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PATIENT REPORT

Patient Age/Sex: Unknown

Specimen Collected: 23-Jun-23 11:34

Autoimmune Neuromuscular Junction Rflx | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:36**

Procedure	Result	Units	Reference Interval
Striated Muscle Antibodies,IgG Screen	**Detected** * t1 i1		[<1:40]
Acetylcholine Binding Antibody	**1.0** H f1 i2	nmol/L	[0.0-0.4]
Acetylcholine Blocking Antibody	**35** H i3	%	[0-26]
P/Q-Type Calcium Channel Antibody	**35.0** H i4	pmol/L	[0.0-24.5]
Voltage-Gated Potassium Channel Ab,Ser	**40** H i5	pmol/L	[0-31]
Titin Antibody	**1.00** H i6	IV	[0.00-0.45]
N-Type Calcium Channel Antibody	**80.0** H i7	pmol/L	[0.0-69.9]
Ganglionic Acetylcholine Receptor Ab	**10.0** H i8	pmol/L	[0.0-8.4]

Striated Muscle Abs, IgG Titer | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:36**

Procedure	Result	Units	Reference Interval
Striated Muscle Antibodies,IgG Titer	**1:160** * i9		[<1:40]

Acetylcholine Receptor Modulating Ab | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:37**

Procedure	Result	Units	Reference Interval
Acetylcholine Modulating Antibody	**50** H i10	%	[<=45]

LGI1/CASPR2 Abs IgG CBA w/Rflx, Ser | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:37**

Procedure	Result	Units	Reference Interval
CASPR2 Ab IgG CBA-IFA Screen, Serum	**Detected** * t2 i11		[<1:10]
LGI1 Ab IgG CBA-IFA Screen,Serum	**Detected** * t3 i12		[<1:10]

CASPR2 Ab IgG Titer by CBA-IFA, Ser | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:37**

Procedure	Result	Units	Reference Interval
CASPR2 Ab IgG CBA-IFA Titer, Serum	**1:20** * i13		[<1:10]

LGI1 Ab IgG Titer by CBA-IFA, Ser | **Received: 23-Jun-23 11:34** | **Report/Verified: 23-Jun-23 11:37**

Procedure	Result	Units	Reference Interval
LGI1 Ab IgG CBA-IFA Titer,Serum	**1:80** * i14		[<1:10]

Interpretive Text

t1: 23-Jun-23 11:34 (Striated Muscle Antibodies, IgG Screen)

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

t2: 23-Jun-23 11:34 (CASPR2 Ab IgG CBA-IFA Screen, Serum)

CASPR2 Antibody, IgG is detected. Titer results to follow.

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

Interpretive Text

t3: 23-Jun-23 11:34 (LGI1 Ab IgG CBA-IFA Screen, Serum)

LGI1 Antibody, IgG is detected. Titer results to follow.

Result Footnote

f1: Acetylcholine Binding Antibody

Acetylcholine receptor binding antibody result is positive. Sample will reflex to modulating antibody testing.

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.

This test was developed and its performance characteristics determined by ARUP Laboratories. It has not been cleared or approved by the US Food and Drug Administration. This test was performed in a CLIA certified laboratory and is intended for clinical purposes.

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.

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Test Information

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

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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

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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

Test Information

i5: Voltage-Gated Potassium Channel Ab, Ser

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.

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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

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.

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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

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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 Information

i8: Ganglionic Acetylcholine Receptor Ab

This test was developed and its performance characteristics determined by ARUP Laboratories. It has not been cleared or approved by the US Food and Drug Administration. This test was performed in a CLIA certified laboratory and is intended for clinical purposes.

i9: Striated Muscle Antibodies, IgG Titer

INTREPRETIVE INFORMATION: Striated Muscle Abs, IgG Titer

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i10: 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.

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i11: CASPR2 Ab IgG CBA-IFA Screen, Serum

INTERPRETIVE INFORMATION: CASPR2 Ab IgG CBA-IFA Screen, 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.

Test Information

i11: CASPR2 Ab IgG CBA-IFA Screen, Serum

This indirect fluorescent antibody assay utilizes CASPR2 transfected cell lines for the detection and semiquantification of the CASPR2 IgG antibody.

This test was developed and its performance characteristics determined by ARUP Laboratories. It has not been cleared or approved by the US Food and Drug Administration. This test was performed in a CLIA certified laboratory and is intended for clinical purposes.

i12: LGI1 Ab IgG CBA-IFA Screen, Serum

INTERPRETIVE INFORMATION: LGI1 Ab IgG CBA-IFA Screen, 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 LGI1 transfected cell lines for the detection and semiquantification of the LGI1 IgG antibody.

This test was developed and its performance characteristics determined by ARUP Laboratories. It has not been cleared or approved by the US Food and Drug Administration. This test was performed in a CLIA certified laboratory and is intended for clinical purposes.

i13: CASPR2 Ab IgG CBA-IFA Titer, Serum

INTERPRETIVE INFORMATION: CASPR2 Ab IgG CBA-IFA Titer, Serum

This test was developed and its performance characteristics determined by ARUP Laboratories. It has not been cleared or approved by the US Food and Drug Administration. This test was performed in a CLIA certified laboratory and is intended for clinical purposes.

i14: LGI1 Ab IgG CBA-IFA Titer, Serum

INTERPRETIVE INFORMATION: LGI1 Ab IgG CBA-IFA Titer, Serum

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