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Research Report

Myasthenia Gravis: Unusual Presentations and Diagnostic Pitfalls

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Abstract.
Background: Myasthenia gravis (MG) is an autoimmune disorder presenting with fluctuating, fatigable muscle weakness. Initial symptoms classically involve ocular and proximal limb muscles. Rarely, MG may onset with unusual features, so it can be misdiagnosed with other neuromuscular diseases.
Objective: To describe unusual and atypical presentations of MG in a large cohort of patients, considering and discussing diagnostic difficulties and pitfalls.
Methods: We report on 21 out of 508 MG patients, coming to our department in the last 27 years and presenting with atypical or unusual features. The diagnosis was achieved performing a careful clinical examination, a proper neurophysiological assessment, the neostigmine test, the AChR and MuSK antibodies assay and chest CT-scan.
Results: Patients with atypical/unusual MG onset were the 4.4% of all MG patients population. We describe seven different clinical categories: asymmetric distal upper limbs weakness, foot drop, isolated triceps brachii weakness and foot drop, post exertional axial weakness with dropped head, acute facial dyplegia, limb-girdle MG and MG with sudden lower limbs weakness and recurrent falls.
Conclusions: Atypical and unusual presentations may increase the risk to misdiagnose or delay MG diagnosis. Isolated limb-girdle presentation is the most frequent atypical form in our series.

Keywords: Myasthenia gravis, limb girdle myasthenia, distal myasthenia, foot drop

INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder, characterized by a wide spectrum of clinical presentations, ranging from purely ocular symptoms to severe generalized forms. It may be related to different antibodies profiles against several neuromuscular junction components (NMJ). The most frequent form of MG is related to antibodies against the acetylcholine receptors (AChR-abs), whereas MG associated to muscle-specific kinase receptors antibodies (MuSK-abs) is generally characterized by a major involvement of bulbar muscles, atrophic tongue, worsening with acetylcholinesterase inhibitors (AChEI) and absent thymic pathology [1–4]. Most recently, several patients with MG associated to lipoprotein related protein 4 (LRP4) antibodies have been reported [5–7]. The clinical hallmark of MG is the presence of fatigable muscle weakness. Classically, onset symptoms involve the ocular muscles, including eyelid ptosis, fluctuant diplopia, or both. Approximately 20% of patients with MG present, at onset, bulbar symptoms, including dysarthria, dysphagia and dysphonia [1–3, 8]. Rarely, patients with MG may present at onset with unusual features, such as head drop syndrome, acute

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bilateral facial weakness or isolated bulbar weakness, sudden lower limbs weakness and falls, or with atypical presentations including foot-drop, isolated distal weakness without atrophy, limb-girdle muscle weakness, in absence of the classical fluctuation of the disturbances and without extra-ocular muscles involvement. For these cases, the diagnosis of MG represents a diagnostic challenge [10–17]. Herein, we report the atypical and unusual presentations of MG in a large cohort of patients, examined at our Neurological department over the past 27 years, considering and discussing diagnostic difficulties and pitfalls.

PATIENTS AND METHODS

In the last 27 years (1987–2014) we followed-up 508 patients affected by MG. Age at onset of this cohort (263 males and 245 female) ranged between 11 and 90 years. Serum antibodies assay revealed the presence of AChR-abs in 413 patients, whereas MuSK-abs were in 23 present. The remaining 72 patients were negative. The diagnosis was achieved performing a careful clinical examination, a proper neurophysiological assessment, including single fiber electromyography (SFEMG) and repetitive nerve stimulation (RNS), the neostigmine test and a chest CT-scan. All patients were tested for AChR- and MuSK-abs; LRP4-abs were not tested in negative patients. The frequency of follow-up visits and hospital admissions was variable, depending on the severity of symptoms and on treatment response. Herein, we report on 21 out of 508 patients, 10 males (M) and 11 females (F), aged from 14 to 70 years, presenting with atypical or unusual clinical phenotypes. In the following paragraphs we describe different categories of unusual MG presentation, classified by symptoms at onset.

ATYPICAL MG PRESENTATIONS: CLINICAL CATEGORIES

Asymmetric distal upper limbs weakness without atrophy (cases 1-2-3)

Three patients (M 30-y, F 20-y, F 40-y) complained of subacute muscle weakness and distal limb muscles fatigability. In particular, in *case 1,* intrinsic hand muscles and wrist extensor muscles involvement had been preceded by a long history of ocular myasthenia, whereas, in *cases 2* and *3*, no other muscles have been previously involved. AChR-abs were increased in all patients, thymoma was found in two out of three patients. They all responded to a combined AChEI and steroid treatment; thymectomy was performed in patients with tymoma. Because of the diagnostic challenge, we reported, in detail, an illustrative case *(case 1).*

Case 1

A 22-year-old man came to our Clinic because of bilateral eyelid ptosis and diplopia, started after a fever. These symptoms fluctuated during the day, so that he was investigated for MG. Serum AChR-abs were negative. SFEMG, performed on the orbicularis oculi muscles, disclosed an abnormal NMJ function, whereas SFEMG of the extensor indicis muscle was normal. Chest CT-scan did not show thymus abnormalities. Patient received a diagnoses of ocular myasthenia and AChEI and prednisone were administered without benefit. After 6 months, he improved and prednisone was gradually tapered. Eleven years later, he was forced to use crutches for 1 month because of a traumatic right tibia and fibula fracture; after two weeks, he was unable to extend his right wrist and, one month later, also the left wrist. Neurophysiological examination, performed in another hospital, ruled out a lesion, due to compression, of the radial nerves or of the posterior interosseous branches, hereditary neuropathy with liability to pressure palsy (HNPP) or multifocal motor neuropathy (MMN). For this reason, the patient come back to our Clinic. Neurological examination disclosed a severe weakness of the right extensor, left wrist and of the third and fourth hand fingers muscles (video 1 - supplementary materials). No sensory symptoms were referred. A generalized form of MG was then suspected. RNS of the right ulnar nerve, recording from the abductor digiti minimi muscle, disclosed a decremental pattern, whereas the SFEMG, performed on the right extensor indicis muscle, evidenced the presence of 80% of recordings with increased jitter, 30% of recordings with conduction blocks and a mean MCD of 95 μs. The neostigmine test showed a considerable improvement of symptoms (video 2 - supplementary materials). Serum AChR-abs were positive (1.90 nmol/L with normal values <0,4 nmol/L), whereas chest CT-scan did not reveal thymic abnormalities. The patient was diagnosed of generalized myasthenia gravis with distal involvement; he was treated with AChEI and prednisone 50 mg/die, with a progressive clinical improvement.

MG with isolated foot drop, without atrophy: (Case 4)

A 16-year-old man came to our attention because of subacute and fluctuant onset of a bilateral foot drop. Neurological examination showed isolated bilateral foot and ankle dorsi-flexors muscles weakness. These symptoms worsened while walking. No sensory symptoms were referred. Details about patient's immunological profile, neurophysiological study and treatment are reported in Table 1.

Isolated triceps brachii weakness and foot drop, without atrophy: (Case 5)

A 40-year-old man complained a 5-years history of progressive difficulty and fatigability to keep his arms raised, worsened by repeated exercise. Neurological examination showed mild facial weakness (orbicular oris and oculi) and bilateral triceps brachii muscle weakness; he was able to keep the upper limbs raised only for few seconds (video 3 - supplementary materials). There was a mild right ankle dorsi-flexors muscles weakness with a slight "steppage". Serum AChR-abs were positive (0.65 nmol/L). SFEMG of triceps brachii muscle showed 40% of recordings with increased jitter, 20% of recordings with blocks and a mean MCD of 112 μs. RNS of the peroneal nerve, recording from the anterior tibial muscle, confirmed a neuromuscular transmission disorder. Chest CT-scan was normal. He had a good response to steroids, which is still ongoing.

Limb-girdle myasthenia gravis (LGMG): (Cases 6-7-8-9+10-11-12-13-14)

Four patients, *cases 6–9*, (M 24-y, M 43-y, M 32-y, F 30-y) were admitted to our Clinic during a 15-years period and their data have been already published [15]. In the last 12 years, we have evaluated other 5 additional cases, presenting with a LGMG phenotype *(cases 10–14)* (F 38-y, F 44-y, M 41-y, F 56-y, M 30-y). Details are reported in Table 1.

UNUSUAL MG PRESENTATIONS: CLINICAL CATEGORIES

Post exertional axial weakness with dropped head: (Cases 15-16-17)

We report three patients (M 70-y, F 65-y, F 43-y) who complained of a progressive neck extensor muscles weakness, worsened by walking, even for short distances (video 4 – supplementary materials). A RNS of the right ulnar nerve, recording from the abductor digiti minimi muscle, was normal in all patients. SFEMG of the extensor digitorum communis muscle (EDC) disclosed, in *case 15,* an increased jitter with conduction blocks, but, in *cases 16* and *17* it was normal at the same site and abnormal on the trapezius muscle, revealing an increased jitter and conduction blocks. Serum AChR-abs were absent, whereas MuSK-abs were found elevated in all patients (ranged from 1.6 to 5.30 nmol/l [n.v. <0.05]). A diagnosis of MuSK positive MG with predominant axial involvement was made. *Case 15* was treated with prednisone and azathioprine, *case 16* received prednisone, azathioprine and plasma exchange (PE), whereas prednisone, cyclosporin, rituximab and intravenous immunoglobulin (IVIG) were administered in *case 17.* All three patients improved with residual symptoms.

Acute facial dyplegia and bulbar weakness: (Cases 18-19)

Two patients (M 47-y and F 26-y) presented with severe and isolated bulbar and respiratory muscles weakness. Both patients presented an acute onset of breathing and swallowing difficulties. They denied weakness and fatigability. Neurological examination disclosed severe facial dyplegia, dysphonia with paralysis of the soft palate, but no ptosis neither ophthalmoplegia were evident. Data about immunological, neurophysiological assessment and therapy are summarized in Table 1.

Myasthenia with sudden lower limbs weakness and falls: (Cases 20-21)

Two patients (F 14-y and F 15-y) complained of episodic, severe lower limb muscles weakness, leading to recurrent falls, often in occasion of fever. Detailed data are summarized in Table 1.

RESULTS

Patients with an unusual/atypical presentation were the 4.4% of all MG patients referred to our department. As reported in Fig. 1, 19% of these ($n = 4$) showed an isolated distal weakness at four limbs: the most commonly involved muscles were: intrinsic hand muscles and forearm extensors at the upper limbs ($n = 3$), and foot and ankle extensor muscles

Table 1
Summary of clinical course, immunological profile, instrumental data and treatment of patients

Case	Sex/Age at onset	Clinical manifestations at onset	Months/years from onset to diagnosis	Immunological profile	SFEMG	RNS	Therapy
1	M/30	DWUL	6 m	AChR-abs	Extensor indicis	Ulnar nerve	AChEI, PDN
2	F/20	DWUL	4 m	AChR-abs	Extensor indicis	Ulnar nerve	Thymectomy AChEI, PDN
3	F/40	DWUL	1 m	AChR-abs	Extensor indicis	Ulnar nerve	Thymectomy AChEI, PDN
4	M/32	Isolatet foot drop	4 m	AChR- abs	Anterior tibial	Peroneal nerve	Thymectomy AChEI, PDN
5	M/35	TWFD	6 y	AChR-abs	Triceps brachii	Peroneal nerve	AChEI, PDN
6	M/24	PWW	10 y	Neg.	Biceps brachii	Accessory nerve	Thymectomy, AChEI, PDN
7	M/43	PWW	1 y	AChR-abs	Biceps brachii	Accessory nerve	Thymectomy, AChEI, PDN
8	M/32	PWW	5 y	Neg.	Biceps brachii	Accessory nerve	Thymectomy, IVIG, AChEI
9	F/30	PWW	15 y	Neg.	Biceps brachii	Accessory nerve	Thymectomy, AChEI, PDN
10	F/38	PWW	3 y	MuSK-abs	Biceps brachii	Accessory nerve	PDN, AZA
11	F/44	PWW	8 y	Neg.	Biceps brachii	Accessory nerve	PDN, AZA
12	M/41	PWW	7 y	MuSK-abs	Biceps brachii	Accessory nerve	PDN, AZA
13	F/56	PWW	12 y	AChR-abs	Biceps brachii	Accessory nerve	PDN, AZA
14	M/30	PWW	6 y	Neg.	Biceps brachii	Accessory nerve	PDN, AZA
15	M/70	AWDH	8 m	MuSK-abs	Trapezius	Ulnar nerve/normal	AChEI, PDN, AZA
16	F/65	AWDH	10 m	MuSK-abs	Trapezius	Ulnar nerve/normal	PDN, AZA, IVIG,
17	F/43	AWDH	10 m	MuSK-abs	Trapezius	Ulnar nerve/normal	PDN, AZA, IVIG, Rituximab
18	M/47	FDBW	2 m	AChR-abs	Frontal muscle	Not done	Thymectomy, PDN, AZA
19	F/26	FDBW	6 m	AChR-abs	Frontal muscle	Not done	Thymectomy, AChEI
20	F/14	SHLLF	6 m	Neg.	Extensor digitorum	Peroneal nerve	Thymectomy, AChEI
21	F/15	SHLLF	5 m	AChR-abs	Extensor digitorum	Peroneal nerve	Thymectomy, AChEI, AZA

Legend - DWUL: Distal weakness at upper limbs; TWFD: Triceps brachii weakness and foot drop; PWW: Proximal wasting and weakness; AWDH: Axial weakness with dropped head; FDBW: Facial dyplegia and bulbar weakness; SHLLF: Sudden hyposthenia at lower limbs and falls.; SFEMG was abnormal in the reported muscles; RNS was decremental stimulating the mentioned nerves, if RNS was normal or not done, it is reported.

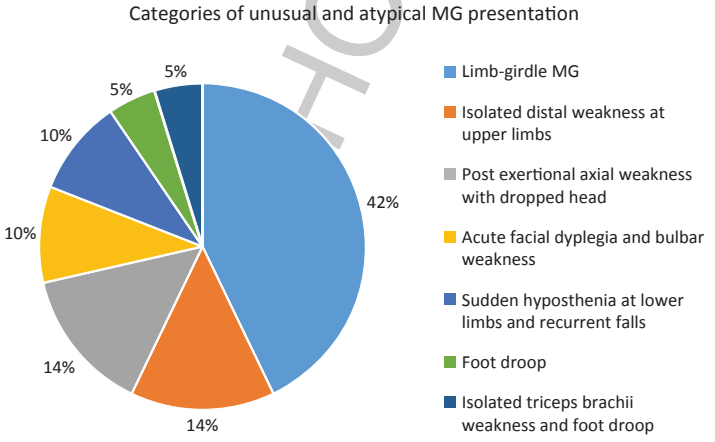

Fig. 1. Diagram of atypical and unusual clinical phenotypes percentages.

at the lower limbs ($n=1$); 4.7% ($n=1$) presented with isolated triceps brachii weakness and foot droop; 14% ($n=3$) had post exertional axial weakness with dropped head; 9.5% ($n=2$) manifested acute facial dyplegia and bulbar weakness. LGMG was the most frequent atypical phenotype (43%) ($n=9$). Furthermore, 9.5% ($n=2$) presented with sudden weakness at lower limbs with recurrent falls. Clinical course, immunological profile, instrumental data and treatment are summarized in Table 1.

DISCUSSION

Herein, we have reported the unusual and atypical presentations of MG in a large cohort of patients, mimicking other neuromuscular conditions, such

as polymiositis, limb-girdle muscular dystrophies, facio-scapulo-humeral muscular dystrophy, peripheral neurophaties. RNS, which is the most easily and commonly performed test, is not sensitive and it is often normal, especially in ocular and mild generalized MG. On the other hand, SFEMG is usually more sensitive, but it can be abnormal in muscles which appear clinically unaffected or in other neuromuscular conditions, as well [18]. In our study, we confirm the importance of SFEMG to reach a proper diagnosis of MG, even though it is much more time consuming and it needs more expertise. Furthermore, SFEMG is crucial for diagnosis when performed on affected muscles, which are not routinely tested (*cases 1, 5, 15, 16, 17*). In conclusion, we suppose that atypical and unusual presentations of MG might increase the risk to delay the diagnosis of a treatable disease; to recognize these phenotypes is mandatory. Experience from various centers all over the world, in the last 2 decades, has contributed to define atypical MG phenotypes [10–25]. Prior studies reported on asymmetric distal upper limbs weakness: Nations et al, in 1999 described a retrospective study on 236 patients, 9 of whom presenting with upper limbs weakness (hand muscles and finger extensors) [21]. Later on, in 2003, Werner et al. reported on 2 out of 84 patients with distal limb weakness at onset [11]. In our cases, 4 patients out of 508 had this similar clinical presentation at onset. As described, i.e. for *case 1*, a misdiagnosis with a neuropathy could be a frequent pitfall. Helpful features to make a MG diagnosis are: fluctuations of symptoms, no sensitive disturbances, no involvement of muscle bulk and tone and normal electrophysiological screening for peripheral nerve involvement. Clinical surveillance and follow-up is mandatory, since MG can become generalized, even after 10 years from onset, as described in *case 1*. Furthermore, comparing the time from symptoms onset to diagnosis, it lasts from 1–6 months in distal MG to 1–15 years in LGMG, confirming that the diagnosis for the latter forms is more difficult. These data are relevant since the majority of patients presenting with unusual forms belong to the LGMG group (9/508). Previous studies performed by Oh and Kuruoglu reported on 12 cases diagnosed after 20 years from onset [23]. This persistent and isolated limb-girdle involvement, sparing cranial nerves, without fluctuations, is peculiar of these forms. For these cases, RNS is really helpful to reach the proper diagnosis and to rule out the diagnosis of Lamber Eaton myasthenic syndrome, that may mimic LGMG [20]. We speculated that the high frequency of LGMG in our series, (1.7% of MG patients in 27 years of observation) could be related to a possible latitude influence. Another possible explanation is that our department is a reference center for muscular diseases and LGMG patients could have been referred to us with a suspicion of a myopathy. However, misdiagnosis with a myopathy is not only related to these forms in fact, recently, Devon et al. reported on a patient with triceps brachii and tibialis anterior muscles weakness, mimicking a primarily myopathic disorder [22], as in *case 5*. Moreover, isolated muscle weakness may involve the extensor neck muscles, configuring the "dropped head syndrome" (DHS), that imply a larger differential diagnosis [24–26]. It is known that DHS is not a rare finding in MG: fluctuations, worsening during the day with improvement after rest could be considered the "red flags" to reach a clinical diagnosis [24, 25]. In our series of MG patients with atypical and unusual manifestations, cases with other atypical aspects such as sphincter incontinence, pseudohemiplegic form, atrophic MG or MG mimicking blephatospams, already reported in the literature [27–32] were not evident. In conclusion, we confirm that the combined use of a proper neurophysiological assessment (SFEMG and RNS), antibody dosage, neostigmine test and treatment response, are essential to support the clinical suspicion. We are aware that a limit of this study is represented by the scarce number of cases in some categories, but we would test like to focus the attention on these atypical presentations, as a warning for clinicians to consider that MG may mimic other, sometimes untreatable, neuromuscular disorders.

FOOTNOTES

Carmelo Rodolico and Daniela Parisi contributed equally to this study as first authors.

CONFLICT OF INTEREST

The authors have no conflict of interest to report.

SUPPLEMENTARY MATERIAL

Supplementary videos are available in the online version of this article: http://dx.doi.org/10.3233/JND-160148

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