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A 32-Year-Old Palestinian Female Patient Diagnosed with Systemic Lupus Erythematosus Two Years After Being Diagnosed with Myasthenia Gravis: A Case Report with Literature Review

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Abstract

Introduction

Systemic lupus erythematosus (SLE) is a chronic, systemic inflammatory autoimmune disorder defined by the presence of several autoantibodies that affect multiple organs. Myasthenia gravis (MG) is an organ-specific autoimmune condition characterized by the presence of several autoantibodies. However, the coexistence of SLE and MG is rare, with only a few cases reported in the literature.

Case Presentation

We report a case of a female patient who presented to the neurology clinic with generalized weakness, fatigue, dyspnea, dysphagia, and dysphonia. She was diagnosed with anti-acetylcholine receptor antibody-positive MG. CT was normal and the EMG was normal. After two years she developed polyarthralgia, oral ulcers, and photosensitivity. Bloodwork revealed a positive antinuclear antibodies (ANA) test. She was diagnosed with SLE and was initiated on hydroxychloroquine (200 m/day).

Discussion

The combination of MG and SLE poses clinical challenges due to the potential for confusion. Both conditions are common in women and both are accompanied by the presence of antinuclear antibody. MG is an organ-selective antibody directed against acetylcholine receptors, while SLE attacks multiple organs and elicits various antibodies, mainly against nuclear antigens and double-stranded DNA. Studies show that people with MG also have an increased likelihood of experiencing severe clinical manifestations of SLE. The most common signs of lupus are polyarthritis and polyarthralgia, while other common symptoms include rash, fever, and pleurisy. Most people with myasthenia gravis require immunosuppressants to achieve treatment goals of full physical function or a relatively good quality of life. 8% of MG patients had SLE disease that is mean the prevalence of SLE in MG Patients was higher than general population.

Conclusion

A literature review revealed 11 cases of SLE/MG overlap described in seven publications from 2012 to 2022. This study suggests that SLE should be suspected in any MG patients with polyarthritis. Further studies are needed to better understand the connection between SLE and MG disorders.

Keywords

Systemic lupus erythematosus, Myasthenia gravis, case report, literature review

Highlights

The majority of SLE/MG overlap patients were females.

The most common MG manifestation in patients with SLE was Ocular weakness.

Arthritis was the most common SLE symptom in MG patients.

Introduction

Systemic Lupus Erythematosus (SLE) is a chronic systemic inflammatory autoimmune disorder defined by the presence of several autoantibodies, and young females are most commonly affected. **[1,4]**

On the other hand, Myasthenia Gravis (MG) is an organ-specific autoimmune condition characterized by the presence of several autoantibodies directed against the nicotinic acetylcholine receptor (nACHR), the muscle-specific tyrosine kinase (MuSK) or lipoprotein receptor-related protein 4 (LRP4). Consequently, clinical presentations such as ocular symptoms, dyspnea, and fluctuating muscle weakness can occur. **[2,3,4]** Men and women are both affected and mostly affects older men and young adult women. **[1,4]**

SLE and MG have common features since both are more common in young women and both have positive anti-nuclear antibodies. MG may appear before or after the onset of SLE. However, SLE and MG coexistence is rare, and few cases have been reported in the literature. **[5,6,7]** Two literature reviews of SLE/MG overlap syndrome were documented in 2011 **[8]**. These reviews included more than 30 cases. The majority of patients meet the ACR criteria for the diagnosis of SLE. Most of them were females in both studies. Arthritis was the most common manifestation in these patients, similar to patients in different SLE cohorts. Ocular weakness was the most common initial presentation in both articles. **[9,10]**

Herein we report a case of MG in a 32-year-old female patient, who developed polyarthralgia, oral ulcers, photosensitivity, and Raynaud's phenomenon, with a laboratory diagnosis of SLE two years after being diagnosed with Myasthenia Gravis.

Case Presentation

A 32 -year-old female patient with no past medical history, presented to the neurology clinic in April 2014 with generalized weakness, fatigue, dyspnea, dysphagia, and dysphonia. She was diagnosed with anti-acetylcholine receptor antibody-positive MG. CT was normal and the EMG was normal. She was treated with azathioprine (100mg), prednisone (10 mg daily), and pyridostigmine (300 mg daily).

The patient was admitted to the hospital multiple times for MG relapse symptoms, including general weakness, dyspnea dysphagia, and dysphonia and she was treated with pulse therapy (methylprednisolone 1gm for three days), Intravenous immunoglobulin (IVIG), and two times with Plasmapheresis. She had a good response to IVIG treatment.

In 2016, she developed polyarthralgia, oral ulcers, and photosensitivity. She was complaining of episodic color changes of the fingers in response to cold and emotional stress, for a long time. Family history was positive for SLE and Rheumatoid arthritis.

On examination, the patient appeared to be oriented, alert, in no respiratory distress, and was afebrile and hemodynamically stable. She had Cushingnoid features with malar rash and muscle wasting. No skin rash or lesions were noted. The liver, spleen, and kidneys were not palpable. Neurologic examination revealed normal upper limb power with lower limb weakness, more on the left side. The coordination, tendon reflexes, gait, and sensory examination were within normal limits. Her chest examination revealed bilateral normal vesicular breathing with no added sounds.

Bloodwork revealed a positive antinuclear antibodies (ANA) test (1:640), normal complement, a negative rheumatoid factor, a positive extractable nuclear antigen (ENA) test, a negative double-stranded deoxyribonucleic acid (dsDNA), and a strong positive Centromere B, RNP\Sm, Ro-52 and Control (co). She was initiated on hydroxychloroquine (200 mg/day). Currently, she receives Azathioprine (100mg daily), prednisolone (30 mg daily), hydroxychloroquine (400mg daily), and pyridostigmine (300mg daily). And her condition is well controlled, and her medical regimen is well tolerated.

Discussion

Myasthenia gravis (MG) is an autoimmune disease characterized by profound fatigue and proximal muscle weakness caused by the degradation of acetylcholine receptors at neuromuscular junctions. Systemic lupus erythematosus (SLE) is another autoimmune disorder that primarily affects the neurological system, lungs, heart, kidneys, skin, joints, and serosa. Approximately 5% of MG patients also have a second autoimmune disorder, with thyroid disease being the most common **[2]**. In most studies, hypothyroidism is more prevalent than hyperthyroidism. There are several explanations for this association, including the potential for distinct ocular and generalized MG being associated with a variety of other autoimmune diseases, cross-immunological response to thyroid and eye muscle epitopes or autoantigens, and a hereditary factor **[11]**. Women in reproductive age groups make up the majority (90%) of SLE patients, with the frequency of SLE ranging from 37 to 178 instances per 100,000 people **[3]**. In one retrospective analysis of 215 SLE patients, 30% also had another autoimmune condition, with Sjögren's syndrome being the most common **[2]**.

The combination of myasthenia gravis (MG) and systemic lupus erythematosus (SLE) can be a clinical challenge due to the potential for misdiagnosis when dealing with muscle involvement in SLE patients. However, both MG and SLE can present with similar symptoms and occur concurrently in a patient, although this is an uncommon occurrence. Women tend to be more affected by both conditions, which are both associated with positive anti-nuclear antibodies **[4]**. It is believed that genetic, environmental, hormonal, and immunological variables contribute to the development of both disorders **[12]**.

The reported prevalence of SLE and MG comorbidity is typically 2.6%, but in a study by Sthoeger Z. et al. on 78 patients, the rate increased to 7.7%, with a predominance of women **[11]**. MG is an organ-selective autoimmune response to acetylcholine receptors, while SLE is a systemic autoimmune disorder that affects multiple organs, causing a broad range of antibodies, mostly to nuclear antigens and ds-DNA, as well as decreased T- and B-cell activity **[12]**. It has been noted that there is a connection between SLE and MG in patients who experience loss of central tolerance following thymectomy, leading to T-cell lymphopenia, polyclonal B-cell activation, and antibody production, or due to molecular mimicry and structural

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similarity between the main immunogenic region of 65-80 of AchR and U1 small nuclear ribonucleoprotein.

Additionally, chloroquine has been observed to have an effect on the neuromuscular junction, producing myasthenia-like syndrome, and dysregulated T cells and B lymphocytes have both been linked to the pathogenesis of MG and SLE, respectively. The common B and T lymphocyte activator CXCL13 has also been implicated **[2]**.

The prevalence of MG and SLE comorbidity varies depending on the main condition, with a frequency of up to 1.3% of MG in SLE patients and up to 8% of SLE in MG patients; however, a high incidence in women is a common feature.**[4]** In the case presented to you, MG manifested before SLE.

The most common symptoms of systemic lupus erythematosus (SLE) are polyarthritis and polyarthralgia. Other common manifestations include skin rashes, fever, cytopenia, and pleuritis. Less frequent signs of SLE include optic neuritis and transverse myelitis.**[13]** Polyarthralgia was the most common symptom present in our patient and his condition was effectively managed with hydroxychloroquine, azathioprine, and low-dose prednisone.**[14]**

Treating myasthenia gravis requires a multifaceted approach. Initially, symptomatic, safe, and supportive techniques are recommended to reduce the severity of symptoms. Long-term treatments typically involve immunosuppressive medications and may involve additional therapies, such as plasma exchange or intravenous immunoglobulins, depending on the individual's response.**[15]**

A variety of immunosuppressive medications are available for myasthenia gravis, including azathioprine, cyclosporine, mycophenolate, tacrolimus, rituximab, and prednisone.**[16]** Azathioprine, mycophenolate, and rituximab are the most commonly prescribed medications, although the choice of medication will depend on the individual's condition and particular response.**[17]**

It is important to note that immunosuppressive medications for myasthenia gravis can have serious side effects, so it is important to closely monitor the patient's response to therapy and adjust the dose as needed.**[18]** In this case, the patient's treatment regimen was well tolerated.

The patient presented in this case was treated first with azathioprine (100mg), prednisone (10 mg daily), and pyridostigmine (300 mg daily) for MG. After that, She was initiated on hydroxychloroquine (200 mg/day) since she was diagnosed with SLE. Currently, she receives Azathioprine (100mg daily), prednisolone (30 mg daily), hydroxychloroquine (400mg daily), and pyridostigmine (300mg daily) And her condition and medication regimen are well controlled and tolerated.

Due to the widespread incidence of myasthenia gravis and its numerous unfavorable effects on people and society and because of different and small numbers of reports of MG\SLE prevalence, a detailed report of the prevalence of these diseases in different characteristic in order to pay more attention to planners and its consequences seemed necessary. A literature search for articles published between 2012 and 2022 was carried out in Medline using the databases PubMed and a comprehensive search strategy that included the terms SLE and MG. 11 patients with the association of MG and SLE were reported **[2,3,5,6,7,10,19,20,21]**. The information on each of these papers is presented in Error! Reference source not found.**,** Error! Reference source not fo und.**, and** Error! Reference source not found.

Table 1 Anti-AChR ab: anti-acetylcholine receptor antibodies; HCQ: hydroxychloroquine; MG: myasthenia gravis; SLE: systemic lupus erythematosus; APS: anti phospholipid syndrome; NR: not reported; SOB: shortness of breath; Anti –VGKC: Anti-voltage-gated potassium channel; ANA: antinuclear antibodies; ENA: extractable nuclear antigens; Anti -SM: Anti -Smith; Anti-RNP: Anti-Ribonucleoprotein.

Patient Demographics and Clinical Pictures

case	Age	SLE onset in comparison to MG diagnosis	SLE after thymectomy	SLE Treatment	MG Treatment	SLE manifestations	MG manifestations	Associated diseases
Liu et al. [5]	19 F	After 2 years	Yes	Nifedipine HCQ Indomethacin	Pyridostigmine Azathioprine Plasmapheresis thymectomy	Polyarthralgias, oral ulcers, patchy alopecia livedo reticularis on the hands and legs	weakness, left-sided ptosis, fatigue and dyspnea	Paraneoplastic Limbic Encephalitis

Klimi et al. [6]	55 M	After 38 years	Yes	NR	Pyridostigmine Azathioprine thymectomy	CVA, arthritis, and lesions on his hands typical of discoid lupus	NR	Nil
Miscovic et al. [3]	48 F	After 20	Yes	glucocorticoid mycophenolate mofetil	Raynaud's phenomenon photosensitivity, polyarthritis, polyserositis lupus nephritis	NR	APS(PE)	ANA, ds-DNA, cryoglobulins, anti-cardiolipin antibodies (IgG,IgM)
Sawamura et al. [7]	75 F	4 month before	Yes	cyclosporine and cyclophosphamide pulse therapy steroid pulse therapy Prednisolone	Pscattered erosive erythema	ptosis and fatigability of the upper limb	pemphigus foliaceus and chronic thyroiditis thymoma-postoperative MG	Anti-AChR anti-thyroglobulin, ds-DNA, anti-desmoglein Proteinuria
Minchenberg et al. [19]	62 F	23 after	Yes	HCQ	Pyridostigmine	polyarthritis	weakness, diplopia, ophthalmoplegia, dysphagia	Nil
Minchenberg et al. [19]	58 F	1 year before	NO	HCQ	NR	MCP Joint pain, SOB,chest pain,seizures, Stroke, malar rash	Ptosis, Diplopia	APS
Minchenberg et al. [19]	56 F	After	Yes	HCQ	Pyridostigmine	NR	weakness, dysphagia, ptosis	Nil
Minchenberg et al. [19]	57 M	After 3 year	No	HCQ, mycophenolate mofetil cholinesterase inhibitor	Synovitis , PIP &MCP joint pain	respiratory dysfunction, proximal muscle weakness, ptosis dyspnea, diplopia, gait instability	APS	anti-AChR ANA

Patient Demographics and Clinical Characteristics

case	age	SLE onset	SLE after thymectomy	SLE Treatment	MG Treatment	SLE manifestations	MG manifestations	Associated diseases	Labs positive

Chitasomba t et al. [20]	38 F	Before 13	No	Azathioprine HCQ	Pyridostigm ine	fever, polyarthritis , oral ulcer, alopecia, and proteinuria	proximal muscle weakness, ptosis	varicella, CMV syndrome/jej unitis L.pneumophi la infection resulting in panniculitis, myositis, and myocarditis	anti-AchR
Fleischman n et al. [21]	52 F	Before 37	No	Steroid Pyridostigmine IVIG Plasmapheresis, methylprednisol one azathioprine, rituximab, belimumab	lupus nephritis	weakness, diplopia, dysphagia , respiratory failure	overlap syndrome of MuSK-Ab-positive MG and SLE , APS, hemophago cytic lymphohisti ocytosis causing multi-organ dysfunction, (EBV)	cardiolipin IgG/IgM,mus cle-specific kinase, beta2-glycoprotein IgG/IgM,AN A, Anti-dsDNA, APSA, VGCC, thyroglobulin	
Nagarajan et al. [2]	38 F	Before One year and 5 month	No	Methylprednisol one, intravenous cyclophosphami de 500 mg, HCQ mycophenolate mofetil	Pyridostigm ine	Polyarthritis , lupus nephritis	Ptosis	Nil	dsDNA, ANA 3+ proteinuria
Our case	32 F	After 2 years	No	HCQ Azathioprine Pyridostigmine IVIG Plasmapheresis methylprednisol one	polyarthralg ia, oral ulcers, photosensiti ve eye dryness. Raynaud's phenomeno n	generalized weakness, fatigue, dyspnea, dysphagia, and dysphonia	Nil	Centromere B, RNP\Sm ,Ro-52 and Control (co) ANA ENA Anti-AChR	

Table 2 Clinical and laboratory findings of patients with MG and SLE

Total N	**12**
SLE onset: Before MG After MG	 5(41.6) 7(58.3)
Female gender	10(83.3)

SLE after thymectomy	5(41.6)
SLE Treatment	
HCQ	8(66.6)
Azathioprine	2(16.6)
mycophenolate mofetil	3(25)
Cyclophosphamide	2(16.6)
MG Treatment	
Pyridostigmine	9 (75)
Plasmapheresis	3(25)
IVIG	2(16.6)
rituximab & belimumab	1(8.3)
Azathioprine	3(25)
SLE manifestations	
Raynaud's phenomenon	2(16.6)
Photosensitivity	2(16.6)
Polyarthritis	7(58.3)
polyserositis	1(8.3)
lupus nephritis	3(25)
oral ulcer	3(25)
patchy alopecia	2(16.6)
livedo reticularis	1(8.3)
CNS	2(16.6)
malar rash	1(8.3)
discoid lupus	1(8.3)
Pscattered erosive erythema	1(8.3)
unknown	1(8.3)
MG manifestations	
respiratory dysfunction	2(16.6)
muscle weakness	8(66.6)
ptosis	7(58.3)
dyspnea	3(25)
diplopia	4(33.3)
gait instability	1(8.3)

dysphagia	4(33.3)
dysphonia	1(8.3)
unknown	2(16.6)
clinical stage at onset	
ocular	2 (18)
generalized	8(66.6)
myasthenia crisis	0(0)
bulbar dysfunction	4(33.3)
Labs	
anti-AchR	8(66.6)
cardiolipin IgG/IgM	2(16.6)
muscle-specific kinase	1(8.3)
beta2-glycoprotein IgG/IgM	1(8.3)
ANA	9(75)
Anti-dsDNA	5(41.6)
APSA	1(8.3)
VGCC	1(8.3)
Thyroglobuline	2(16.6)
SM	1(8.3)
RNP	1(8.3)
DRB114DRB116:02(HLA)	1(8.3)
Cryoglobulins	1(8.3)
Centromere B, RNP\Sm, Ro-52 and Control (co)	2(16.6)
ENA	

All patients fulfilled four of the 11 ACR criteria for the diagnosis of SLE. 1.3% of SLE patients developed MG. In addition, 8% of MG patients had SLE disease that is mean the prevalence of SLE in MG Patients was higher than in the general population. **[10]**

The majority of patients were females (83.3%). The diagnosis of MG preceded the SLE diagnosis in seven of the patients (58.3%). Of these, five patients developed SLE after thymectomy .The frequencies of the SLE clinical manifestations are similar to those observed in different SLE cohorts. Arthritis was the most common manifestation in these patients (58.3%). Other frequent manifestations included Raynaud's phenomenon (16.6%), photosensitivity (16.6%), oral ulcers (25%), serositis (8.3%), renal disorder (25%), and skin manifestation (41.6%). Neurological disorders other than MG were reported in only two patients (16.6%). Nine patients were ANA positive (75%) and five patients were anti-dsDNA positive (41.6%). Three patients (25%) had the antiphospholipid syndrome. Anticardiolipin antibodies were found in two patients (16.6%). Another associated disease is presented in **[**Error! Reference source not found.**]**. Treatment of SLE n

ecessitated HCQ (66.6%), Azathioprine (16.6%), cyclophosphamide (16.6%), and/ or mycophenolate mofetil (25%).

The most common MG manifestations in these patients were Ocular weakness, with fluctuating ptosis and/or diplopia (75%), five patients (41.6%) presented with ptosis, two (16.6%) with diplopia and two patients (16.6) had ptosis with diplopia. Muscle weakness was reported in 8 patients (66.6%) two patients (16.6%) had respiratory dysfunction and one of them died 10. The most common immunological abnormality in MG is the presence of AChR antibodies. These were detected in eight patients (66.6%). However, we found one patient had positive MuSK antibodies. Nine patients were treated with acetylcholinesterase inhibitors (75%). Treatment of MG also required steroid pulse therapy (33.3%), azathioprine (27.2%), mycophenolate mofetil (16.6%), intravenous immunoglobulins (16.6%), and/or plasma exchange (25%).

Table3 Patients with MG, SLE, and Other Associated Diseases. SS; Sjogren's Syndrome

Associated Diseases
SS &DVT [8]
non-uremic calciphylaxis[8]
Paraneoplastic Limbic Encephalitis [2]
APS [4,6,10]
pemphigus foliaceus [5]
chronic thyroiditis[5]
varicella[7]
CMV syndrome[7]
L.pneumophila infection resulting in panniculitis, myositis, and myocarditis [7]
Giant cell myositis, metastatic thymoma, granulomatous hypercalcemia [9]
hemophagocytic lymphohistiocytosis causing multiorgan dysfunction, (EBV) [10]

Conclusion

In conclusion, the association between SLE and MG rarely occurs. MG diagnosis preceding the diagnosis of SLE in some patients and coexisted in other patients. In any MG patients with arthritis, SLE should be suspected. In addition, interdisciplinary care and communication between medical teams and patients are paramount for the successful management of both illnesses. Understanding the risks and outcomes of coexisting diagnoses of SLE and MG is important for the improvement of patient outcomes and quality of life.

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