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A case series of false positive anti-acetylcholine receptor antibody

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Abstract

Myasthenia gravis is an autoimmune neuromuscular disorder typically characterized by fluctuating weakness. Antibody-mediated immunologic attack in the postsynaptic membrane of neuromuscular junction is the key pathophysiology of the disease. Hence, anti-acetylcholine receptor (AChR) antibody test is very specific for myasthenia gravis. However, there are rare occurrences of false positive anti-acetylcholine receptor antibody. Here, we illustrated a case series of three different neurological diseases without typical courses of myasthenia gravis but having positive AChR antibody tests. Patient 1 was a 43-year-old woman presented with progressive lower limb weakness for eight months and bulbar symptom for one month and intubated for respiratory distress. She underwent intensive investigation, received steroid, plasmapheresis but not responded to treatment and required tracheostomy. She was finally diagnosed as motor neuron disease and was discharged with home ventilator. Patient 2 was a 67-year-old woman complained of progressive bilateral eye ptosis and ophthalmoplegia for 6 years without limb weakness and bulbar symptom. She did not respond to pyridostigmine. The repetitive nerve conduction study and single fiber electromyography were normal and was diagnosed as chronic progressive external ophthalmoplegia (CPEO). Patient 3 was a 50-year-old woman with recent tonsillitis presented with bulbar symptom, limbs weakness, ataxia, urinary and bowel retention. She received intravenous immunoglobulin (IVIG) and followed by plasmapheresis and steroid, responding well to treatment. Serial nerve conduction study was performed and she was eventually diagnosed as acute motor sensory axonal neuropathy, a variant of Guillain-Barré syndrome (GBS) with treatment related fluctuation. In conclusion, these cases illustrate the importance of clinical correlation with neurophysiological test. Presence of AChR antibody is not always equivalent to myasthenia gravis.

Keywords: Anti-acetylcholine receptor antibody, myasthenia gravis, motor neuron disease, chronic progressive external ophthalmoplegia, Guillain-Barré syndrome

INTRODUCTION

The anti-acetylcholine receptor (AChR) antibody assay is a cornerstone in the diagnosis of myasthenia gravis (MG), with a reported sensitivity of 85–90% in generalized MG and 44–66% in ocular MG, and specificity approaching 95–100%.[1-3] Despite its high diagnostic utility, false positive result though uncommon have been documented in several neurological and systemic conditions, posing a risk for misdiagnosis and inappropriate immunotherapy. False positivity may arise from technical limitations of enzyme-linked immunosorbent assays (ELISA), cross-reactive antibodies in autoimmune disorders, or disease-related immunological alterations observed in motor neuron disease, mitochondrial myopathies, and immune-mediated neuropathies.[4-7] Misinterpretation of serological results without appropriate clinical correlation can lead to delays in reaching the correct diagnosis, unnecessary exposure to immunosuppression or invasive procedures, and increased healthcare burden.

We present a case series of three patients who were found to have positive AChR antibodies yet were ultimately diagnosed with motor neuron disease, CPEO, and acute motor sensory axonal neuropathy, a variant of GBS with treatment related fluctuation respectively. This case series highlights the importance of

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integrating serological results with clinical features, electrophysiology, and imaging before establishing a diagnosis of MG.

CASE REPORTS

Patient 1

A 43-year-old woman presented to the orthopaedic department with eight months history of progressive weakness in both legs, which started in January 2024. By April 2024, she was wheelchair bound. She also developed difficulty swallowing, slurred speech, and weakness in both arms starting in May 2024. There was no family history of similar symptom and no history of trauma. MRI of her spine showed multiple disc bulges, particularly at the L4/L5 level, but these were not severe enough to explain her symptoms, so the cause was likely not spinal. She was referred to the medical team for further evaluation.

In July 2024, she came to the emergency department with shortness of breath, but without other symptoms like fever, cough, or chest pain. She also denied issues with vision, headache, or bladder/bowel problems. She had no history of smoking or alcohol use. Examination showed normal vital signs. Neurologically, she was alert and oriented, GCS E4V5M6, no ophthalmoplegia, no ptosis. She had signs of neurological issues including horizontal nystagmus, tongue atrophy and fasciculation, exaggerated jaw reflex, and muscle weakness in both upper limbs proximally 2/5 while distally 3/5 as per medical research council muscle grading scale. Meanwhile, her whole lower limbs power 0/5 with flexor plantar reflexes. Both her upper and lower limbs reflexes were absent but intact sensation.

Blood tests for full blood count, renal profile, liver profile, lipid profile, thyroid function test, cortisol, fasting blood glucose, vitamin B12 and folate were normal, and viral screenings for hepatitis B, C and HIV were negative. CT pulmonary angiography showed no evidence of pulmonary embolism. CT brain was reported as hypodensities at bilateral lentiform nuclei suspicious of infarct which was not correlated with her clinical presentation. Her lumbar puncture was normal, and no infection or abnormal cells were found with an opening pressure of 23mmH2O, cell count negative, normal protein and glucose ratio, culture negative, Cryptoccocal antigen negative, acid-fast bacilli stain negative, cytology benign findings, mycobacterium tuberculosis culture no growth.

During her hospital stay, she required increasing respiratory support, starting with nasal-prong oxygen and later non-invasive ventilation. Tests for autoimmune diseases revealed a positive AChR antibody 1.24nmol/L (positive>0.5) by ELISA method, suggesting MG. Hence, she was treated with IVIG and steroid, but her condition did not improve. The diagnosis was reconsidered, and she was treated for refractory MG, undergoing plasmapheresis and additional treatments, though there was still no improvement.

Further tests, MRI brain was done which shown old lacunar infarct at right centrum semiovale and MRI cervical spine shown mild cervical spondylosis which did not fit with her symptoms. After a month of ventilation, she had a tracheostomy and was transferred to a hospital with electrodiagnostic facilities in September 2024 which revealed anterior horn cell disease (motor neuron disease) based on Gold Coast criteria with possible critical illness polyneuropathy.

Based on these results, she was diagnosed with motor neuron disease, and her care was shifted to palliative treatment, including riluzole as well as rehabilitation support. She suffered from infection and pressure sores during her hospital stay, which were treated by the appropriate teams. After almost five months in the hospital, she was discharged in December 2024 with home ventilation.

Nerve conduction study (NCS) revealed sensory fibres are within normal limits meanwhile motor study shown absence of compound motor action potential (CMAP) on all tested nerves. Electromyography (EMG) revealed no spontaneous activity on muscles of lower limbs, except for increased insertional activity. Left abductor pollicis brevis, brachioradialis, biceps shown presence of active denervation (spontaneous activity) with chronic neurogenic changes (increased duration of motor unit action potential (MUAP). Genioglossus showed no spontaneous activity, but increased duration of MUAP. These findings are in keeping with anterior horn cell disease (motor neuron disease) based on Gold Coast criteria with possible critical illness polyneuropathy.

Table 1: Electrophysiology study of Patient 1

Sensory NCS

Nerve/Sites	Rec. Site	Latency ms	Amp Pk-p µV	Dist. cm	Vel. m/s
L Median - Dig II Ortho					
Dig II-Wrist	Wrist	4.01	21.1	13	32.4
R Median - Dig II Ortho					
Dig II-Wrist	Wrist	2.81	26.1	13	46.2
L Ulnar - Digit V Ortho					
V-Wrist	Wrist	2.24	30.6	10	44.7
R Ulnar - Thumb - Digit V Ortho					
V-Wrist	Wrist	1.61	28.0	10	61.9
L Radial – Thumb					
Forearm	Thumb	1.09	25.0	6	54.9
R Radial – Thumb					
Forearm	Thumb	1.72	33.0	10	58.2

Motor NCS

Nerve/Sites	Latency ms	Amp.Pk-p mV	Distance Cm	Lat Diff ms	Velocity m/s	PkDur ms	Area mVms
L Ulnar – above-below elbow							
Wrist	2.71	0.4		2.71		5.21	0.7
Below Elbow	8.96	0.4	19	6.25	30.4	5.57	1.0
Above Elbow	10.83	0.5	10	1.88	53.3	4.53	0.9
				8.13			
R Ulnar – above-below elbow							
Wrist	2.71	0.9		2.71		5.26	2.0
Below Elbow	7.19	0.7	19	4.48	42.4	5.63	1.5
Above Elbow	9.48	0.6	10	2.29	43.6	5.47	1.4
				6.77			
L Median – APB							
Wrist	5.42	0.6		5.42		10.94	2.6
Elbow	11.35	0.3	20	5.94	33.7	11.15	1.1
R Median – APB							
Wrist	NR	NR		NR		NR	NR
Elbow	NR	NR		NR		NR	NR
L Tibial – AH							
Ankle	NR	NR		NR		NR	NR
Knee	NR	NR		NR		NR	NR
R Tibial – AH							
Ankle	NR	NR		NR		NR	NR
L Peroneal – EDB							
Ankle	NR	NR		NR		NR	NR
Fib Head	NR	NR		NR		NR	NR
Knee	NR	NR		NR		NR	NR
L Peroneal – Tib Ant							
Fib Head	NR	NR		NR		NR	NR
Knee	NR	NR		NR		NR	NR

Needle EMG

EMG summary table											
			Spontaneous					MUAP			Recruitment
Muscle	**Nerve**	**Roots**	**IA**	**Fib**	**PSW**	**Fasc**	**H.F.**	**Amp**	**Dur.**	**PPP**	**Pattern**
L Vastus lateralis	Femoral	L2-L4	N	None	None	None	None	N/A	N/A	N/A	N/A
L Gastrocnemius (medial head)	Tibial	S1-S2	N	None	None	None	None	N/A	N/A	N/A	N/A
L Tibialis anterior	Deep peroneal (Fibular)	L4-L5		None	None	None	None	N/A	N/A	N/A	N/A
L Abductor pollicis brevis	Median	C8-T1	N	None	1+	1+	None	N	1+	N	1-
L Abductor digiti minimi (manus)	Ulnar	C8-T1	N	None	None	None	None	N	N	N	N/A
L Brachioradiolis	Radial	C5-C6	N	1+	1+	None	None	N	1+	N	1-
L Biceps brachii	Musculo-cutaneous	C5-C6	N	2+	2+.	None	None	N	1+	N	1-
L Genioglossus	Hypo-glossal	Me-dulla	N	None	None	None	None	N	1+	N	N
R Vastus lateralis	Femoral	L2-L4	N	None	None	None	None	N/A	N/A	N/A	N/A

Patient 2

A 67-year-old woman was referred to the neurology clinic due to gradually ptosis over the past 6 years, along with impaired eye movements. The ptosis did not fluctuate throughout the day, and she had no eye pain, redness, or history of trauma or surgery. Her vision was generally good, and she had no other significant symptoms such as headache, dysphagia, hoarseness, breathless or limb weakness.

Her medical history includes a right hemithyroidectomy in 2019 for a benign thyroid nodule, and she is clinically euthyroid. She also has a family history of ptosis, including her late father, younger brother and children, who all developed ptosis around age of 30s.

Her clinical findings included bilateral partial ptosis with palpebral fissure 2mm with impaired extraocular eye movements. Her visual acuity right eye myopia: unaided 6/24, with pinhole 6/12, left eye myopia with astigmatism and presbyopia: unaided 6/18, with pinhole 6/12, negative bedside fatiguability test and no signs of muscle weakness or myotonia. Otherwise, no other neurological deficit detected.

Investigations done revealed AChR antibody test positive 1.05 nmol/L (positive>0.5), thyroid function, renal and liver profile, and ECG were all normal. Her CT brain and orbit showed no significant findings.

Given the positive AChR antibody result, the patient was started on pyridostigmine 60 mg TDS, but she reported diarrhea and no improvement in ptosis. Further testing included nerve conduction studies and single fiber EMG were normal, ruling out a postsynaptic neuromuscular junction disorder. (Table 2) Also, 4977-bp mitochondrial DNA deletion for chronic progressive external ophthalmoplegia (CPEO) was not detected.

Based on her family history of ptosis and the clinical presentation, she was diagnosed CPEO. She declined further genetic testing and a skeletal muscle biopsy for confirmation. Eventually, she had a referral to an ophthalmologist for eyelid surgery to address the ptosis.

Table 2: Electrophysiology study of Patient 2. The repetitive nerve stimulation is normal. There is no evidence of post-synaptic neuromuscular junction disorder. Single fiber EMG study is normal

Repetitive stimulation

Anatomy/ Train	Ampl. mV	d. Ampl 1%	d. Ampl 2%	Fac Ampl %	Area mVms	d. Area 1 %	d. Area 2%	Fac Area %	Rate Hz	Time
R Abductor digiti minimi (manus)										
Baseline	6.8	0.5	-4	100	20.1	-2.6	-7.2	100	3	0:00:00
Im. Post-Ex	6.5	-0.3	-2.2	96.7	19.9	-4.8	-6.8	99.1	3	0:01:34
@1:00	6.0	1.5	-2.3	89.5	20.2	-3.7	-9.9	101	3	0:02:49
@2:00	5.6	0.9	5.3	82.3	19.0	-3.3	-4.2	94.5	3	0:04:44
@3:00	6.2	-4.4	1.8	91.1	20.0	-5.9	-6.6	99.6	3	0:05:51
R Trapezius (upper)										
Baseline	4.3	-0.1	-1.3	100	30.7	-3.6	-5	100	3	0:00:00
Im. Post-Ex	4.4	-2	-0.1	102	27.6	-4	-2.4	89.9	3	0:01:24
@1:00	4.2	1.6	-4.2	97.6	27.7	-4.4	-7.9	90	3	0:03:32
@2:00	3.9	0.9	0.7	90.4	25.4	-2	-7.3	82.8	3	0:04:58
@3:00	3.8	-1.1	-6.3	89	24.1	-2	-5.4	78.5	3	0:06:02
R Nasalis										
Baseline	0.9	-2.1	-3.3	100	2.7	-9.6	-2.2	100	3	0:00:00
Im. Post-Ex	0.8	2.7	1.8	90	2.2	-10.6	-17.9	81.7	3	0:02:27
@1:00	0.9	2.5	6.6	92.1	2.2	4.9	5	83.7	3	0:04:17
@2:00	0.9	1.2	4.2	94.1	2.3	-1.1	1.4	85.7	3	0:05:15
@3:00	0.9	0.5	2.4	95.4	2.4	0.8	-1.4	88.9	3	0:07:35

Stim SFEMG

Muscle		N	Jitter µs	Block	MIPI µs	MCD µs	MSD µs	FR Hz
R Nasalis	**% Blocked**			0				
	Mean		18	-	6362	18	18	10
	1.1Pair	100	18	-	7019	18	18	10
	1.2 Pair	97	24	-	8425	24	24	10
	2.1 Pair	100	21	-	3349	21	21	10
	3.1 Pair	98	18	-	4850	18	19	10
	4.1 Pair	100	26	-	5971	26	26	10
	5.1 Pair	99	20	-	5929	20	20	10
	6.1 Pair	100	17	-	6112	17	17	10
	7.1 Pair	100	12	-	6603	12	12	10
	7.2 Pair	100	15	-	7142	15	15	10
	7.3 Pair	100	17	-	7569	17	17	10
	8.1 Pair	97	12	-	7008	12	12	10

Patient 3

A 50-year-old woman with a history of diabetes and hypertension was admitted in September 2024 into a private hospital for hoarseness, dysphagia, urinary and bowel retention and ataxic gait with limb weakness. She had recent acute tonsillitis one month ago treated with antibiotic. MRI of the brain done was normal. Also, she had a recent CT neck scan from June 2024 showed osteophytes at the C5-C7 junction causing oesophageal collapse. Hence, an oesophagogastroduodenoscopy was done during admission in September 2024 which revealed a Forrest 3C ulcer at D1 while nasopharyngolaryngoscopy done was normal. Clinical assessment revealed her GCS E4V5M6, she had no ptosis, no ophthalmoplegia, no nystagmus, normal saccade and pursuit. Her upper limbs assessment revealed both muscle tone normal, power right upper limb: shoulder abduction 3+, elbow flexion 4+, wrist dorsiflexion 3+, wrist palmar flexion 3+, finger dorsiflexion 3+, finger palmar flexion 3, hand grip 4; left upper limb: shoulder abduction 3, elbow flexion 4, wrist dorsiflexion 3, wrist palmar flexion 3, finger dorsiflexion 4, finger palmar flexion 3, hand grip 3, areflexia. She had past pointing on left side. Lower limb assessment revealed muscle tone normal, power right lower limb: hip flexion 5, knee extension 5, ankle dorsiflexion 3, ankle plantar flexion 4; left lower limb: hip flexion 4, knee extension 5, ankle dorsiflexion 2, ankle plantar flexion 4, areflexia, plantar reflex downgoing, anal tone intact. However, she had no demonstrable fatiguability.

A nerve conduction study on 1st October 2024 demonstrated sural sparing, demyelinating polyradioculopathy, clinically and neurodiagnostic consistent with GBS. However, she refused lumbar puncture and discharge against medical advice and sought for second opinion in our hospital. Hence, she was readmitted 2weeks later and lumbar puncture done which revealed elevated protein levels in the cerebrospinal fluid, further supporting the GBS diagnosis. However, she also had positive acetylcholine receptor antibody test 0.98nmol/L (positive>0.5) which alerted possible concomitant MG and GBS. Other investigation such as HIV, HBsAg, antiHCV and VDRL were non-reactive, serum paraneoplastic panel for neurological syndrome was negative, tumor marker: CEA, Ca19.9, Ca125 were within normal range, peripheral blood film revealed no rouleax formation, no leukoerythroblastic picture, serum protein electrophoresis shown no paraproteinemia, serum anti-ganglioside antibodies were negative. A repeated electrodiagnostic study in our hospital on 6th November 2024 concluded as findings are in keeping with mixed demyelinating and axonal sensori-motor polyneuropathy. Current study shows deterioration in comparison to study on 1st October 2024 done in private hospital.

Regarding treatment, she received 4 days of 0.5g/kg/day IVIG therapy. However, she didn't show much improvement in neurological function, still required ryles tube feeding. Hence, 3 weeks later, she was admitted for 5 cycles of plasmapheresis using human albumin 5% in alternate day. Then, she was discharged on 17th November 2024 with medication, including prednisolone 1mg/kg/day, T pantoprazole 40mg OD, T metformin 1g BD, T calcium carbonate 500mg BD, T calcitriol 0.25mcg OD, syp lactulose 15ml BD/prn. Throughout her stay, she was managed by a multidisciplinary team, including neurologists, physiotherapists, dietician, and occupational therapists.

Follow-up in December 2024, showed significant improvement in which she could walk (though ataxic), swallow solid foods, and had improved muscle strength in both arms and legs. Hence, she was diagnosed acute motor sensory axonal neuropathy, a variant of GBS with treatment related fluctuation. Reassessment revealed power right upper limb: shoulder abduction 4, elbow flexion 5, wrist dorsiflexion and palmar flexion 5; left shoulder abduction 5, elbow flexion 5, wrist dorsiflexion 4+, wrist palmar flexion 5; right lower limb hip flexion 4, knee extension 5, ankle 5; left hip flexion 5, knee extension 5, ankle 4+. She attended regular physiotherapy.

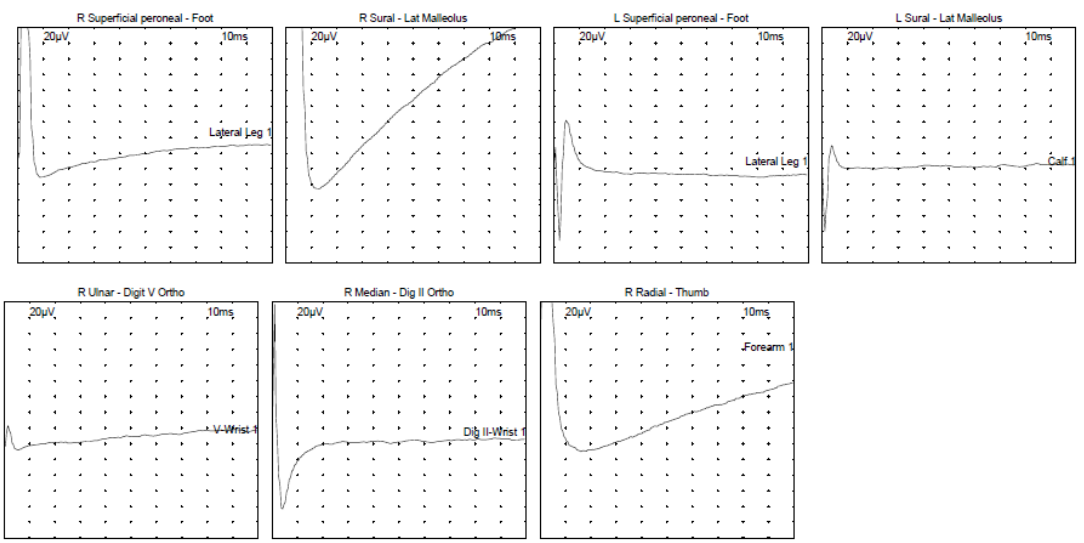
By January 2025, her condition continued to improve, with normal speech and gait, able to return to work. Her prednisolone was tapered off and gabapentin was added for neuropathic symptoms. By March 2025, she was fully recovered with no neurological deficits. A repeat NCS showed improvements. She was discharged to community clinic to continue diabetes follow up.

Sensory study: All tested nerves have no response. Motor study:Right ulnar: low CMAP, prolonged distal motor latency (DML), normal conduction velocity (CV). Right median: low CMAP, prolonged DML, slow CV (demyelinating range). Right tibial: CMAP< 1mV, Left tibial: low CMAP, prolonged DML, slow CV. Bilateral

Table 3: Electrophysiological study of Patient 3. NCS are in keeping with mixed demyelinating and axonal sensori-motor polyneuropathy

Sensory NCS 6th November 2024

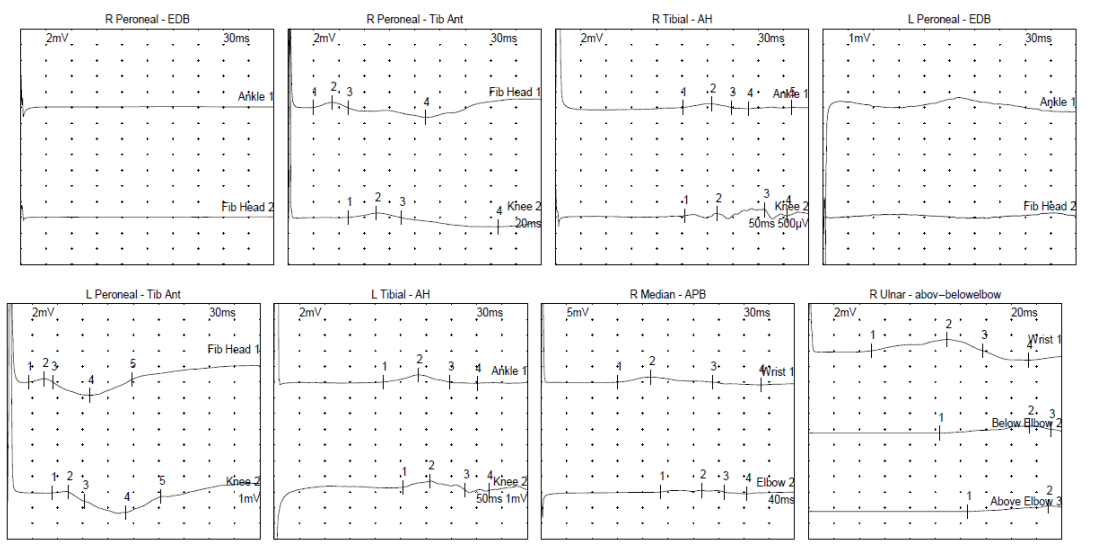
Nerve/Sites	Rec. Site	Latency ms	Amp Pk-P µV
R Median – Dig II ortho			
Dig II – Wrist	Wrist	NR	NR
R Ulnar – Digit V ortho			
V- Wrist	Wrist	NR	NR
R Sural – Lat malleolus			
Calf	Lat Malleolus	NR	NR
L Sural – Lat malleolus			
Calf	Lat Malleolus	NR	NR
R Radial – Thumb			
Forearm	Thumb	NR	NR
R Superficial peroneal – Foot			
Lateral leg	Foot	NR	NR
L Superficial peroneal – Foot			
Lateral leg	Foot	NR	NR



Motor NCS 6th November 2024

Nerve/Sites	Latency ms	Amp.Pk-p mV	Distance cm	Lat Diff ms	Velocity m/s	Pk Dur ms	Area mVms
R Ulnar- above-below elbow							
Wrist	4.95	2.7		4.95		8.80	7.1
Below Elbow	10.36	1.3	25	5.42	46.2	8.75	2.1
Above Elbow	12.55	1.1	10	2.19	45.7	7.81	0.8
				7.60			
R Median – APB							
Wrist	9.06	2.6		9.06		11.20	8.6
Elbow	18.85	1.1	27.5	9.79	28.1	10.05	3.7

R Tibial – AH							
Ankle	15.05	0.6		15.05		5.78	1.6
Knee	25.52	0.1	38	10.47	36.3	15.83	0.9
L Tibial – AH							
Ankle	12.92	1.1		12.92		7.92	3.6
Knee	25.47	0.6	41	12.55	32.7	12.24	3.4
R Peroneal – EDB							
Ankle	NR	NR		NR		NR	NR
Fib head	NR	NR		NR		NR	NR
L Peroneal – EDB							
Ankle	NR	NR		NR		NR	NR
Fib head	NR	NR		NR		NR	NR
R Peroneal – Tib Ant							
Fib head	2.97	1.9		2.97		4.11	1.5
Knee	4.74	1.8	5	1.77	28.2	4.17	1.3
L Peroneal – Tib Ant							
Fib head	2.55	2.1		2.55		2.92	0.7
Knee	5.42	1.3	5	2.86	17.5	3.75	0.6



F Wave 6th November 2024

Nerve	Min F Lat ms	Max F Lat ms	Mean F Lat ms
R Median – APB	NR	NR	NR
R Ulnar – ADM	NR	NR	NR

Needle EMG 6th November 2024

EMG Summary Table											
			Spontaneous					MUAP			Recruitment
Muscle	Nerve	Roots	IA	Fib	PSW	Fasc	HF	Amp	Dur	PPP	Pattern
R Biceps brachii	Musculo-cutaneous	C5-C6	N	None	None	None	None	2+	N	N	Reduced
R First dorsal interosseous	Ulnar	C8-T1	N	None	None	None	None	N	N	N	Reduced
R Abductor digiti minimi (manus)	Ulnar	C8-T1	N	None	None	None	None	N	N	N	Reduced
R Deltoid	Axillary	C5-C6	N	None	None	None	None	2+	N	N	Reduced
R Vastus lateralis	Femoral	L2-L4	N	None	None	None	None	N	N	N	Reduced
R Tibialis anterior	Deep peroneal (Fibular)	L4-L5	N	None	None	None	None	N	N	N	Reduced
R Gastrocnemius (Medial Head)	Tibial	S1-S2	N	None	None	None	None	2+	N	N	Reduced
L Tongue	Hypo-glossal	Medulla	N	None	None	None	None	N	N	N	N

peroneal at EDB: no response. Bilateral peroneal at TA: low CMAP, slow CV (demyelinating range). F waves: Absent at right median & ulnar. EMG: chronic neurogenic changes at right biceps, deltoid and gastrocnemius.

DISCUSSION

This case series illustrates three distinct clinical scenarios in which positive AChR antibodies were misleading, resulting in initial diagnostic uncertainty and, in one case, escalation of therapy for presumed refractory MG. The key message is that AChR antibody positivity alone is insufficient for diagnosis, and over-reliance on serology may result in misdiagnosis.

The first patient presented with progressive bulbar dysfunction, generalized limb weakness, and eventual respiratory failure. Although her AChR antibody was positive (1.24 nmol/L), she exhibited several "red flags" against MG: absence of fatiguability, tongue atrophy and fasciculations, prominent upper motor neuron signs (jaw jerk), symmetrical areflexic quadriparesis, poor or absent response to IVIG, steroids, and plasmapheresis. Electrodiagnostic studies ultimately demonstrated widespread active and chronic denervation fulfilling the Gold Coast criteria for motor neuron disease.[8]

False positive AChR antibodies have been reported in amyotrophic lateral sclerosis (the most common form of motor neuron disease), particularly at low titers.[5,9] Possible mechanisms include nonspecific immune activation or assay cross-reactivity. Thus, in patients with progressive weakness, bulbar involvement, and no electrophysiological evidence of neuromuscular junction disorder, motor neuron disease must be considered despite a positive AChR antibody.

The second patient had chronic, slowly progressive bilateral ptosis and ophthalmoplegia with strong family history, non-fluctuating symptoms, and no bulbar or generalized weakness. Although she tested positive for AChR antibodies (1.05 nmol/L), her electrophysiological evaluation including repetitive nerve stimulation and single fiber EMG were normal, which excluded MG. Lack of improvement with pyridostigmine further argued against MG.

CPEO, a mitochondrial myopathy, is a recognized mimicker of ocular MG. Rare false positive AChR antibody results have been

described in CPEO and other mitochondrial disorders.[6] ELISA-based assays for AChR antibody test are more prone to false positives compared with the radioimmunoprecipitation assay.[10] In this case, clinical phenotype and family history strongly supported CPEO, despite nondiagnostic mitochondrial DNA testing.

The third patient developed subacute bulbar symptoms, limb weakness, areflexia, and gait ataxia one month after an upper respiratory tract infection. Neurophysiology demonstrated demyelinating polyradiculoneuropathy (GBS), and cerebrospinal fluid analysis showed albuminocytologic dissociation. Although AChR antibody was positive (0.98nmol/L), she had no typical clinical course of concomitant MG and GBS as reported.[11] She improved with IVIG and plasmapheresis, consistent with GBS with treatment-related fluctuation. This case reinforces that positive AChR antibodies should not override clear clinical and electrophysiologic evidence of GBS.

In regards to clinical implications, across the three cases, several important themes emerge. First, low-titer AChR antibody results require cautious interpretation. In contrast to nonmyasthenic patients, myasthenic patients were more likely to have an initial clinical presentation raising suspicion for MG and higher mean AChR binding antibody titer.[12] Second, electrodiagnostic testing is crucial. NCS and EMG clarified the diagnoses in all cases and prevented mislabeling patients as refractory MG. Third, serology must never supersede clinical presentation. MG is characterized by fatiguability, fluctuating weakness, preserved reflexes, and electrophysiologic neuromuscular junction transmission defects however none of which were present in these patients. Fourth, over-treatment with immunotherapy carries risks. In patient 1, exposure to IVIG, high-dose steroids, and plasmapheresis delayed the correct diagnosis and increased morbidity.

In conclusion, this case series highlights the diagnostic pitfalls associated with relying solely on positive AChR antibody results. False positivity may occur in motor neuron disease, mitochondrial myopathies, and acute immune-mediated neuropathies such as GBS. Accurate diagnosis requires careful correlation with clinical features, electrophysiology, and imaging. Clinicians should exercise caution when interpreting low-titer AChR antibody results as presence of AChR antibody is not always equivalent to myasthenia gravis.

DISCLOSURE

Ethics: Consent for publication obtained from all the patients.

Conflict of interest: None

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