants further investigation. Moreover, the study identified 4.30% of patients who tested negative for NMO CSF, but their results came out positive for MOG CSF, while 0.14% were positive for both NMO CSF and MOG CSF. These findings were consistent with a study by de Seze et al., in which MOG antibodies were detected in the sera of more than 20% of NMO-seronegative patients, but not in the sera of patients with multiple sclerosis. The presence of MOG antibodies in NMO-seronegative patients, indicate a distinct immunopathological process. In NMO-seronegative patients, these antibodies may be part of a bystander immune response to neural tissue damage rather than directly targeting a specific antigen. Tissue damage can expose various neural proteins, leading to the production of antibodies against them, including MOG. Therefore, in NMO-seronegative patients with tissue damage, MOG antibodies might be generated as a secondary response. To tissue damage.[12] Also, as stated in a study by Zamvil et al, while NMO IgG is considered as the the hallmark serologic marker in most of the cases of NMOSD, NMO IgG is not detected in approximately one-fourth of the patients diagnosed with NMO spectrum disorder (NMOSD) . The sera or CSF of such patients may, however, show the presence of MOG-IgG, which may further impact the clinical presentation, requiring further studies for confirmatory diagnosis and onward treatment.

According to the Public Summary Document released by the Medical Services Advisory Committee in July 2020, the Department contracted Assessment Report (DCAR) recommends two diagnostic options in cases where brain and/or spinal cord MRI is negative or not typical for multiple sclerosis (MS) but indicative of neuromyelitis optica spectrum disorder (NMOSD). Option 1 involves serum AQP4-Ab testing followed by MOG-Ab testing in negative cases. A positive test confirms AQP4-Ab NMOSD,

but if it's negative, serum MOG-Ab testing is recommended. A positive test indicates MOG-Ab NMOSD diagnosis. Option 2 involves concurrent serum AQP4-Ab and MOG-Ab testing. A positive test confirms AQP4-Ab NMOSD or MOG-Ab NMOSD. If both tests are negative, additional testing such as OCB, IgG, or AQP4-Ab testing in CSF is recommended for differential diagnosis of MS or AQP4-Ab NMOSD or MOG-Ab NMOSD.

In our retrospective study, we found that the concordance between NMO serum and CSF was almost 98%, while for MOG serum and CSF, the concordance was around 80%. It was found that only 2 cases that were negative for NMO Ab in serum showed positivity in CSF. The DCAR report, showed that 32% of cases found to be AQP4-Ab positive in serum were not found to be positive in CSF. It is rare to detect AQP4 antibodies in CSF when they have not been detected in serum. Therefore, routine CSF testing for AQP4-Ab testing in seronegative patients is not recommended, which is consistent with the findings of Wingerchuk et al. (2015).[13]

The blood-brain barrier selectively restricts AQP4 antibodies from entering the CSF, leading to lower detection rates compared to serum. AQP4 antibodies are mainly produced in peripheral lymphoid tissues and released into the bloodstream, explaining lower concentrations in the CSF. Serum assays for AQP4-IgG are more standardized and sensitive compared to CSF assays, resulting in potential discrepancies in antibody detection.

Another study by Majed M et al, conducted in the US, found that CSF specimens were negative for AQP4-IgG if serum specimens were negative. The study validates that serum testing is more informative than CSF testing for detecting AQP4-IgG in NMOSD patients, provided assays are standardized and sensitive. Testing CSF for AQP4-IgG offers no additional benefit if serum testing yields a negative result.

Increased CSF tests for AQP4-IgG suggest that neurologists are becoming more aware of autoimmune NMDA receptor encephalitis. However, this guideline may have been inappropriately applied to other inflammatory CNS disorders where serum may be more informative. A critical serum to CSF gradient is required for IgG to penetrate the CNS in pathogenic quantity. Serum AQP4-IgG titers were higher around the attack time, which plausibly explains why antibody detection in CSF was more frequent at that time.[14]

Among the 259 patients who were tested for MOG antibodies in both serum and CSF, only 0.77% showed agreement between MOG antibodies negativity in serum and MOG antibodies positivity in CSF. Extending CSF testing to all patients is unlikely to capture a significant number of additional patients. Therefore, we suggest that CSF MOG-IgG testing should be performed only when clinically indicated. Future research should focus on studying paired serum and CSF samples from patients and controls to better understand the sensitivity and specificity of different methods of measuring CSF MOG-IgG in different patient cohorts.[15]

MOG-IgG antibodies can be found in the serum and may enter the cerebrospinal fluid (CSF) when the blood-brain barrier is breached or during central nervous system (CNS) inflammation. While serum tests are more sensitive, CSF testing can be useful in specific clinical scenarios. Identifying MOG-IgG in the CSF can help diagnose MOGAD in the right clinical context. Its presence at the start of the disease is linked to more severe disability during attacks and more extensive brain and spinal cord involvement. Further research is needed to understand its relevance in non-MS encephalomyelitis and its apparent irrelevance in clinically definite MS patients.[16]

5. Conclusion

In summary, while NMO and MOG antibody-associated diseases share some clinical features and affect the central nervous system, they are distinct conditions with differences in underlying antibodies, clinical manifestations, and treatment approaches. Proper diagnosis and differentiation between the two are essential to guide appropriate treatment and management strategies. NMOSD has been described worldwide, but it seems to be less frequent in White populations compared with Asian and African ones suggesting the importance of genetic factors in disease susceptibility.

In our Indian study, it was found that the prevalence of MOG Ab was higher than NMO Ab. The prevalence of NMO Ab was higher in females compared to MOG Ab. The 19-30 age group showed higher NMO positivity, while MOG was more prevalent in children below 12 years of age. It is crucial to diagnose seropositive patients early on and provide prompt treatment to prevent attacks and deficits.

6. Limitation

The Clinical History required for the retrospective study was not available.

7. Source of Funding

None.

8. Conflict of Interest

None.

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