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Case Report

A Case of Myasthenia Gravis in a 52-Year-Old Man

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Abstract

Myasthenia gravis (MG) is one of the most frequent neuromuscular diseases, its etiopathogenesis is autoimmune, acquired and its typical clinical triad is: ptosis, diplopia and fatigability of proximal predominance. In addition to weakness of the facial and proximal muscles, it can be associated with thymomas, its prognosis will depend on a rapid diagnosis and treatment.

In this case that we present, the patient had visited the emergency room several times due to proximal weakness. The approximate prevalence is 8 - 15 cases per 100,000 inhabitants [1-5]. Its age of presentation has two peaks of greater incidence between 20 - 25 and another peak from 50. It is more common in women. In women, it usually appears at an earlier age. In men it is later. It is the best characterized autoimmune disease. It is generally predominantly proximal.

Keywords: *Myasthenia Gravis (MG); Neuromuscular Diseases; Thymomas*

Introduction

Myasthenia gravis (MG) is one of the most frequent neuromuscular diseases, its etiopathogenesis is autoimmune, acquired and its typical clinical triad is: ptosis, diplopia and fatigability of proximal predominance. In addition to weakness of the facial and proximal muscles, it can be associated with thymomas, its prognosis will depend on a rapid diagnosis and treatment.

In this case that we present, the patient had visited the emergency room several times due to proximal weakness. The approximate prevalence is 8 - 15 cases per 100,000 inhabitants [1-5]. Its age of presentation has two peaks of greater incidence between 20 - 25 and another peak from 50. It is more common in women. In women, it usually appears at an earlier age. In men it is later. It is the best characterized autoimmune disease. It is generally predominantly proximal.

Myasthenia gravis is a postsynaptic disorder, autoimmune attack of anti-Ach receptor antibodies (acetylcholine), selective attack against acetyl choline (Ach) receptors of the postsynaptic terminal, weakness is aggravated with exercise and improves with cold, hence the Importance of the ice test to verify if the eyelid ptosis improves somewhat in this type of patient. The diagnosis is made based on the neurological examination and is confirmed through immunological and neurophysiological study [1,4]. We will focus our study more on the electroneurographic and EMG analysis (proximal and distal repetitive stimulation and EMGFU) of our patient in order also to improve the training of specialists who perform this technique, I have had lucky to be able to do about 1500 - 1700 SF-EMG (single fiber electromyography) in the last 18 years of professional experience.

It usually affects the cranial nerves. MG (myasthenia gravis) can mimic any pattern of supra or infranuclear oculomotor palsy. It is relatively common in our environment for patients with diplopia and or ptosis to request the EMG jitter protocol (EMGFU) and repetitive stimulation (ER).

MG usually presents with weak facial mimicry, ptosis, diplopia, fatigue, in our patient he often referred to us that he had serious difficulties in closing his mouth. It is perhaps the symptom that our patient told us the most. Weakness of speech, chewing, swallowing, weakness that can reach the respiratory muscles, as well as the proximal muscles of the extremities. In the EMG, it is possible to verify the decreases in repetitive stimulation, not only of the facial but also of the spinal accessory nerve, picking up the trapezius muscle, in addition to the typical facial protocol [4]. The initial involvement of MG is usually the facial and proximal muscles.

Within the neuromuscular diseases of neuromuscular transmission, the following should be highlighted:

1. Postsynaptic: Myasthenia Gravis.
2. Presynaptic: Eaton Lambert Syndrome and Botulism
3. Pre or postsynaptic: congenital myasthenic syndromes and due to drugs [5].

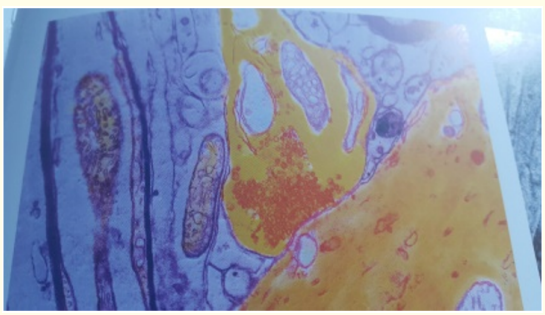


Figure 1

Objective with the presentation of this clinical case

The objective of this presentation is to demonstrate how isolated fiber EMG and repetitive stimulation are a very reliable test with a high diagnostic sensitivity for neuromuscular transmitted diseases such as:

- Myasthenia gravis.
- Botulism.
- Eaton Lambert syndrome.
- Myastheniform syndromes.

Clinical Case: Presentation

Reason for admission: 58-year-old male patient with several visits to the emergency room in the last month due to difficulty swallowing and chewing.

Clinic: Ptosis, weakness and dysphagia.

Background: Allergy to penicillin, ibuprofen and tramadol. HT in treatment. No DM or DLP. No toxic habits. No known heart disease. Extrinsic bronchial asthma in treatment with bronchodilators and allergic conjunctivitis in treatment with antihistamines. Right hemithyroidectomy, postsurgical hypothyroidism under replacement treatment. Poliomyelitis in childhood with sequelae of atrophy in MID. Fracture of the greater trochanter of the femur in 2016, not operated on. On follow-up by the pain unit for lumbar spondyloarthritis and canal stenosis, under treatment with TENS and periodic infiltrations. Adjustment disorder with depressed mood. Pathological grief. Parasuicidal gesture in July 2020.

Baseline situation: Wanders with unilateral support due to polio sequelae in MID, no dyspnea or orthopnea. chronic treatment: Euthyrox 112 (M-F) and 125 (S AND D), Adalat retard 20, Ebastel Forte Flas 20, Sertraline 100, Orfidal 1 mg if insomnia, Omeprazole 20, Paracetamol/Zaldiar if pain, Ventolin, Fostair.

Current disease: 58-year-old patient with several visits to the emergency room in the last month due to difficulty swallowing and chewing. In addition, she reports changes in the tone of the voice, drooping of both eyelids and difficulty in ocular mobility without appreciating diplopia, all symptoms of the same evolution (approximately 1 month). She has presented several episodes of choking, both with solids and liquids, for which she has decreased her intake, with unquantified weight loss. She does not report loss of appetite. She is using her hands to help close her mouth during the intake. In addition, she refers to noticing that the last month is exhausted earlier when walking. She occasional dyspnea on exertion. She denies breakthrough symptoms or recent changes in medication.

Physical exploration

NH and NC, afebrile, normotensive, eupneic, $SatO_2$ (room air) 98%, normal ACP.

NRL: Adequate level of alertness, oriented, normal language without dysphasic elements, other apparently preserved superior functions. Myasthenic facies, with bilateral palpebral ptosis, activation of the frontal ms and bilateral inferior facial paresis. He speaks dysarthric, with change to bitonal at the minute of starting the interview. Weakness in cervical extension and mandibular closure. No velopalatine paresis, gag reflex preserved.

MOE: limitation of bilateral abduction, more pronounced in RE, without diplopia (refers to the left amblyopic eye since childhood). It protrudes and mobilizes the tongue, not atrophy or fasciculations. No weakness or fatigability in MMSS.

MMII not assessable (polio sequelae, hip fracture). No sensory alterations or DN dysmetria. Paretic march (sequel).

Complementary examinations: Analytical: renal and hepatic biochemistry, ions, lipemic profile, iron metabolism, tsh, pth within normal values. Except: Serum calcium (corrected for proteins) * 10.9 mg/dL 8.6 - 10.0, Seric calcium (corrected for albumin) * 10.10 mg/dL 8.60 - 10.00, Hemogram: Red blood cells * 4.2 x10 ^ 6/uL 4.5 - 5.9, Hemoglobin * 12.3 g/dL 13.5 - 17.5, Hematocrit * 36.4% 41.0-53.0, Mean corpuscular volume 87.7 fL 80.0 - 100.0, Mean corpuscular hemoglobin 29.6 pg/cell 26.0 - 34.0. Hemoglobin concentration corp. mean 33.8 g/dL 31.0 - 36.0 Red cell distribution width (CV) 12.9% 11.5 - 14.5, Red cell distribution width (DS) 41.1 fL 38.0 - 52.0, white series and platelets within normality. Erythroblasts 0.00 x10 ^ 3/uL 0.00 - 0.03, Erythroblasts% 0.00/100WBC 0.00 - 0.05, Folate * 2.5 ng/ml

3.8 - 16.0, Severe deficiency: < 2.7 ng/mL Vitamin B12 379 pg/ml 200 - 770 vitamin D 10, severe deficit Ac. Anti-Nuclear (Hep-2) Negative Ac. Onco-Neuronal (serum) (IFI) PEND Anti: NMDA-R, AMPA-R 1y2, GABAR, LGl1, CASPR2. Negative Serum Ac. Anti-GAD PEND, Ac. Anti-acetylcholine receptor positive Ac. Anti-MUSK negative Ac. Anti-Gangliosides IgG and IgM PEND Ac. Anti-Striated Muscle/Myocardium PEND Ac. Anti-Proteinase 3 (cANCA) 0 U/ml 0 - 20 Ac. Anti-Myeloperoxidase (p-ANCA) 0.2 U/ml 0 - 20 Ac. Anti-Cardiolipins (IgG) 0 U-GPL/ml 0 - 20 Negative Ac. anti-Cardiolipins (IgM) 0 U-MPL/ml 0 - 10 Ac. Anti-Beta 2 - GPI (IgG) 0 GPL-U/ml 0 - 20 Negative Ab anti Beta 2 - MPI (IgM) 0.1 MPL-U/mL 0 - 10 Ac. Anti-Thyroperoxidase * 55.8 IU/ml 0.0 - 25.0 Positive serologic lues, Borrelia brucella negative.

Chest X-ray: Prominent aorta, without signs of condensation or effusion.

TAC: TORAX: No suspicious pulmonary opacities of malignancy are identified. Tracts pleuroparenchymal in both lung bases. Mediastinal structures without evidence of focal lesions and without relevant findings.

No pleural or pericardial effusion. There are no axillary, supraclavicular, mediastinal, or hilar lymphadenopathy.

Lower abdominal cuts included in the study without relevant findings. Degenerative changes in the axial skeleton.

Diagnostic impression: Study without signs, findings suggestive of thymoma.

Cerebral and cervical MRI 01/13/2021: Multiple small foci of altered signal intensity are observed in the subcortical white matter, in the frontal and parietal lobes, bilaterally, some of them confluent, of probable small vessel vascular origin. Single focus of microbleeding in the left parietal lobe. No other deposits of hemosiderin are observed. Ganglia of the base, talamos and normal internal capsules. Posterior fossa without significant alterations. In the diffusion study, no foci of alteration to diffusion. Ventricular system, cerebral sulci and cistern spaces of normal size. Optic nerves and optic chiasm of normal morphology and signal intensity. Symmetric extrinsic muscles without alterations. Lacrimal glands without alterations. Slight mucous thickening in the nasal sinuses without air-fluid levels suggesting current infection.

Conclusion: Moderate leukopathy (Fazekas grade 2) of probable small vessel vascular origin. Rest of the brain and orbital exploration without significant alterations. Cervical MRI: Cranio-vertebral junction with normal characteristics. Vertebral bodies of normal morphology, alienation and signal intensity. Medullary canal and foramina of normal caliber, without appreciating significant medullary or root compression. Clear signal alterations in the spinal cord are not identified, although the assessment is very limited by movement artifacts.

Conclusion: Cervical spine examination without objective alterations.

Book description EMG. Myasthenia gravis protocol

It has been explored: Conventional EMG of the orbicularis oculi muscles left. side affected by ptosis. ENG motor of N. left motor facial nerve. side most affected by ptosis. Left motor facial nerve ER. and right motor median nerve. JITTER DE M. Orbicularis muscles of the left eyes.

Findings

Proximal repetitive stimulation at low frequencies (3Hz, 5Hz and 10Hz) in the left and right nasalis muscle (left and right facial) at rest and after tetanization, SI shows decremental responses, the higher the frequency of the stimulus. Decremental responses from 23 to 35%. greater the decrease in motor potentials, characteristic of ocular MG. There are no facilitating phenomena that increase the motor potential characteristic of SEL. Decremental responses are observed above all after 1-3 min post exercise. Decremental responses from 23 to 35%.

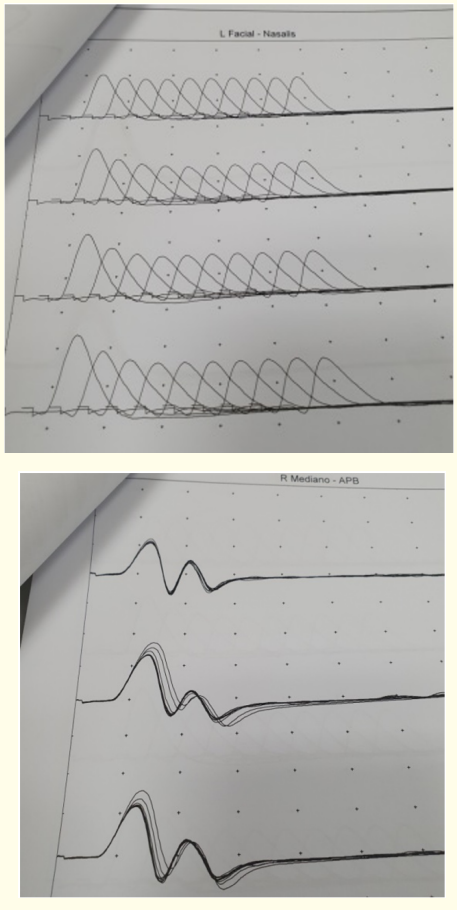
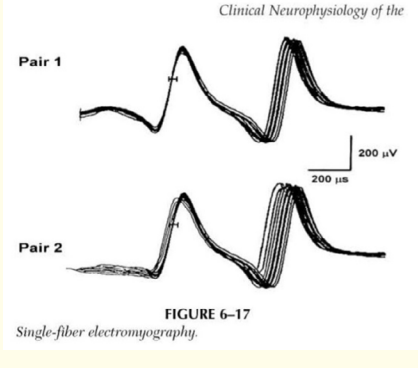
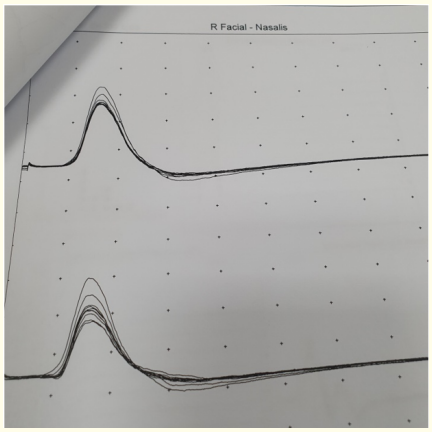
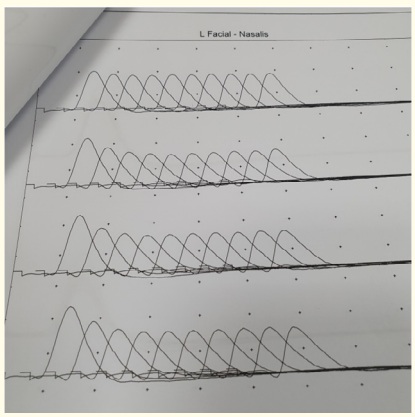


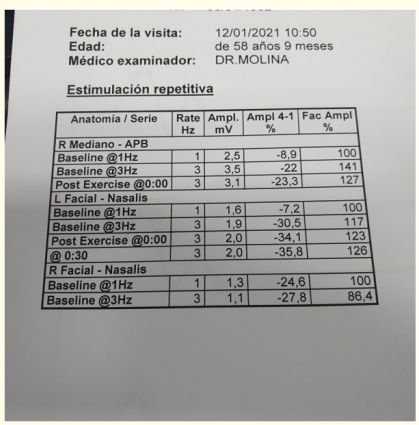
Figure 2

Distal repetitive stimulation at low frequency (3Hz, 5Hz and 10Hz) in the abductor digiti minimi (right ulnar) muscle at rest and after tetanization also shows decremental responses in motor potentials, from 23 to 35%. Characteristic in the generalized MG. There are no facilitation phenomena that increase motor potential characteristics of SEL. Decremental responses are observed above all after 1 - 3 min post exercise.









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Edad: de 58 años 9 meses
Médico examinador: DR.MOLINA

Estimulación repetitiva

Anatomía / Serie	Rate Hz	Ampl. mV	Ampl 4-1 %	Fac Ampl %
R Mediano - APB				
Baseline @1Hz	1	2,5	-8,9	100
Baseline @3Hz	3	3,5	-22	141
Post Exercise @0:00	3	3,1	-23,3	127
L Facial - Nasalis				
Baseline @1Hz	1	1,6	-7,2	100
Baseline @3Hz	3	1,9	-30,5	117
Post Exercise @0:00	3	2,0	-34,1	123
@ 0:30	3	2,0	-35,8	126
R Facial - Nasalis				
Baseline @1Hz	1	1,3	-24,6	100
Baseline @3Hz	3	1,1	-27,8	86,4

Figure 3

Jitter with proximal axonal microstimulation, without voluntary activation of the muscle by the patient, performed with a monopolar needle at the level of the orbicularis oculi muscle on the right side, most affected by bilateral ptosis. SI shows significant variations at the level of the interpotential intervals, in 25 pairs studied. If end plate blockages are observed.

Current data to define today disease of the neuromuscular junction of proximal location proximal and distal pathological ER. distal ER also pathological. Study of jitter with axonal microstimulation, without voluntary activation by the patient, at the m level. orbicularis of the right eyes. Pathological which points in favor of the possibility of a Myasthenia Gravis with proximal and distal involvement.

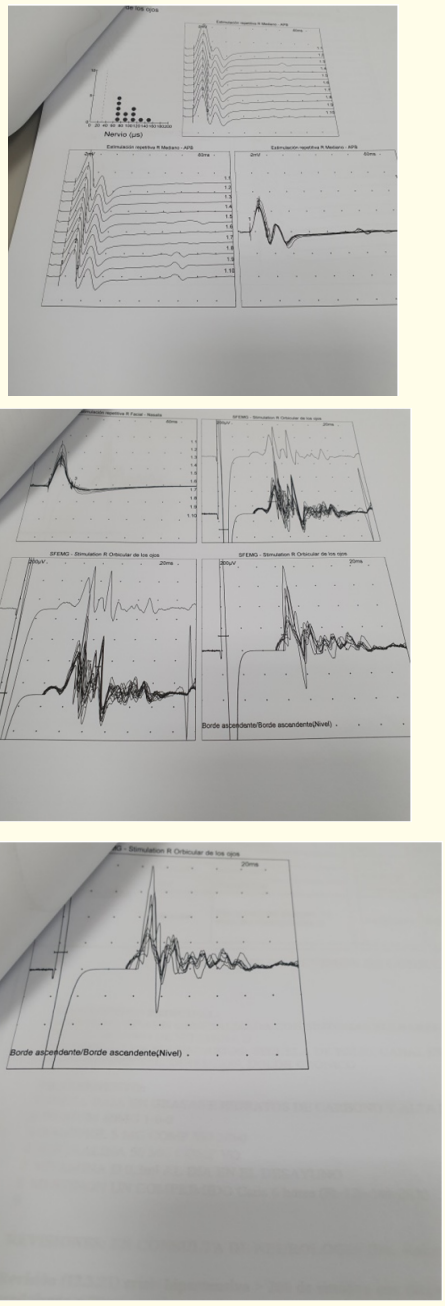

Figure 4

Discussion

Myasthenia gravis (MG) is a postsynaptic neuromuscular disorder, autoimmune attack by anti-Ach receptor antibodies (acetylcholine), selective attack against acetylcholine receptors of the postsynaptic terminal, weakness worsens with exercise and improves with cold, hence the importance of the ice test to verify if the eyelid ptosis improves somewhat in this type of patient. Lowering the temperature with the ice test reduces cholinesterase activity and increases the amplitude of depolarization produced by a single acetylcholine molecule.

It is recommended that the temperature of the skin to do the test is about 35 degrees, and the anticholinesterase drugs should be withdrawn at least 12 hours before the test.

In MG there is usually an elevation of the antibody titer against acetylcholine in 80-90% of cases. In these cases, Ig G anti-nicotinic acetylcholine receptor antibodies are detected in the postsynaptic membrane of the neuromuscular junction [4,7] as a consequence of the autoimmune blockade-attack of these Ach receptors, which translates into a difficulty in the depolarization of the membrane although acetylcholine is released normally, but receptors are reduced, due to autoimmune attack by antirecpetor antibodies.

If the affectation is predominantly ocular, the antibodies appear in 50% of the patients, therefore the negativity to the antibody test against Ach receptors does not exclude the diagnosis.

In some cases, these antibodies have their origin in immunological cross reactions with protein domains that share the Ach receptor with proteins of viruses as common as herpes simplex [8]. There is also the possibility of drug induction (D-penicillamine, procaine and aminoglycosides) in the generation of a myastheniform syndrome.

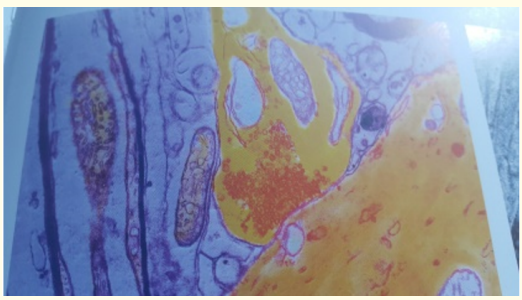

Figure 5

Histologically, what occurs is a decrease in the number of Acetylcholine receptors in the postsynaptic terminal and a flattening of the folds in the postsynaptic membrane. There is evidence that the thymus plays a role in the origin of the synthesis of these antibodies [4,7,9]. More than 70% of these patients have thymic hyperplasia or a thymoma in 10 - 15% of patients, the autoimmune reaction begins in the thymus [10] reactive T cells migrate in very early stages to other locations [8] when the disease begins in those over 50 years of age, there is the immunological peculiarity of the presence of anti-striated muscle antibodies in 50% of cases. This autoantibody is considered a marker for the existence or not of an associated thymoma [10,11].

10% of MG cases are ocular. On rare occasions the weakness affects the respiratory muscles, with a serious prognosis (myasthenic crisis). MG can present as a paraneoplastic syndrome (more frequently associated with adenocarcinoma of the lung) and that sometimes precedes the diagnosis of the tumor by years [12,13].

Another very interesting clinical test that gives more clues to the explorer is the Simpson test: increase in ptosis after looking up for 2 min. Repetitive exercise worsens the clinic. The ice test improves it [5].

It usually affects the cranial nerves. MG can mimic any pattern of supra or infranuclear oculomotor palsy. It usually presents with weak facial mimicry, ptosis, diplopia, fatigue, in our patient he referred us on many occasions that he had serious difficulties in closing his mouth. Weakness of speech, chewing, swallowing, weakness that can reach the respiratory muscles, as well as the proximal muscles of the extremities.

Sometimes in the EMG it is possible to verify the decreases in the repetitive stimulation of the spinal accessory nerve, picking up the trapezius muscle, in addition to the typical facial protocol. The initial involvement of MG is usually the facial and proximal limb muscles. It usually occurs with a nasal voice, difficulty keeping the neck upright, difficulty opening and closing the mouth, fluctuating dysphonia, swallowing disorders. There may be a strabismus in which the relationship between the ocular axes varies in the different gaze directions (not concomitant) [3,5].

For the diagnosis there are several diagnostic techniques:

- Proximal and distal repetitive stimulation (median, ulnar, facial, and spinal accessory nerve).
- Isolated fiber EMG. Jitter.
- Other studies: ENG- EMG.

In repetitive stimulation (RE) it is the most specific neurophysiological study in the diagnosis of disorders of the neuromuscular junction [5]. The study must be done with a temperature greater than 30 - 35 degrees Celsius, with a supramaximal stimulus (which activates the greatest number of muscle fibers), making sure of the stable amplitude of the CMAP). The cold improves the plaque and the weakness masking the process, for this reason it is important that the extremity to be explored is at 30 - 35 degrees centigrade º.

In presynaptic disorders (there is a significant increase in the amplitude of the CMAP, brief exercise can recover the potentials diminished by slow stimulation, in Postsynaptic disorders, after 2 - 4 minutes of the sustained contraction (the reserves of Ach), the decrease is more clearly evident compared to the pre-exercise values [10].

The amplitude of the CMAP indicates the number of muscle fibers activated by that stimulus. When the action potential reaches the presynaptic nerve terminal, 20% of the Ach (acetylcholine) stored in the terminal or presynaptic button is released, the stimulus trains at a frequency of 2, 3, 5 Hz, (low frequency) produce that the immediate release Ach is consumed, therefore there is a decrease in the vesicles released in the first nervous stimuli, these causes it to decrease in a physiological way, a little (less than 5% of the amplitude of the CMAP) in the motor plate, although In our experience, CMAP in healthy people remains very stable and this stability can be seen by averaging the signal [3,5]. With the activation of stored Ach, which occurs between the 4^{th} and 5^{th} potential, sde stabilizes the amount of Ach released and also the potential of the motor plate. Being the decreases in a physiological way always 10% of the 1^{st} potential. This occurs in normal patients free of neuromuscular junction disease. A decrease in the amplitude of the 4^{th} - 5^{th} potential greater than 10% is considered pathological [1,3,5].

In the ER (Repetitive nerve stimulation) (3 Hz) in presynaptic diseases it will be observed: decreased CMAP. The low frequency ER decreases the 4 - 5 potentilla and the high frequency ER produces a significant increase in amplitude (facilitation) [3,5].

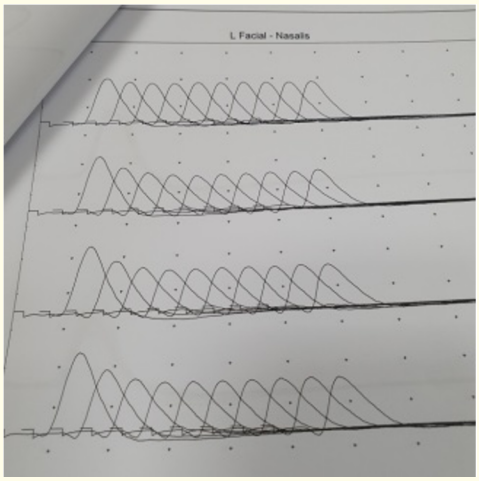

Figure 6

Electromyography shows a decrease of more than 10% in the 4 - 5th potential with respect to the first at low frequency 3 hz, giving, as we can see in the figure of our patient, the characteristic U shape or inverted horseshoe, due to the electrodecrease of the potential. The same is true for Eaton Lambert Syndrome (SEL) and for Botulism. But in congenital myasthenia it is not usually present.

Jitter is the selective recording of the action potential of an isolated muscle fiber in a radius of 300 micrometers. It provides detailed information on the physiology and stability of the neuromuscular plate, any muscle can be explored, the frontal muscles, orbicularis oculi, common extensor of the fingers, and dorsal interosseous are of choice. It is performed through mild and sustained voluntary muscle activation (more difficult to obtain) or medium microstimulation at a stable low frequency (easier to obtain) and more indicated in children, psychiatric patients, the elderly and in people who do not cooperate, the jitter (EMGFU) allows us to analyze the behavior and stability of 1 or 2 or more muscle fibers belonging to the same motor unit. For the study of jitter, an insulated fiber needle is used, which has inside an electrode with a very small recording surface (25 microns) less than the average diameter of the muscle fibers, the recording area of the electrode is 270 micrometers, and this electrode is 3 mm from the tip of the needle.

Also, in many laboratories a concentric needle can be used and although it has a larger recording surface, properly adjusting the filters, the values of both voluntary and stimulated jitter are perfectly obtained with experience, showing different MCD values being EMGFU voluntary or stimulated, the Stimulated MCD values are lower than Voluntary Jitter. Jitter in short is the analysis or study of the variability of the activation of two muscle fibers belonging to the same motor unit, said variability is minimal and almost stable, when there is a disorder of the neuromuscular junction (NM), said variability and stability are altered, giving values of dysfunction and instability (blockages and increase of said variability, increases in jitter), by supposing the averaging of response, the stability or instability of the complex is perfectly registered.

Objective with the presentation of this clinical case

The objective of this presentation is to demonstrate how isolated fiber EMG and repetitive stimulation are a very reliable test with a high diagnostic sensitivity for neuromuscular transmitted diseases such as:

- Myasthenia gravis.

- Botulism.
- Eaton Lambert syndrome.
- Myastheniform syndromes.

The protocol to be followed is presented, valid by the Spanish Society of Clinical Neurophysiology, and a brief explanation of the etiopathogenesis and diagnostic techniques that help improve the diagnostic process in the event of a neuromuscular plaque disease. With particular emphasis on the Jitter as the test - that well performed and with the appropriate filters and following the protocols, together with repetitive stimulation at low frequencies, allows a high diagnostic sensitivity.

If there is MG or disease of the NM plate, the NM disorder is usually increased and with blockages, which is what shows us the instability of the neuromuscular plate, a healthy plate is perfectly stable and all the potentials are superimposed, generating a stable graph. unlike the neuromuscular disorders type MG, SEL, and botulism where the clear instability of the jitter is demonstrated [3,5]. If the transmitting neuromuscular disorder is severe, and therefore with marked symptoms, the nerve impulse is insufficient to reach the post-synaptic membrane depolarization threshold and the muscle membrane action potential is not triggered and there is the so-called intermittent stimulus block, which is usually associated with MCD values greater than 100 msec.

It is necessary to study at least 20 pairs of fibers to obtain 20 individual values, it will be a pathological Jitter if the mean jitter exceeds the normal limit values and if more than 10% of pairs of fibers present an increased Jitter (2 of 20 pairs) and for assuming the presence of blockages in postsynaptic transmission.

In voluntary jitter the MCD varies from one muscle to another of 23 - 33 msec. For the common extensor muscle of the fingers [5,6] 2012 (Kouyoumdjian, Stalberg) [1,10].

For the orbicularis oculi muscle, the MCD of voluntary jitter ranges between 24 - 34 msec.

For the stimulated jitter the values are somewhat lower, for the common extensor of the fingers between 18 msec and 22.6 and for the orbicularis oculi or frontalis (21 - 25 msec) and 16 - 24 respectively [5,6,16] 2012 (Kouyoumdjian, Stalberg).

Normally, in hospitals, the edrophonium (anticholinesterase) test is performed for Myasthenia gravis, which improves the patient's symptoms, specifically muscle weakness, if it is negative, it does not rule out MG. And the detection of anti-Ach receptor antibodies is also carried out: MG markers, which are usually positive in 80% of cases. (6,16) and of course the neurophysiological tests previously exposed.

EMGFU is the ideal diagnostic method, especially in ocular forms, ER is very unprofitable in non-affected muscular territories. Increased JITTER has a higher diagnostic performance than ER. There is a significant correlation between the weakness of the patient, the symptoms (ptosis, diplopia, fatigability) and the increase in jitter values and the presence of blockages. Fiber density in MG is usually normal [1,5,6,10].

The impulse conduction blocks, described above, are more frequent when the jitter is greater than 100 microseconds. And they are indicative of marked degrees of weakness.

Sensory neurography of a patient with myasthenia usually shows normal values. Motor neurography is also usually within normality, rarely in patients with great weakness we can find slightly decreased CMAP amplitudes (many muscle fibers do not contract with the stimulus). The EMG is usually normal, although fibrillations have sometimes been described (they are very rare) and some myopathic

PUM (short duration, increased polyphasia, unstable, is produced by the blockage and physiological slowing of neuromuscular transmission during Voluntary activation [1,3,5].

Conclusion

The findings are consistent with:

- Current data to define today disease of the neuromuscular junction of proximal and distal location, proximal ER and pathological distal. ER distal response decrease.
- The jitter with proximal axonal microstimulation, without voluntary activation of the muscle by the patient, performed with a monopolar needle at the level of the orbicularis oculi muscle of the right side, most affected by bilateral ptosis. SI shows significant variations at the level of the interpotential intervals, in 25 pairs studied. If end plate blockages are observed.
- Current data to define today disease of the neuromuscular junction of proximal location proximal and distal pathological ER. distal ER also pathological. Study of jitter with axonal microstimulation, without voluntary activation by the patient, at the m level. orbicularis of the right eyes. Pathological which points in favor of the possibility of a Myasthenia Gravis with proximal and distal involvement.

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