nosed with generalized MG (7,8). Studies have shown that patients with MG and MUSK antibodies have a better response to prednisone and other immune therapies, including rituximab and a poor response to plasma exchange (2,7). Currently, two antibodies are known to be specific for and play a causative role in MG: anti-AchR antibodies and antiMUSK antibodies but, among patients with MG, antibodies to potassium channels, agrin and the ryanodin receptor have been detected (2,4). Patients with dSNMG should be tested for anticortactin antibodies too, but a test that could find these antibodies is not yet available (1). Some recent studies have concluded that the serological examination of antibodies and single-fiber electromyography confirm the diagnostic but they do not correlate with disease activity and response to treatment (5).

Myasthenia gravis it is diagnosed based on one or more of the following criteria:

1. positive results of AchR antibodies or anti-MUSK antibodies;
2. electrophysiological findings that confirm a postsynaptic neuromuscular junction disorder and
3. positive therapeutic response to cholinesterase inhibitors (1). In dSNMG, the diagnosis is confirmed by abnormal findings on a neurophysiological exam and typical decremental responses at repetitive nerve stimulation testing (1).

The association of another autoimmune disorders is very rare and appears in about 13-22% of the patients diagnosed with MG (9).

Thymic hyperplasia can worsen the disorder by initiating the immune response and producing antibodies, especially AchR antibodies (2). It is becoming clear that MG is not a single disease but a number of clinical subtypes that may be distinguished by their auto-antibody profile, as well as by thymic pathology and clinical presentation (2).

CONCLUSIONS

MG is a rare autoimmune disease that sometimes can be associated with other autoimmune disorders, most frequently autoimmune thyroiditis. It is specified in literature that in most cases, patients with thymic hyperplasia have anti-AchR antibodies. Our case was diagnosed with thymic hyperplasia but no antibodies were found in patient`s plasma (dSNMG). When it is possible, the patient should be tested for anti-cortactin antibodies. This would not give a prognostic to the case, but it could help describing subtypes of MG related to the antibodies found in patient`s plasma.

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REFERENCES

1. **Cortés-Vicente E., Gallardo E., Ángeles Martínez M., Díaz-Manera J. et al.** Clinical Characteristics of Patients With Double-Seronegative Myasthenia Gravis and Antibodies to Cortactin, Jama Neurology, 2016;73(9):1099-104. doi: 10.1001/jamaneurol.2016.2032.
2. **Meriggioli M., Sanders D.** Muscle autoantibodies in myasthenia gravis: beyond diagnosis? *Expert Rev Clin Immunol.* 2012 Jul; 8(5): 427–438. doi: 10.1586/eci.12.34
3. **Meriggioli M., Sanders D.** Autoimmune myasthenia gravis: emerging clinical and biological heterogeneity, *Lancet Neurol.* 2009 May; 8(5): 475–490. doi: 10.1016/S1474-4422(09)70063-8
4. **Suzuki S., Utsugisawa K.,Nagane Y.** Three Types of Striational Antibodies in Myasthenia Gravis, Autoimmune Diseases Volume 2011 (2011), Article ID 740583, 2011/740583
5. **Gilhus N.E., Verschuuren J.J.** Myasthenia gravis: subgroup classification and therapeutic strategies, *Lancet Neurology,* 2015; 14(10):1023-1036.
6. **Rodríguez Cruz P.M., Al-Hajjar M., Huda S. et al.** Clinical features and diagnostic usefulness of antibodies to clustered acetylcholine receptors in the diagnosis of seronegative myasthenia gravis, *Jama Neurology,* 2015; 72(6):642-649.
7. **Daroff R.B., Fenichel G.M., Jankovic J.** Bradley's Neurology in clinical practice, 2015
8. **Conti-Fine B.M., Milani M., Kaminski H.J.** Myasthenia gravis: past, present, and future, *J Clin Invest,* 2006; 116(11):2843-2854.
9. **Nacu A., Andersen J.P., Lisnic V. et al.** Complicating autoimmune diseases in myasthenia gravis: a review, published online 2015 doi: 10.3109/08916934.2015.1030614.