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Tracy I. George, MD, Chief Medical Officer

Patient Report

Patient Age/Gender: Unknown

Specimen Collected: 02-Sep-20 12:39

Autoimmune Neuromuscular Junction Rflx | **Received: 02-Sep-20 12:44** | **Report/Verified: 02-Sep-20 13:24**

	Result	Units	Reference Interval
Striated Muscle Antibodies,IgG Screen	**Detected** * t1 i1		<1:40
Acetylcholine Binding Antibody	**0.5** H i2	nmol/L	0.0-0.4
Acetylcholine Blocking Antibody	**27** H i3	%	0-26
P/Q-Type Calcium Channel Antibody	24.5 i4	pmol/L	0.0-24.5
Voltage-Gated Potassium Channel Ab, Ser	**32** H i5	pmol/L	0-31
Titin Antibody	0.45 i6	IV	0.00-0.45
N-Type Calcium Channel Antibody	69.9 i7	pmol/L	0.0-69.9
Ganglionic Acetylcholine Receptor Ab	8.4 i8	pmol/L	0.0-8.4

Striated Muscle Abs, IgG Titer | **Received: 02-Sep-20 12:44** | **Report/Verified: 02-Sep-20 13:24**

	Result	Units	Reference Interval
Striated Muscle Antibodies,IgG Titer	**1:40** *		<1:40

Acetylcholine Receptor Modulating Ab | **Received: 02-Sep-20 12:44** | **Report/Verified: 02-Sep-20 13:27**

	Result	Units	Reference Interval
Acetylcholine Modulating Antibody	44 i9	%	<=45

LGI1/CASPR2 Abs IgG w/Rflx to Titer, Ser | **Received: 02-Sep-20 12:44** | **Report/Verified: 02-Sep-20 13:27**

	Result	Units	Reference Interval
CASPR2 Ab IgG Screen by IFA,Serum	<1:10 t2 i10		<1:10
LGI1 Ab IgG Screen by IFA,Serum	<1:10 t3 i11		<1:10

Interpretive Text

t1: 02-Sep-20 12:39 (Striated Muscle Antibodies, IgG Screen)

Striated Muscle Antibodies, IgG detected. Titer results to follow.

t2: 02-Sep-20 12:39 (CASPR2 Ab IgG Screen by IFA, Serum)

CASPR2 Antibody, IgG is not detected. No further testing will be performed.

t3: 02-Sep-20 12:39 (LGI1 Ab IgG Screen by IFA, Serum)

LGI1 Antibody, IgG is not detected. No further testing will be performed.

*=Abnormal, #=Corrected, C=Critical, f=Result Footnote, H=High, i=Test Information, L=Low, t=Interpretive Text, @=Performing Lab

Test Information

i1: Striated Muscle Antibodies, IgG Screen

INTERPRETIVE DATA: Striated Muscle Antibodies, IgG Screen

In the presence of acetylcholine receptor (AChR) antibody, striated muscle antibodies, which bind in a cross-striational pattern to skeletal and heart muscle tissue sections, are associated with late-onset myasthenia gravis (MG). Striated muscle antibodies recognize epitopes on three major muscle proteins, including: titin, ryanodine receptor (RyR) and Kv1.4 (an alpha subunit of voltage-gated potassium channel [VGKC]). Isolated cases of striated muscle antibodies may be seen in patients with certain autoimmune diseases, rheumatic fever, myocardial infarction, and following some cardiotomy procedures.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement A: aruplab.com/CS

i2: Acetylcholine Binding Antibody

INTERPRETIVE INFORMATION: Acetylcholine Binding Ab

Negative ..... 0.0 - 0.4 nmol/L
Positive ..... 0.5 nmol/L or greater

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

i3: Acetylcholine Blocking Antibody

INTERPRETIVE INFORMATION: Acetylcholine Blocking Ab

Negative ..... 0-26 percent blocking
Indeterminate ..... 27-41 percent blocking
Positive ..... 42 percent or greater blocking

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with

Test Information

i3: Acetylcholine Blocking Antibody

clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

i4: P/Q-Type Calcium Channel Antibody

INTERPRETIVE INFORMATION: P/Q-Type Calcium Channel Antibody

0.0 to 24.5 pmol/L ..... Negative
24.6 to 45.6 pmol/L ..... Indeterminate
45.7 pmol/L or greater..... Positive

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

i5: Voltage-Gated Potassium Channel Ab, Ser

INTERPRETIVE INFORMATION: Voltage-Gated Potassium Channel (VGKC) Antibody, Serum

Negative ..... 31 pmol/L or less
Indeterminate... 32 - 87 pmol/L
Positive ..... 88 pmol/L or greater

Voltage-Gated Potassium Channel (VGKC) antibodies are associated with neuromuscular weakness as found in neuromyotonia (also known as Issacs syndrome) and Morvan syndrome. VGKC antibodies are also associated with paraneoplastic neurological syndromes and limbic encephalitis; however, VGKC antibody-associated limbic encephalitis may be associated with antibodies to leucine-rich, glioma-inactivated 1 protein (LGI1) or contactin-associated protein-2 (CASPR2) instead of potassium channel antigens. A substantial number of VGKC-antibody positive cases are negative for LGI1 and CASPR2 IgG autoantibodies, not all VGKC complex antigens are known. The clinical significance of this test can only be determined in conjunction with the patient's clinical history and related laboratory testing.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

i6: Titin Antibody

INTERPRETIVE INFORMATION: Titin Antibody

Negative ..... 0.00 - 0.45 IV
Indeterminate ... 0.46 - 0.71 IV
Positive ..... 0.72 IV or greater

Test Information

i6: Titin Antibody

The presence of titin antibody is associated with late onset of myasthenia gravis (MG) and a variable risk for thymoma. Titin antibody may be detected in 20-40 percent of all patients with MG; higher frequency in older population as a whole.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

i7: N-Type Calcium Channel Antibody

INTERPRETIVE INFORMATION: N-Type Calcium Channel Antibody

0.0 to 69.9 pmol/L .....Negative
70.0 to 110.0 pmol/L .....Indeterminate
110.1 pmol/L or greater.....Positive

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

i8: Ganglionic Acetylcholine Receptor Ab

REFERENCE INTERVAL: Ganglionic Acetylcholine Receptor Ab

Negative . . . . . 0.0-8.4 pmol/L
Indeterminate. . . . . 8.5-11.6 pmol/L
Positive . . . . . 11.7 pmol/L or greater

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

i9: Acetylcholine Modulating Antibody

INTERPRETIVE INFORMATION: Acetylcholine Modulating Ab

Negative ..... 0-45 percent modulating
Positive ..... 46 percent or greater modulating

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

i10: CASPR2 Ab IgG Screen by IFA, Serum

INTERPRETIVE INFORMATION: CASPR2 Ab IgG w/Reflex to Titer,

Test Information

i10: CASPR2 Ab IgG Screen by IFA, Serum
Serum

Contactin-associated protein-2 (CASPR2) IgG antibody may occur as part of the voltage-gated potassium channel (VGKC) complex antibodies.

The presence of CASPR2 IgG antibody is associated with a wide spectrum of clinical manifestations, including acquired neuromyotonia, limbic encephalitis, painful neuropathy and Morvan syndrome. Tumors such as thymoma, small-cell lung cancer, and other rarer tumors may occur. The full-spectrum of clinical disorders and tumors associated with the CASPR2 IgG antibody continues to be defined. Results should be interpreted in correlation with the patient's clinical history and other laboratory findings.

This indirect fluorescent antibody assay utilizes contactin-associated protein-2 (CASPR2) transfected cell lines for the detection and semi-quantification of the CASPR2 IgG antibody.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

i11: LGI1 Ab IgG Screen by IFA, Serum
INTERPRETIVE INFORMATION: LGI1 Ab IgG w/Reflex to Titer,
Serum

Leucine-rich, glioma-inactivated 1 protein (LGI1) IgG antibody may occur as part of the voltage-gated potassium channel (VGKC) complex antibodies.

The presence of LGI1 IgG antibody is mainly associated with limbic encephalitis, hyponatremia and myoclonic movements. LGI1 IgG antibody is rarely associated with tumors but may occur infrequently in Morvan syndrome, neuromyotonia and idiopathic epilepsy. The full-spectrum of clinical disorders associated with the LGI1 IgG antibody continues to be defined. Results should be interpreted in correlation with the patient's clinical history and other laboratory findings.

This indirect fluorescent antibody assay utilizes leucine-rich, glioma-inactivated 1 protein (LGI1) transfected cell lines for the detection and semi-quantification of the LGI1 IgG antibody.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS