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Live Cell-Based Flow Cytometry Assay Versus Commercial Cell-Based Indirect Immunofluorescence Assay of Aquaporin-4 Antibody in Neuromyelitis Optica: A Comparative study

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ABSTRACT

Introduction: Neuromyelitis Optica (NMO) is an inflammatory disorder affecting the central nervous system, notably the optic nerve and spinal cord. Seropositive NMO is marked by serum IgG antibodies against aquaporin-4 (AQP4). The accurate identification of AQP4-IgG is crucial for distinguishing NMO from other demyelinating diseases of the central nervous system. However, traditional diagnostic assays have limitations in sensitivity and specificity. Here, we introduce our in-house flow cytometry live cell-based assay (FC-LCBA) for detecting AQP4 antibodies with enhanced sensitivity and specificity. Our objective is to report the accuracy and compare the efficacy of our newly developed in-house FC-LCBA against the commercial cell-based indirect immunofluorescence assay (IIFA) in detecting AQP4 antibodies.

Methods: This single-blind study was approved by the ethical committee and involved 101 serum samples. Twenty-five samples (including retests) from 17 patients evaluated in the NMO spectrum who had at least one positive cell-based IIFA test during the diagnosis or follow-up are tested in parallel with our in-house FC-LCBA and cell-based IIFA. In addition, 36 serum samples from myelin oligodendrocyte glycoprotein-associated disease (MOGAD) patients and 40 serum samples from healthy subjects are also referred for specificity analysis.

Results: Our in-house FC-LCBA displayed superior sensitivity, detecting positive results even when the cell-based IIFA yielded negative results in patients under immunosuppressive treatments. Additionally, FC-LCBA exhibited high specificity for NMO, showing negligible antibody levels in patients with MOGAD diagnosis and healthy individuals. The assay's stability was confirmed through consistent results in retests.

Conclusion: Our in-house FC-LCBA emerges as a promising diagnostic tool for detecting AQP4 antibodies, offering improved sensitivity, specificity, and reliability, instilling confidence in its potential.

Keywords: Aquaporin-4 antibodies, flow cytometry, live cell-based assay, neuromiyelitis optica, serological diagnosis

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INTRODUCTION

Neuromyelitis Optica (NMO), recognized as Devic's Disease, is a rare and potentially severe inflammatory disorder affecting the central nervous system. Originating from 19 th-century descriptions by French neurologist Dr. Eugène Devic, the distinction between NMO and multiple sclerosis (MS) was clarified in 2004 with the identification of antibodies targeting Aquaporin-4 (AQP4), a water channel located in astroglial cells (1–3). These antibodies play a pivotal role in diagnosing and treating NMO characterized by intense inflammation, especially in the optic nerve and spinal cord, but also may manifest in the brainstem, diencephalon, area postrema, and cerebral involvement (4). Since the first identification of AQP4 antibodies, these antibodies have been tested with numerous techniques, some via in-house assays and some via commercial kits with different sensitivities and specificities (5,6). As is now accepted, cell-based assays (CBA) for AQP4-IgG are sensitive and highly specific and thus perform better than tissue-based and protein-based assays (7–12). In Türkiye, AQP4-IgG detection has been primarily conducted through widely available commercial cell-based indirect immunofluorescence assays (IIFA) that use prefixed cells on chips rather than live cells. Regional limitations in the availability of live CBA, primarily due to resource constraints, make cell-based IIFA a more feasible and accessible

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Highlights

- **FC-LCBA enhances AQP4 antibody detection, crucial for accurate NMOSD diagnosis.**
- **FC-LCBA provides improved specificity and diagnostic reliability over classic IIFA tests.**
- **Using live cells preserves native AQP4 structure, minimizing false-negative results.**

alternative in our region. However, the live CBA is a slightly more sensitive technique in some studies due to the presentation of antigens in their natural conformation. Potential threats to the integrity of transfected cells that may occur with cell fixation are not seen in live CBA techniques (13,14). Our developed in-house flow cytometry live cell-based assay (FC-LCBA), when compared to cell-based IIFA, also stands out due to the utilization of flow cytometry that provides significant innovation in AQP4 testing (11,12,15,16,17).

Here, we assessed the samples to report the accuracy and compare the efficacy of our newly developed in-house FC-LCBA by comparing sensitivity among samples from patients whose seropositive status had been previously demonstrated at least once with cell-based IIFA and specificity among samples from patients with myelin oligodendrocyte glycoprotein-associated disease (MOGAD) diagnosis and healthy individuals.

METHODS

Sample collection

One hundred and one serum samples were obtained from the neuroimmunology outpatient clinic of Sancaktepe Sehit Prof. Dr. Ilhan Varank Training and Research Hospital. Twenty-five samples (including retests) from 17 patients evaluated in the NMO spectrum who had at least one positive IIFA test during the diagnosis or follow-up were tested. These samples were also known to be negative for MOG-IgG with commercially available cell-based IIFA tests. Thirty-six serum samples from MOGAD patients (17) fulfilled the international MOGAD Panel proposed criteria (2023) (18), and 40 serum samples from healthy subjects were also referred for specificity analysis.

Generating AQP4 protein over-expressing cells for FC-LCBA

Human codon-optimized full-length AQP4-M23 isoform was amplified from an Addgene (#126464) plasmid, and Not-1 and Ecor1 restriction sites were added with PCR and then cloned into a lentiviral expression vector with multiple cloning site separated by a GFP reporter via an Internal Ribosomal Entry Site (IRES). AQP4 construct was co-transfected into Human Embryonic Kidney 293(HEK-293) T cells at approximately 80% confluency using Lipofectamine 3000 (Invitrogen) as per the manufacturer's protocol. After 24 hours, supernatants were collected and stored at 4°C until another 24 hours had elapsed. Viral supernatants were centrifugated at 500× g for 10 minutes and filtered through a 0.45 µm filter membrane (millipore) to remove cellular debris. The collected viral supernatants were mixed with Polyethylene Glycol (PEG) 3000 on 1:2 ratio and incubated at 4°C overnight. The mixed supernatants were centrifugated at 2000× g for 1 hour at 4°C, and the resulting pellet was resuspended in an appropriate volume of media to obtain 10x concentrated virus. Viral titers were measured by performing a threefold serial dilution and adding the pseudotyped virus to target cells. After 72 hours, the level of pseudotyped infection was determined by counting the number of GFP-positive cells using flow cytometry, and the infection units per ml were calculated. HEK-293 cells were then transduced with AQP4 lentivirus to generate stable HEK-293 cells expressing the AQP4 protein on their surface. The cells were cultured in complete RPMI 1640 medium supplemented with 10% FBS (Atlanta Biologicals, Lawrenceville, GA), 8% GlutaMAX (Life Technologies), 8% sodium pyruvate, 8% MEM vitamins, 8% MEM nonessential amino acid, and 1% penicillin/streptomycin (all from Corning).

AQP4 antibody detection using FC-LCBA

To measure AQP4-IgG levels, we utilized AQP4 (HEK-AQP4), and for the control, GFP (HEK-GFP) expressing 293 HEK cells were used. For assay, 101 serum samples (at a dilution of 1:10, 1:30, 1:100, 1:300, 1:1000, 1:3000, 1:10000, 1:30000) were incubated with 1×10^5 HEK-AQP4 cells for 30 minutes at 4°C. Cells were then washed twice and incubated with fluorochrome-conjugated anti-Human IgG (Biolegend 409306, APC) at a concentration of 2 ug/ml for 30 minutes at 4°C. The cells were rewashed, resuspended in PBS, and analyzed using BD flowcytometry. The data were analyzed using FlowJo software. Before experiments, aliquots of plasma samples were heat-inactivated at 56°C for 30 min.

FC-LCBA analyses were performed in a single-blind manner at Acibadem Human Immunology Center, Istanbul, Türkiye.

AQP4 antibody detection using cell-based IIFA

All samples were also tested in a single-blind manner using available commercial cell-based IIFA according to the manufacturer's protocol (5,6) at a local laboratory in Istanbul, Türkiye.

The researchers who performed the laboratory tests were also blinded to each other's serological status and clinical information.

Data analysis

Statistical analysis determined sensitivity, specificity, and a 95% confidence interval. Cohen's kappa coefficient (K) assessed the degree of agreement between assays. All data were analyzed using GraphPad Prism V9 software.

RESULTS

Determining AQP4 antibody levels, we developed an in-house FC-LCBA that takes advantage of using live cells and flow cytometry. In this assay, we first cloned full-length AQP4 into an expression vector containing GFP reporter. We then co-transfected 293 cells with the AQP4-encoding expression vector and packaging plasmids to produce AQP4 protein lentivirus particles. HEK-293 cells were then efficiently transduced with AQP4 protein pseudovirus encoding GFP, and GFP expressing empty vector was used as control. AQP4 overexpression was confirmed by staining with a patient's plasma who had known AQP4-IgG positive followed by secondary staining with an anti-IgG Fc antibody.

To demonstrate the accuracy and compare the efficacy of our in-house FC-LCBA, we simultaneously measured the levels of AQP4 antibodies in 25 samples (including retests) from 17 patients who had been evaluated in the NMO spectrum and had at least one positive cell-based IIFA during the diagnosis period or follow-up, using in-house FC-LCBA and cell-based IIFA. Samples were added at different dilutions (as mentioned in the method) on those cell lines. Then, AQP4-IgG was measured using anti-IgG Fc-specific secondary antibodies conjugated to a fluorescent tag APC.

Table 1 summarizes the basic demographic data and test results. According to these results, 15 of the 17 patients were cell-based IIFA

positive at symptom onset. The remaining two were cell-based IIFA positive during the follow-up period, and 5 of 17 turned negative within the scope of the study.

As shown in Table 2, in 11 of these 17 patients, both the cell-based IIFA and FC-LCBA tests detected AQP4 antibodies (co-positivity 73.33%), while in two patients, both assays yielded negative results (co-negativity 33.33%). The agreement percentage between the two assays was 76.47% (Cohen's kappa coefficient 0.358; 95% CI -0.130–0.847).

Three patients who were under disease-modifying therapy tested positive for FC-LCBA alone. While 1/300 was the titer that could indicate FC-LCBA and/or IIFA positivity, only FC-LCBA positivity was notable in titers below this titer (Table 1). From one of four discordant samples, IIFA tested positive, while the FC-LCBA tested negative. However, testing the patient's sample with a live cell-based assay (other than cell-based IIFA) for MOG yielded high titer positive results in that patient and confirmed a MOGAD diagnosis fulfilling the international MOGAD Panel proposed criteria. Finally, our in-house FC-LCBA showed a sensitivity of 87.5% (95% CI 61.65–98.45) compared favorably with the cell-based IIFA (sensitivity 68.75%; 95% CI 41.34–88.98).

To assess the specificity of our in-house FC-LCBA for AQP4-IgG, we conducted antibody level measurements on 40 healthy individuals and 36 patients diagnosed with MOGAD. The assay demonstrated negligible antibody levels in both control groups, confirming its high specificity (100%) for detecting AQP4 antibodies. Cell-based IIFA positivity was observed in one of 36 MOGAD patients who was undoubtful MOG positive with high-titers with a live cell-based assay, also fulfilling the international MOGAD Panel proposed criteria. To evaluate the stability of our developed assay, a subset of samples (n=8) were retested at two different time points with FC-LCBA in a single-blind manner. The results revealed high consistency and similarity between the initial measurements.

Table 1. The basic demographic data and the whole test results of seventeen patients

Patient	Sex (F: Female; M: Male)	Age at disease onset	Disease duration (month)	Oligoclonal band (Negative: 0, Positive: 1)	Past IIFA result taken at the time of symptom onset (Negative: 0, Positive: 1)	IIFA results during follow-up (Negative: 0, Positive: 1)	IIFA results within the scope of the study (Negative: 0, Positive: 1)	FC-LCBA results within the scope of the study (Negative: 0, Positive: 1)	Titers (Negative: 0)	Re-test titers (Negative: 0)
1	F	60	57	0	1	NA	0	1	1/30	1/10
2	F	38	100	0	1	NA	0	1	1/300	1/100
3	F	41	52	0	1	NA	0	1	1/30	1/10
4	F	43	112	1	1	NA	1	1	1/300	1/300
5	F	37	52	0	1	NA	1	1	1/300	NA
6	F	54	31	0	1	NA	1	1	1/1000	1/1000
7	M	48	24	1	1	NA	1	1	1/10000	1/3000
8	F	40	160	0	1	NA	1	1	1/10000	NA
9	F	32	36	0	1	NA	1	1	1/1000	NA
10	F	14	52	0	1	NA	1	1	1/1000	NA
11	F	57	72	0	1	NA	1	1	1/10000	NA
12	F	55	3	0	1	NA	1	1	1/10000	NA
13	F	31	136	0	0	1	1	1	1/1000	NA
14	F	41	160	0	0	1	1	1	1/10000	NA
15	F	23	39	0	1	NA	0	0	0	0
16	F	36	64	NA	1	NA	0	0	0	0
17*	F	25	29	0	1	NA	1	0	0	NA

FC-LCBA: The flow cytometric live cell-based assay; IIFA: Indirect immunofluorescence assays.
NA: not applied.
17*: The patient diagnosed with MOGAD within the scope of the study.

Table 2. Detection of AQP4-IgG levels by cell-based IIFA and in house FC-LCBA

	Patient (n)	Comparison	
		Cell-based IIFA	FC-LCBA
At the time of diagnosis Cell-based IIFA (+) (n=15)	9	+	+
	3	-	+
	2	-	-
	1	+	-
At the time of diagnosis Cell-based IIFA (-) (n=2)	2	+	+

FC-LCBA: The flow cytometric live cell-based assay; IIFA: Indirect immunofluorescence assays.

DISCUSSION

Aquaporin-4 is crucial in NMO pathophysiology, with AQP4-IgG being a key disease factor. In the context of a rare disease like NMO, developing and improving a reliable assay is vital and holds substantial implications for clinical diagnosis and treatment decisions (8). In the present study, we demonstrated the sensitivity and specificity of our in-house FC-LCBA. We showed its advantages over the commercial cell-based IIFA in detecting AQP4 antibodies.

In this pilot study, the cell-based IIFA and FC-LCBA tests detected AQP4 antibodies in eleven of these seventeen patients. Both assays yielded negative results in two patients, which resulted in a total agreement of 76.47%. No antibodies were detected in two patients in both tests, suggesting they might genuinely be negative for AQP4-IgG, the tests' sensitivity could have been insufficient, or they might have turned negative by immunosuppressive treatments. Four of seventeen samples showed discrepant results between the cell-based IIFA and FC-LCBA tests. Three patients who were cell-based IIFA positive at disease onset were negative for cell-based IIFA but still positive for FC-LCBA, and they were all definite NMOs. AQP4-IgG remains mostly detectable during remission periods in most patients, but their antibody titers could be low in relapse-free periods under immunosuppressive treatments (19). All three samples of our patients were also taken during such a period and showed low AQP4-IgG titers. This data aligns with previous studies (12,16). It also underscores the importance of testing samples obtained during relapses and before immunosuppressive treatments. Otherwise, retesting with improved techniques should be considered to avoid false negatives.

There are several factors affecting antibody assay sensitivities and specificities. Higher nonspecific bindings to various antigens in prefixed cells than in living cells is one of the most robust reasons (20). In addition, the subjective nature of interpreting IIFA results instead of objective quantification, like evaluating fluorescence intensity and pattern, can also contribute to inconsistencies across different observers (3,16). Isomer selection as the substrate for transfection also stands out as one of the influential factors. The choice of the M23 isomer of AQP4 with its higher orthogonal array of particle formation rather than the M1 isomer may enhance the sensitivity for detection of AQP-IgG, as shown in some of the previous studies (21,22). At this point, we used the M23 isomer of AQP4 rather than M1, and we utilized flow cytometry to provide objective quantification.

The last discrepant data between the four samples was due to the false IIFA positivity observed in a patient whose diagnosis of MOGAD was subsequently confirmed in a live cell-based assay that was negative at first examination with cell-based IIFA for MOG antibody. This case underscores the challenges in MOG antibody testing by cell-based IIFA, as MOG antibodies are conformation-sensitive, and the chemical fixation process of the cells may alter the specific conformational epitopes of the MOG protein and can lead to false negative results (13,14). The live cell-based assay positivity for MOG in that patient confirmed the abovementioned data but also demonstrated the specificity of our assay from a different angle.

In 40 healthy individuals and 36 MOGAD patients, testing with our in-house FC-LCBA has indicated that our test is entirely (100%) specific with a lack of false positivity, unlike IIFA. One of the samples belonging to a definite MOGAD patient tested high titer positive with a live cell-based assay for MOG antibody resulted in low titer AQP4 positive in cell-based IIFA. In this context, the robust specificity of our in-house FC-LCBA enhances its potential as a more reliable diagnostic tool for distinguishing seropositive NMO from MOGAD, inspiring trust for its future use.

Our study demonstrated the robustness and reliability of our assay, as retests consistently yielded similar results in both AQP4-IgG positive and negative patients. This finding underscores the consistent performance of our assay over time.

It is important to note that our study and our in-house FC-LCBA assay have some limitations. Firstly, the number of patients was too small. The purpose of the current study was not to analyze the diagnostic value of these tests. For this reason, patients who already showed AQP4 positivity by IIFA at disease onset or during follow-up were selected. In addition, our in-house FC-LCBA requires special skills, experience, and time, which are valid reasons limiting more centers from using it.

In conclusion, the results' sensitivity, specificity, and reproducibility suggest that our in-house LCBA is one of the best available tests alone or as a complementary technique for AQP4-IgG detection in NMO diagnosis and follow-up. The lack of false positive results, and its enhanced sensitivity among low-titer sera are the most substantial aspects of our assay. However, further studies on large series are urgently needed to advance our understanding and contribute to the evolving landscape of diagnostic methodologies for seropositive NMO and related disorders.

Ethics Committee Approval: The study has received ethical committee approval with the number 25.09.2023/ E-46059653-050.99-225231255 from the Şehit Prof. Dr. İlhan Varank Sancaktepe Eğitim ve Araştırma Hastanesi ethical committee.

Informed Consent: All patients included in the study signed an informed consent form.

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