A Dual Diagnosis of Myasthenia Gravis and Sjogren's Syndrome in a Hispanic Patient: A Case-based Discussion of Clinical Findings, Therapeutic Implications, and Culturally Competent Care

Annelise Velasco de Azevedo-Pereira; Maria Paula Montes-Lozano; Nicolle Suarez-Otero; Christopher Owens, Alfredo Lopez-Yunez

Corresponding Author

Annelise Velasco de Azevedo Pereira
avap96@gmail.com

ABSTRACT

The co-occurrence of myasthenia gravis (MG) and Sjogren's syndrome (SS) in a young, Hispanic, female patient underscores the diagnostic and therapeutic complexities inherent in autoimmune disorders. MG, a rare neuromuscular disorder characterized by autoantibodies impairing neuromuscular transmission, leads to variable muscle weakness and fatigue, notably affecting ocular and bulbar muscles. SS, an autoimmune condition with systemic manifestations, primarily affects secretory glands, resulting in symptoms such as xerostomia and xerophthalmia. Both conditions have higher prevalence rates in females, with MG particularly affecting young women under 40. Herein we present a case report and case-based discussion on diagnostic and management challenges of these two autoimmune conditions while highlighting issues specific to the Hispanic and underrepresented populations. Our goal is to highlight the intricate interplay between these two autoimmune pathologies to allow for more rapid recognition and reduce delay in diagnosis.

Keywords: *Muscle weakness, Myasthenia gravis (MG), Autoimmune Disorders, Sjogren's Syndrome (SS), Autoantibodies, Diagnostic challenges*

1. Introduction

1.1 Muscle weakness in the primary care clinic:

True muscle weakness, in contradistinction to fatigue or pain or joint-related motor impairment, is a common presenting complaint to the primary care physician. While the differential diagnoses are broad and include electrolyte deficiency, endocrine, rheumatologic, genetic, medication or toxin-related, infectious, and neurodegenerative disorders,[1] a careful history noting the distribution of the weakness and physical examination provide diagnostic clues. For example, bulbar weakness, defined as oral and pharyngeal muscle weakness and characterized symptomatically by dysphagia, dysarthria, and dysfunctional mastication,[2] may be caused by neurologic etiologies like myasthenia gravis (MG), amyotrophic lateral sclerosis (ALS), syphilis (bulbar variant), and also endocrine pathologies like hyperthyroidism.[1] Herein we aim to provide an overview of two relatively rare causes of bulbar muscle weakness co-presenting in a single patient along with their clinical manifestations, diagnostic criteria, and treatment rationale. Our rationale for evaluating them together in the context of this case study is to increase clinical awareness and improve diagnostic efficiency as delayed diagnosis of one or both of these has clinical and psychological import.

1.2 Myasthenia gravis: epidemiology and classification:

Myasthenia gravis (MG) is a complex autoimmune neuromuscular disorder characterized by the production of autoantibodies directed against the acetylcholine receptor (AChR) or other proteins critical for neuromuscular signaling. This disease affects the neuromuscular junction, leading to profound muscle weakness and fatigue, especially in the ocular, bulbar, and proximal muscles of the extremities.[3] Its incidence per year is estimated to be between 8 to 10 cases per million, with an approximate prevalence of 150 to 200 cases per million in the United States.[4] According to the National Institute of Neurological Disorders and Stroke this condition is more likely to affect young women under 40 years of age and men over 60 years of age.[5]

In Central and South America, prevalence figures are much lower than those mentioned above, generally less than 100 cases per 100,000 habitants. Given that figures from the Iberian Peninsula are generally consistent with other reported prevalence rates, the low rates in Central and South America are likely from underreporting[6]. Regardless of where the statistics are derived from, epidemiological data consistently show a higher incidence in women, with a peak between 20 and 40 years and another between 60 and 80 years, while in men it predominates at advanced ages with a sustained increase from 60 years.[6]

The diagnostic nomenclature of myasthenia gravis is complicated and can be intimidating to the most seasoned clinician. Given that it is based on clinical, epidemiological, immunological, and genetic findings, as well as thymus pathology, it can quickly become confusing by its overlap and redundancy. We suggest first sub-classifying myasthenia gravis (MG) as to antibodies against acetylcholine receptors (AChR-MG) which are found in approximately 80% of MG patients. Within this sero-positive-AChR-MG category, further classification is based on both the location of the affected muscles and the timing of onset; pure ocular MG (OMG), early-onset MG (EOMG, onset <45 years), and late-onset MG (LOMG, onset >45 years).[3,7]

EOMG typically presents with lymph follicular hyperplasia of the thymus. In contrast, LOMG is characterized by an age-dependent involution of the thymus.[7] Additionally, 10–15% of all patients within this AChR-MG classification are affected by thymoma.[7] When MG occurs together with a thymoma, MG is considered a paraneoplastic syndrome. Though the presence of antibodies to the AChR is the main cause of weakness in these patients, they often have other antibodies to the striated muscle protein titin or to a calcium channel in the sarcoplasmic reticulum known as RyR.[8]

In the OMG subtype most of the patients with ocular symptoms at onset will progress to generalized forms of the disease, usually within two years of onset,[3] with 90% of the remaining continue to have ocular manifestations only.[3] Hence, ocular MG is defined by isolated extra-ocular involvement for a period of ≥2 years. Over half of the patients in this group have antibodies against AChRs.

In addition to anticholinergic antibodies, other subtypes include antibodies to a muscle specific tyrosine kinase (Anti-MuSK-Ab-associated MG) (MAMG). MAMG is found in approximately 7–10% of all MG patients and up to 40% of patients with generalized MG who are seronegative for anticholinergic receptor antibodies (AchR Abs). This variant often has an acute onset affecting the facial and bulbar muscles. There is an 85% female predominance.[3] More rarely antibodies directed against the low-density lipoprotein receptor -related protein 4 (Lrp4 Antibody-Associated MG) (Lrp4-MG), are present in 2–50% of the so-called double seronegative MG cases.[3,14] "Seronegative" MG (SNMG) is a heterogeneous group of patients who share negative results for AChR and MuSK antibody testing. It is likely that this subgroup of patients has some other yet undetected antibodies.[7]

Clinical classification of MG is based on the Myasthenia Gravis Foundation of America's Clinical Classification[9]: Class I: Any eye muscle weakness, possible ptosis, no other evidence of muscle weakness elsewhere. Class II: Eye muscle weakness of any severity, mild weakness of other muscles • Class IIa: Predominantly limb or axial muscles •

Class IIb: Predominantly bulbar and/or respiratory muscles Class III: Eye muscle weakness of any severity, moderate weakness of other muscles • Class IIIa: Predominantly limb or axial muscles • Class IIIb: Predominantly bulbar and/or respiratory muscles Class IV: Eye muscle weakness of any severity, severe weakness of other muscles • Class IVa: Predominantly limb or axial muscles • Class IVb: Predominantly bulbar and/or respiratory muscles (Can also include feeding tube without intubation) Class V: Intubation needed to maintain airway.[9]

These classification systems, though unwieldy, offer therapeutic and prognostic clues like the likelihood of response to steroids, probability of remission, relapse, exacerbation, generalization in the first six months and appearance of other autoimmune diseases.[10]

1.3 Sjogren's Syndrome:

Sjögren's syndrome (SS) is a chronic, autoimmune and multisystem disorder that affects the secretory glands, in particular, the lacrimal and salivary glands, producing xerophthalmia and xerostomia, respectively, which can lead to symptoms like dysphagia, difficulty articulating, dysphonia and flaccid dysarthria.[11] Additionally patients can present with a variety of extra glandular (systemic) manifestations such as arthralgia or arthritis, Raynaud's phenomenon, lymphadenopathy, lung disease, vasculitis, kidney disease, lymphomas, splenomegaly, peripheral neuropathy and myositis. This wide variety of signs and symptoms are due to autoimmune lesions of multiple organic systems such as the central nervous, vascular, joint, muscular, skin, lung, and kidney systems.[11]

Global incidence rates range between 3 and 11 cases per 100,000 patients, while the prevalence is around 0.01-0.72%.12 There is a 10:1 (women: men) prevalence ratio. Severe ocular involvement predominates in men, while systemic manifestations are much greater in women.[12]

Anti-SSA/Ro (anti-Sjogren's syndrome related antigen A autoantibodies) and Anti-SSB/La (anti Sjogren's syndrome type B) antibodies are distinctive markers in primary Sjögren's syndrome (pSS), found in 40–80% of patients.[12] Individuals with these antibodies, particularly anti-Ro/La, exhibit the highest prevalence of various systemic, hematologic, and immunologic alterations. Immunological studies commonly detect antinuclear antibodies (ANA), with anti-Ro/SSA being the most specific marker. Notably, anti-SSA/Ro and anti-SSB/La autoantibodies may be present in the serum long before the clinical manifestation of Sjögren's syndrome.

Genetic susceptibility to SS is linked to HLA class II markers. The association with HLA-DRB1*03 suggests that HLA alleles predispose individuals to autoantibody secretion, though without a clear connection to clinical outcomes. HLA-DR15 promotes the production of anti-SSA, while HLA-DR3 is linked to both anti-SSA and anti-SSB production. In a cohort of 400 SS patients, Garcia-Carrasco et al. demonstrated that antinuclear antibodies, anti-Ro/SSA antibodies, rheumatoid factor (RF), and anti-La/SSB antibodies are the most prevalent immunologic patterns.[13]

Both SS and MG are illustrative examples of diseases of dysfunctional self-recognition often producing multiple and heterogeneous autoantibodies against both nuclear and peripheral targets.

2. Case Report

extremities both proximally and distally. She was able to sustain a posture in the upper and lower extremities for 60 seconds indicating no fatigability. Fine motor movements were normal bilaterally. Sensation was intact to light touch, pinprick, vibration, and proprioception throughout. Reflexes were symmetrical in all muscles bilaterally. No dysmetria was noted on finger-nose-finger or heel-knee-shin. She had normal rapid alternating movements and fast finger tapping with normal amplitude and speed was able to be demonstrated.

Her single-count breath test was 26, which was normal.[14-1]*[1] Repetitive stimulation of the right abductor pollicis brevis was performed and found normal. Trains of six square wave pulses were applied through bipolar stimulation electrodes at a 3Hz frequency. No decrement in amplitude was observed in the compound motor action potentials, which is a normal result.[1]

Differential diagnoses were carefully considered. A normal creatine kinase level made muscular dystrophies less likely ruled out myopathiesA 26-year-old Hispanic female previously healthy and independent in her activities of daily living (ADL), with no past medical history or known family medical history, presented to the neurology department with the chief complaint of 6 months of gradual loss of voice, nasal speech, difficulty swallowing both solids and liquids and blurred vision. She denied aspiration events but admitted to occasionally choking on food. She denied weight loss and other constitutional symptoms like fever or night sweats. The patient reported that the symptoms started suddenly and gradually worsened over time. She denied any precipitating triggers like recent infection, surgery, or stress. She also denied known exposures to organophosphates or other chemicals, had not taken any recent medications, and no exposure to tick bites.

Upon admission, the physical examination revealed the patient to be a well-dressed, engaging woman, alert and oriented to person, place, and time. Her vital signs were all normal and she was breathing comfortably at 16 breaths per minute. Initially in the interview, it was noted that her speech was fluent, but she had mild difficulty articulating certain words and had difficulty controlling the pitch of her voice. She demonstrated a nasal quality to her speech. Over the course of the interview, her speech became more fatigued with an increased breath and nasal quality. The hoarseness of her voice was evident when asked to make a high pitched "eeee" sound. No anatomic abnormalities in the pharynx or larynx were visible. She was able to sustain a lateral eye gaze for 60 seconds and an upper eye gaze for 60 seconds without demonstrating fatigable diplopia or ptosis. Eyelid closure strength was normal. The visual acuity exam and fundoscopic evaluation were normal. Pupils were equal, round, and reactive and extraocular movements were intact.

Muscles of facial expression were symmetrically intact with normal sensation throughout the three branches of the trigeminal nerve, movements of the tongue were present without fasciculations, no lip trembling or drooling found positive during examination, gag reflex was present and normal, bedside swallowing test was normal with no choking present, there was no fatigability on the temporalis and masseter muscles. Hearing was intact bilaterally. Her neck flexor strength was normal indicating minimal risk for respiratory symptoms.

Peripheral muscle bulk and tone were normal, and strength was 5/5 in all four. The absence of weight loss and other systemic symptoms suggested the unlikely presence of an underlying neoplasm, while no history of skin rash eliminated dermatomyositis and lupus from consideration. She also denied any recent viral infections and episodes of diarrhea. Space-occupying lesions in the brainstem and Chiari malformation were ruled out by MRI and CT scans. Similarly, demyelinating diseases such as multiple sclerosis were excluded. A normal electromyography ruled out amyotrophic lateral sclerosis.

Furthermore, leptomeningeal carcinomatosis or lymphomatosis were also considered unlikely, due to the absence of pain and other related symptoms, and a normal cerebrospinal fluid analysis.

[1] The single-count breath test (SCBT) is a bedside and inexpensive test in which the examiner asks the patient to take a deep breath and to count starting with the number one at a pace of approximately 2 numbers per second. The score is the last number counted before the patient had to take a second breath. It has shown moderate correlation with the forced vital capacity measured in spirometry. A score of 25 is considered normal but the ideal is to have a baseline score specific to each patient and measure it at every encounter.

Pertinent findings among the laboratory tests, (Table 1), included a negative test for Lyme's disease antibodies. Systemic connective tissue diseases were investigated, with an antinuclear antibody (ANA) test at a 1:320 dilution and Sjogren's SS-A/Ro were positive. Anti-SSB/La, rheumatoid factor, and anti-CCP were all negative. Antibodies to the acetylcholine receptor and blocking antibodies were both positive, confirming a diagnosis of Myasthenia Gravis. A computed tomography scan of the chest revealed normal thymus structures without evidence of thymoma or thymus hyperplasia.

In summary, we present a 26-year-old Hispanic woman with progressive bulbar symptoms exhibited by mild dysphagia, fatigable dysphonia and flaccid dysarthria with positive antibodies to the AChR in the setting of ANA and SSA/Ro positivity. A diagnosis of early onset myasthenia gravis and Sjogren's syndrome was made.

Hydroxychloroquine was initially started at 200mg when her antibodies for Sjogren's were reported as positive. However, within a few days it was discontinued after the acetylcholine receptor antibodies were reported, because of the potential impairment of neuromuscular conduction and muscle toxicity that may occur with hydroxychloroquine.15 Prednisone 40 mg daily was also prescribed until improvement was observed. For symptomatic treatment of her bulbar symptoms, the cholinesterase pyridostigmine bromide was initiated at a dose of 60 mg, two to three times a day.

Six weeks after initiation of treatment, the patient reported significant improvement in phonation and in nasal speech. However, a recess from steroids was attempted but aborted due to the return of symptoms and fatigue. As a result, prednisone was restarted at 60 mg daily, and mycophenolate was initiated at a dose of 500 mg, twice a day. She remains minimally symptomatic and much improved.

3. Discussion

Herein we report a case of a young Hispanic female patient with no previous medical history or family history of autoimmune diseases who co-presented with Type IIb generalized ACR Ab positive MG and SS.

This case illustrates the most common subtype of MG presenting in a young female patient with acetylcholine receptor binding and blocking antibodies. She is thus further categorized as generalized AChR Ab positive MG EOMG. This represents approximately 85% of MG patients. There is no apparent correlation between the level of antibodies and the severity of the disease.[17] While it is known that patients in this category have a higher incidence of thymic hyperplasia it was not detected in this case. Among the published literature, some articles says iIt is estimated that 50% of patients with a thymoma will eventually develop positive AChR antibodies without clinical manifestations, while approximately 30% will develop clinical MG,. while others says thymoma occurs in 10% to 12% of patients with MG, and 15% of thymoma patients have MG. Conversely, 10 to 20% of patients with MG either have at the time of diagnosis or will develop thymomas.[3] It is for these reasons that it is recommended that the thymus undergo surveillance every five years with imaging.[15]

As was evident in this case, the diagnosis of MG is mostly clinical with laboratory and imaging being confirmatory. The anti-AChR Ab test is very specific, and it confirms the diagnosis in patients with classical clinical findings.

Electrophysiologic tests are relevant in patients who are seronegative for antibody testing. Commonly employed tests for MG are the repetitive nerve stimulation test and single-fiber electromyography.[16] Both tests assess for conduction delays in the neuromuscular junction. Routine nerve conduction studies are usually performed to determine the functioning of the nerves and muscles before undertaking these tests.[16] Although these tests are relevant, they have limited sensitivity and specificity, especially in the cases where symptoms are limited to bulbar involvement, as they were in our patient's case.

Particularly relevant for ocular MG, the Edrophonium (Tensilon) Test is indicated. Edrophonium is a short-acting acetylcholinesterase inhibitor that increases the availability of ACh in the NMJ. Electrophysiologic testing is not applicable for ocular MG, so edrophonium is administered intravenously, and the patient is observed for improvement in symptoms of ptosis or diplopia. It has a sensitivity of 71% to 95% for MG diagnosis.[16]

Myasthenia gravis commonly coexists with other autoimmune disorders. Patients with MG may have inherited genes that make them more likely to develop other autoimmune conditions.[18] Worldwide, about 5 out of every 100 people have one or more autoimmune disorders while in patients with MG, 13 to 22 out of every 100 people have a second autoimmune condition indicating an underlying systemic immune disorder.[18] Autoimmune conditions like lupus, multiple sclerosis, and rheumatoid arthritis are much more common than MG. Out of 100,000 people with any autoimmune condition, only 35 will have MG.

In the case of the patient presented, who had generalized EOMG, other autoimmune pathologies include thyroiditis, lupus, type 1 diabetes, alopecia, giant cell myocarditis, myositis, red cell aplasia, autoimmune hepatitis, Sjogren's syndrome, Addison's disease, and Guillain–Barre syndrome must be considered and sought for.

Thyroiditis is the most common secondary autoimmune condition that people with MG have. Approximately 1 in 10 people with MG also have thyroiditis. Lupus occurs in between 1 and 8 out of 100 people with MG. Rheumatoid arthritis, dermatomyositis/polymyositis, and Addison's disease follow.[18, 19]

Treatment of MG must be individualized; however, the overall goal of treatment is to restore normal function and to minimize adverse effects of the disease. Treatment selection depends on many factors, including the distribution, duration, and severity of the disease. Among the most important factors to consider is patient preference. Guided shared decision making should consider the dosing schedule, route of administration e.g., infusion, costs, and insurance coverage.[28] The first-line medication used to treat MG patients is an inhibitor to acetylcholinesterase. Pyridostigmine is the most used acetylcholinesterase inhibitor, it works as a symptomatic therapy by increasing the amount of ACh in the synaptic cleft of neuromuscular junction, with a starting dose of 30 to 60 mg every 4-6 hours. Doses beyond 120 mg every 4 hours are not often effective and are more likely to cause cholinergic side effects, which include diarrhea, sweating, bradycardia, stomach cramps, and increased secretions. [17]

Immunotherapy is used to induce and maintain remission of symptoms, being considered the definitive form of treatment for MG. It aims to suppress the production of antibodies or the damage caused by them. Corticosteroids, such as prednisone, are a priority in treatment due to their high response rate, although their initiation may cause a temporary worsening of MG, so it is recommended to start with low doses and increase gradually.[29,30]

Retrospective studies have revealed a corticosteroid response rate between 70% and 80%, underscoring their importance and priority in the treatment of MG. Its use is recommended in combination with a corticosteroid-sparing agent. It is important to note that some patients may experience a temporary worsening of MG when high-dose corticosteroids are initiated. This worsening usually occurs between 4 and 10 days after the start of treatment and can sometimes trigger an attack.[29,30] It is recommended to closely monitor liver function, kidney function, blood count, and urinary biochemistry during treatment.[29,30]

Azathioprine and mycophenolate are considered the first-line immunosuppressive agent for MG, supported by evidence from randomized clinical trials and expert consensus. However, it is important to note that some patients treated with azathioprine may develop deficiency in the enzyme thiopurine methyltransferase (TPMT), which exposes them to an increased risk of adverse effects and may require the use of lower doses.[2] Rituximab, a monoclonal antibody directed

against B cells, is used in patients with MG refractory to other treatments or in severe cases where conventional treatments have failed. Intravenous immunoglobulins are also used to suppress the immune response and may be effective in reducing mechanical ventilation time during myasthenic crises. [30,31]

Therapeutic plasma exchange (TPR) is used for seronegative MG and anti-MuSK MG, although its repeated use is not recommended to achieve prolonged immunosuppression in MG. [29,30,31] As in many cases, our patient initially responded with steroids and anticholinesterase therapy, but symptoms returned soon after reducing the dose of steroids, reason why. Reason why the final therapy consisted of pyridostigmine, corticosteroids and mycophenolate.

Interestingly, the patient was diagnosed simultaneously with Sjogren's syndrome (SS), which resulted in changes in her initial management since, paradoxically, hydroxychloroquine, used to treat SS symptoms, can exacerbate symptoms, unmask previously undiagnosed MG or precipitate MG. Chloroquine, a precursor of hydroxychloroquine, might stimulate the production of AChR antibodies, which act as immune checkpoint inhibitors (ICI), thereby exacerbating MG through a direct impact on neuromuscular transmission.[8] This overlap between MG and SS emphasizes the complexity of autoimmune diseases and the importance of considering coexisting conditions in patient evaluations.

Sjogren's syndrome (SS) is considered a complex disorder affecting multiple systems and presents diverse neurological manifestations. The documented occurrence of neurologic manifestations in SS varies between 5% to 57%.[20] About 36% of cases might solely involve the nervous system. Intriguingly, neurological signs may manifest before characteristic symptoms appear by as much as 6 years in 47% of cases and can overlap with other autoimmune pathologies like Myasthenia Gravis or Guillain Barre Syndrome. The neuropathy linked to SS often goes unnoticed, despite its prevalence.[20]

Delays in diagnosis are common as the nerve-related symptoms may appear before the broader systemic signs. Utilizing a minor salivary gland biopsy proves more effective in diagnosis compared to presently available blood markers.[20] However, the prognosis for those diagnosed with SS-associated neuropathy is not typically unfavorable in the future.[20]

Multiple simultaneous autoimmune disorders present challenges in treating MG due to overlapping symptoms and the need for multiple immune system drugs.[18, 21]

In the case discussed, the patient was initially administered hydroxychloroquine. However, this treatment was halted immediately after a diagnosis of Sjögren's syndrome (SS) was made. Consequently, the patient exhibited only mild symptoms of SS, which were primarily neurological, such as dysphagia and difficulties in swallowing. These symptoms are common in MG as well. Notably, the patient did not experience symptoms related to the secretory glands, such as xerostomia or xerophthalmia, indicating she did not need any other treatment. This allowed for the successful management of SS with steroids and mycophenolate mofetil. Fortunately, these medications are also used in the treatment of MG, which eliminated the need for multiple drugs.

Most patients with MG have a near-normal lifespan with the current treatment modalities. Morbidity results from the intermittent muscle weakness leading to aspiration pneumonia and the adverse effects of medications. Age of onset (<40 years), early thymectomy, and administration of prednisolone are found to be associated with reduced risk of relapse.[22] However, patients with concomitant autoimmune disease showed a high rate of relapse.[22] Severe morbidity due to Sjogren's syndrome is uncommon in patients with both diseases. Thus, controlling MG is the critical aspect of treatment.

Considering the findings presented by Pierce et al. in their epidemiological investigation of swallowing disorders in Sjogren's Syndrome (SS), it is plausible to consider that the patient's symptoms were a result of both diagnosed pathologies. The prevalence of a current self-reported swallowing disorder in SS patients, as reported, was 64.4%. Notable symptoms associated with this include taking smaller bites, thick mucus in the throat, difficulty placing food in the mouth, and wheezing while eating. Furthermore, additional risk factors such as the presence of a voice disorder, frequent neck or throat tension, and frequent throat clearing could have compounded the patient's dysphagia.[23]

Considering these factors in conjunction with the patient's MG, which itself can cause muscle weakness affecting swallowing, it becomes increasingly evident that her symptoms likely stem from a complex interplay of both conditions.

A comprehensive literature review was done to contextualize our patient's case within the broader landscape of concurrent MG and SS. As of 2020, there have been approximately 20 case reports documenting the co-occurrence of these two conditions. Notably, most of these cases have been reported in countries like China, India, Germany, and the United States.[24] In India, while there have been multiple reports of Myasthenia overlap, only one case has been definitively identified as MG coexisting with Sjogren's.To the best of our knowledge, there have been no reported cases of patients of Hispanic origin with this particular combination of diagnoses in the existing literature.

The most recent contribution to this field is a 2022 report indicating an ongoing interest and recognition in the medical community regarding the intersection of these two diseases titled "Association of early-onset myasthenia gravis and primary Sjögren's syndrome: a case-based narrative review," published in the Clinical Rheumatology journal.[26] This indicates an ongoing interest and recognition in the medical community regarding the intersection of these two diseases.

This article highlights the rarity and complexity of managing a patient with both Myasthenia Gravis and Sjogren's Syndrome, particularly in underrepresented populations given that these communities often encounter various obstacles in obtaining health care and medical treatments, it is important to note that Hispanic adults generally have lower rates of health insurance coverage and access to preventive medical services compared to other American groups.[27] Barriers related to language and culture, along with issues like increased poverty rates, especially among recent immigrants from Hispanic backgrounds, play a significant role in the differing health results observed within the Hispanic population in America.[27] Disparity outcomes are well known between Hispanic Americans vs. other groups and it is estimated that 44% of this is related to communication and cultural barriers.[27] It is interesting to note that this patient drove from over 500 miles away to our clinic because she heard that care was rendered in Spanish in a culturally sensitive manner.

Table 1: Patient's laboratory results:

Laboratory	Patients Results	Reference Interval
Acetylcholine Receptor Ab, All	-	-
AChR Binding Abs, Serum	3.65	Negative: 0.00 - 0.24 Borderline: 0.25 - 0.40 Positive: >0.40
AChR Blocking Abs, Serum	68	Negative: 0 - 25 Borderline: 26 - 30 Positive: >30
Lyme Total Antibody CIA	Negative	Negative
ANA Direct	Positive	Negative
Anti-DNA (DS) Ab Qn	1	0 - 9
RNP Antibodies	<0.2	0.0 - 0.9
Smith Antibodies	<0.2	0.0 -0.9
Sjogren's Anti- SS-A	>8.0	0.0 - 0.9
Sjogren's Anti-SS-B	0.2	0.0 - 0.9
Phosphorus	4.5	3.0 - 4.3
PTH	34	15 - 65

Laboratory	Patients Results	Reference Interval
Cell Count, CSF	-	-
Color	Colorless	Colorless
Clarity	Clear	Clear
Nucleated Cell	1	0 - 5
RBC	650	None Seen
Comment	Rare lymphocyte seen	

Preexisting health conditions are also seen as a factor: 40% say a major reason for worse health outcomes is that Hispanic people are more likely to have preexisting conditions.[27] Also, health care providers being less likely to give Hispanic people the most advanced medical care and that hospitals and medical centers giving Hispanics' well-being lower priority are a major reason for health disparities.[27] All these underscore the need for continued reporting of such cases to enhance our understanding and improve the diagnostic and therapeutic approaches for similar patients in the future.

Laboratory	Patients Results	Reference Interval
Glucose, CSF	61	49 – 73
Protein, Total, CSF	12.1	0.0 - 44.0

Table 2: Differential Diagnosis of MG

Diagnosis	Differentiating clinical features
Thyroid eye disease	Exophthalmos
Motor neuron disease	No ocular involvement, upper motor neuron features, muscle wasting.
Botulism	Pupillary involvement
Congenital cranial nerve palsies.	In distribution of cranial nerves
Myopathies	Muscle weakness, including ocular or bulbar muscle weakness that can mimic MG.
Myositis	Muscle weakness with common constitutional symptoms such as fever and weight loss.
Guillain-Barre Syndrome	Muscle weakness shown with an ascending pattern. Autonomic nervous system involvement, diminished or no reflexes, sensory signs and symptoms
Lambert-Eaton myasthenic syndrome	Spared ocular muscles. Hyporeflexia and involvement of autonomic nervous system.
Cytopathies (mitochondrial)	There can be fluctuations in mitochondrial disorders e.g. with increase in metabolic demands, such as in illnesses. Clinical features that can help differentiate with MG, is the involvement of other systems such as cardiac, ocular (other than extraocular muscle and eyelid motility), GI, cognitive impairment and neuropsychiatric symptoms, and seizures.
Oculopharyngeal muscular dystrophy	Tongue weakness or atrophy. Dysphagia (with solid foods)
Congenital myasthenic syndromes	No response to immunotherapy. Onset in childhood. Seronegativity

Adapted from Sathasivam S. Diagnosis and management of myasthenia gravis. Prog Neurol Psychiatry. 2014;18(1):6-14. doi:10.1002/pnp.315, table no. 2.

Table 3: Etiology of Oropharyngeal Dysphagia

Category	*Diseases / Conditions*
Neurologic	Stroke, Parkinson's, MS, ALS, Alzheimer's, Huntington's
Myopathic	Myositis, Dermatomyositis, Muscular dystrophies
Metabolic	Hyperthyroidism, Advanced diabetes
Inflammatory/Autoimmune	Amyloidosis, Sarcoidosis, SLE, Sjogren's, Myasthenia Gravis, Rheumatoid arthritis
Infectious	Meningitis, Diphtheria, Botulism, Lyme, Syphilis, Various viral infections, HIV/AIDS

Structural	Inflammatory (Pharyngitis, Abscess, Tuberculosis), Congenital Webs, Zenker Diverticulum, Neoplasms, Compression, Eosinophilic esophagitis, Bullous Skin Diseases.
Iatrogenic	Drug side effects, Radiation, Corrosive injury

Adapted from Gasiorowska, Anita & Fass, Ronnie. (2009). Current Approach to Dysphagia. Gastroenterology and Hepatology. 5.

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Protection of human and animal subjects. Informed consent was obtained from the patient for publication of this case report.
Confidentiality of data. No data that identifies patients are revealed.

Note from authors: In addressing the dual diagnosis of Myasthenia Gravis and Sjogren's Syndrome in our Hispanic patient, we, as a predominantly Hispanic group of authors, bring a personal and professional understanding of the ethical, legal, and cultural dimensions critical to providing effective and empathetic care. Ethically, we prioritize culturally competent care, ensuring our approach respects the patient's cultural values and beliefs, thereby enhancing patient trust and treatment adherence. Legally, we adhere to informed consent and confidentiality, employing language services and culturally tailored materials to ensure the patient's full understanding and participation in their care.

Culturally, we recognize and address the barriers Hispanic patients often face in healthcare, such as language barriers and cultural stigmas, through empathetic engagement and integrating traditional health practices where appropriate. Our shared cultural background with the patient enriches our perspective, allowing us to navigate these complexities with sensitivity and insight, aiming to reduce health disparities and promote equitable care. This commitment reflects our dedication to advancing health equity and underscores the importance of culturally informed research and practice in medicine.

Disclaimer: The views expressed in this paper are those of the authors and not necessarily of the authors' organizations or the National Hispanic Medical Association (NHMA). The paper is intended to help inform and stimulate discussion. It is not a report of NHMA.

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