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ORIGINAL ARTICLE

The association of myasthenia gravis and immune-mediated myopathies

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Summary

Introduction/Aim: Myasthenia gravis (MG) is a chronic autoimmune disease of the neuromuscular junction, characterized by muscle weakness and fatigability. Idiopathic inflammatory myopathies (IIM) are an immune-mediated group of diseases characterized by progressive painful proximal weakness of the extremities. The coexistence of these two diseases is extremely rare and so far, only about fifty cases have been reported worldwide. The aim of this study was to analyze the frequency of coexistence of IIM and patients with *de novo* MG.

Material and Methods: The study is retrospective in nature and was conducted at the "Neurology Clinic", University Clinical Center of Serbia. It included 97 patients diagnosed with myasthenia gravis between January 1, 2014 and December 31, 2018.

Results: The average age of the MG patients was 54.1±18.9 years. At the time of diagnosis, 19 (19.6%) participants had at least one of the anamnestic data observed as potential indicators for the existence of immune-mediated myopathy. Finally, one patient clinically presented with generalized seropositive (anti-AchR positive) myasthenia gravis associated with the diagnosis of antisynthetase syndrome. In addition, the main characteristics of patients with combined occurrence of *de novo* MG and antisynthetase syndrome are presented.

Conclusion: Although the simultaneous occurrence of MG and IIM is a very rare phenomenon, we need to think about the possibility of combined occurrence of these two autoimmune diseases, with the aim of early recognition and adequate treatment, and thus a better prognosis of both diseases.

Keywords: Myasthenia gravis, inflammatory myopathy, antisynthetase syndrome, coexistence

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INTRODUCTION

Myasthenia gravis (MG) belongs to the group of autoimmune diseases in which antibodies directed against different postsynaptic membrane antigens lead to neuromuscular transmission impairment (1). About 80% of MG patients have antibodies directed against the nicotinic acetylcholine receptor (AchR), while 40% of initially seronegative patients have antibodies directed against muscle-specific tyrosine kinase (MuSK) (2). This rare disease is clinically characterized by variable weakness and excessive fatiguability of various skeletal muscles, especially after repeated or prolonged muscle activity, but also by an improvement in strength after rest or after the administration of anticholinesterase medication (3). In 15% of patients, the disease presents solely with eye symptoms, while in more than two-thirds of the patients, generalized weakness of facial muscles, bulbar musculature, limb muscles, and sometimes also respiratory musculature weakness are observed (4). On the other hand, idiopathic inflammatory myopathies (IIM) belong to the group of acquired, immune-mediated muscle diseases that are clinically manifested by progressive and often painful muscle weakness, predominantly of the proximal musculature of the extremities (5). A significantly elevated serum muscle enzyme creatine kinase (CK) value is a typical laboratory finding in these patients (6). Other systemic manifestations often occur in these patients, such as interstitial lung disease, arthritis, arthralgia, Raynaud's phenomenon, and various skin changes (7). Myositis-specific and myositis-associated autoantibodies can be detected in the serum taken from such patients (7).

Although both disorders are considered part of the "autoimmune spectrum of neuromuscular diseases", they are clinically, electrophysiologically, and pathophysiologically different entities. Thus, their co-occurrence is extremely rare and so far, only around 50 cases have been described worldwide, mostly in the form of case reports and small case series (7–9). However, none of the described cases belonged to this part of Europe.

Thus, the aim of our study was to analyze the frequency of signs and symptoms of IIM in a large cohort of MG patients, as well as the specificity of the clinical characteristics of these patients.

MATERIAL AND METHODS

Subjects

The study included 97 patients who were diagnosed with myasthenia gravis in the period between January 1, 2014 and December 31, 2018 at the Clinic for Neurology, University Clinical Centre of Serbia (UCCS). The diagnosis of myasthenia gravis was established in all patients based on the typical clinical presentation (in the form of weakness and pathological fatigue of various skeletal muscles), positive pharmacological test (positive neostigmine test), and/or decremental response to repetitive nerve stimulation (RNS) and/or positive findings of specific antibodies (AchR or MuSK) (8). The presence of signs and symptoms of idiopathic inflammatory myopathy in MG patients (those with elevated serum levels of creatine kinase (CK) and/or lactate dehydrogenase (LDH) and/or aspartate aminotransferase (AST) and/or alanine aminotransferase (ALT)), was assessed according to EULAR /ACR criteria (European League Against Rheumatism/American College of Rheumatology) (10). The diagnosis of IIM was further confirmed using the Web calculator for IIM of the Department of Biostatistics, Karolinska Institute, Stockholm, Sweden (9). Also, the presence of current electrophysiological criteria for the IIM diagnosis was analyzed in all MG patients (10). All patients signed informed consent to participate in the study and the study was approved by the Ethical Board of the Neurology Clinic, University Clinical Centre of Serbia and performed in compliance with the Declaration of Helsinki. In order to strengthen the certainty of the established diagnosis, data were collected at two time-points (at the moment of diagnosis and during the follow-up visit six months later). Patients who did not have a follow-up outpatient examination after six months, as well as patients who were already treated with corticosteroid and other immunosuppressive therapy (azathioprine, cyclophosphamide, cyclosporine, mycophenolate mofetil, and rituximab) at the time of MG diagnosis (non-drug naïve), were excluded from this study.

Methods

Sociodemographic and diagnostic data were collected from patients and their medical records, both at the time of initial testing and retesting. The existence of provoking or precipitating factors (stress, infection, pregnancy, surgery, and malignancy), and the presence and treatment of idiopathic hyperlipidaemia (statins and/or fibrates) were taken into consideration. Other therapeutic modalities and significant comorbidities (including other autoimmune diseases) were also noted. Disease severity was evaluated in accordance with the *Myasthenia Gravis Foundation of America* (MGFA) clinical classification at both time points. MGFA clinical classification divides MG presentations into different classes by clinical features with increasing disease severity. There are 5 main classes and several subclasses. The MGFA classifies MG forms as pure ocular (class I), mild generalized (class II), moderate generalized (class III), severe generalized (class IV), and MG requiring intubation/myasthenic crisis (class V) (11). The term "improvement" or "deterioration" of the MGFA score was defined as a change greater than or equal to one degree according to the MGFA classification. For patients in whom the change in MGFA score did not meet the criteria for change, the condition

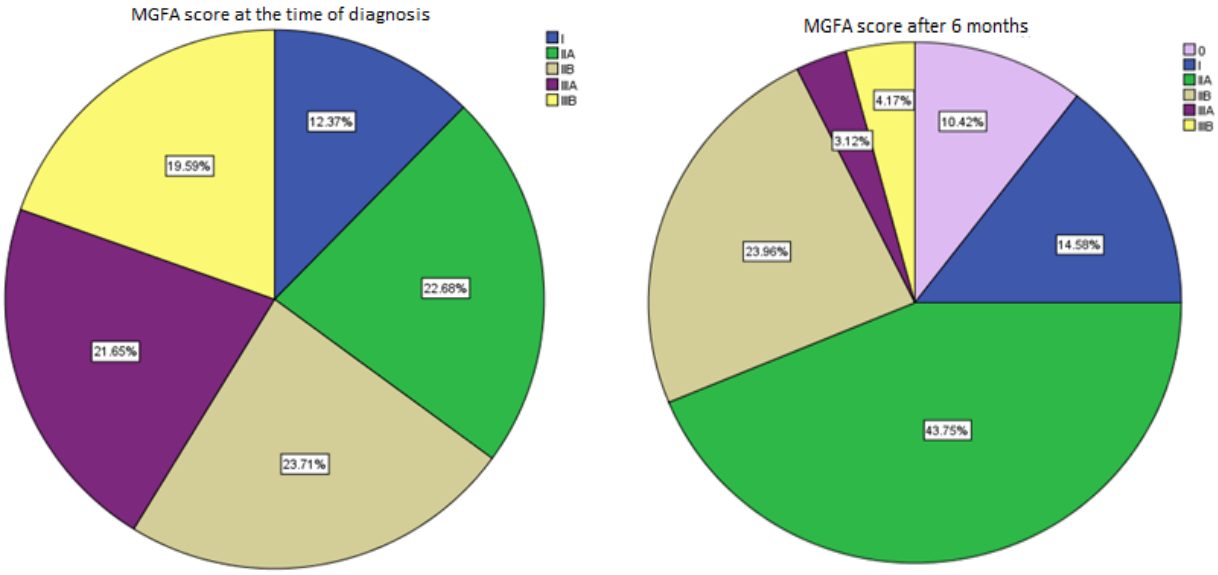


Figure 1. The frequency distribution of patients according to MGFA score at the moment of diagnosis and after 6 months (n= 97)

was considered "unchanged". Patients were thoroughly neurologically examined using quantitative scores to assess muscle weakness and fatigue: *The Quantitative Myasthenia Gravis Score* (QMGS), and the *Medical Research Council-Sum Scale* (MRC-SS) (12, 13).

The following laboratory parameters were analyzed in all subjects: complete blood count and biochemical parameters, including the values of creatine kinase (CK), lactate dehydrogenase (LDH), aspartate transaminase (AST), alanine transaminase (ALT), thyroid-stimulating hormone (TSH), free thyroxine fraction (fT4), anti-thyroglobulin antibodies (anti-Tg), thyroid peroxidase antibodies (anti-TPO), antinuclear antibodies (ANA) and antineutrophil cytoplasmic antibodies (ANCA). Patients were also tested for the presence of myositis-specific antibodies (Jo-1 (histidyl-transfer RNA synthetase), SRP (signal recognition particle), Synthetase, Ku, Mi-2 (NuRD subunit), and Ro 52 (anti-SSA 52 - anti–Sjögren's-syndrome-related antigen A) antibodies), as well as anti-AchR and anti-MuSK antibodies specific for MG. In addition, as part of the diagnostic protocol, RNS tests, electromyographic (EMG) evaluation, and computed tomography of the chest (chest CT examination) were performed in all patients.

Statistical analysis

Nominal and numeric data were tested using descriptive statistics methods. The assumption of data normality was tested with the Shapiro-Wilks test. The IBM SPSS program (The Statistical Package for the Social Sciences, version: SPSS v22) was used for statistical analyses.

RESULTS

A total number of 97 patients with confirmed diagnosis of *de novo* MG were included in our study. The average age of our patients was 54.11±18.98 years, of which 51 (52.6%) were females. The main socioepidemiological features of our MG patients are presented in **Table 1**.

Table 1. Main socioepidemiological characteristics of our patients with MG (n=97)

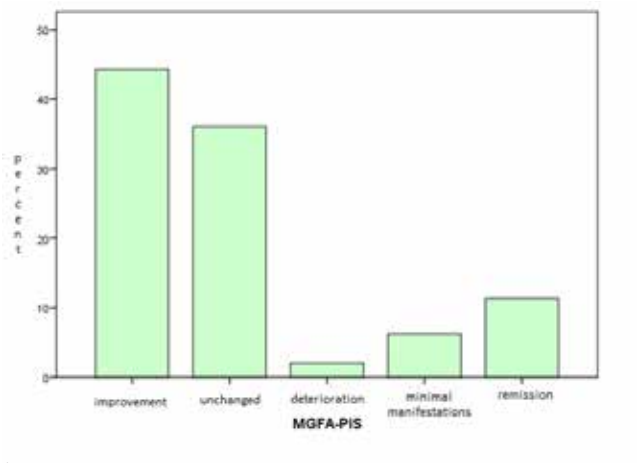
Patient characteristics	
Males (n (%))	46 (47.4%)
Females (n (%))	51 (42.6%)
Age of onset (year, mean ± SD)	54.11±18.98
Disease duration > 1 year (n (%))	54 (56.7%)

The ocular form of MG was noted in 11.3% of patients, while the remaining part of the cohort had the generalized form of the disease. The stages of MG according to the MGFA classification are shown in **Figure 1**.

In 40 (41.2%) patients, there was no change in the MGFA score during the diagnostic follow-up. The distribution of disease outcomes assessed according to MGFA Post-intervention Status (MGFA-PIS) is shown in **Figure 2**.

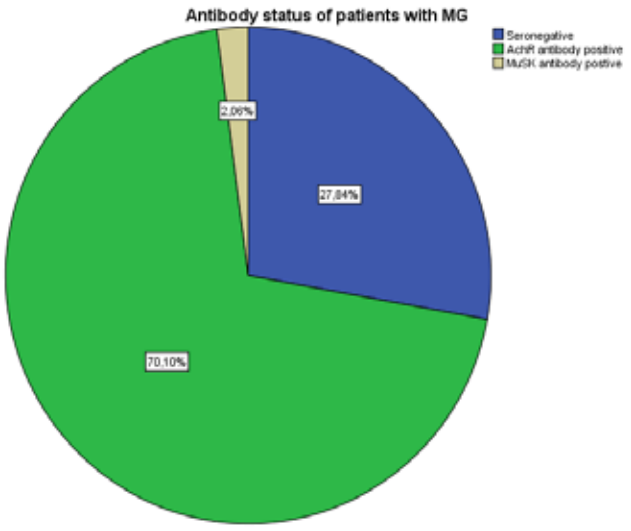
No patient had a primary manifestation of myasthenia gravis in the form of a myasthenic crisis. **Table 2** and **Table 3** show the clinical presentation and neurological status of patients with myasthenia gravis.

As part of the diagnostic workup, the presence of AchR antibodies was observed in 68 patients (70.1%), while MuSK antibodies were detected in two patients (2.1%). Tests for the presence of other rare antibodies (seronegative MG, SN-MG) in MG were not performed. The antibody status of our patients with MG is shown in **Figure 3**.



*MGFA-PIS- MGFA Post-intervention Status

Figure 2. The frequency distribution of patients according to the change in MGFA score according to MGFA-PIS (n= 97)



AchR - acetylcholine receptor; MuSK - muscle-specific tyrosine kinase

Figure 3. Antibody status of our patients with myasthenia gravis (MG).

Table 4 shows the main laboratory and electrophysiological data of our MG patients.

Of all analyzed MG patients, at the time of diagnosis, 19 (19.6%) patients had at least one anamnestic data which was considered a potential indicator for the existence of immune-mediated myopathy (pain in muscles and joints, joint swelling, different skin changes, fever, and other autoimmune diseases). One patient was found to have significantly elevated serum creatine kinase value (value typically observed in IIM) at the time of diagnosis of MG. After entering the available data into the IIM calculator, the patient was classified as a case of "probable idiopathic inflammatory myopathy" with an estimated probability of 62-99% (min-max).

In the remaining 96 patients in whom the diagnosis of myasthenia gravis was confirmed, the existence of any subtype of idiopathic inflammatory myopathies was not noted.

All patients were treated with adequate symptomatic and/or immunosuppressive therapy. Therapeutic modalities used to treat our patients with MG are shown in **Table 5**. Most of our patients (97%) were treated with acetylcholinesterase inhibitors, 92.8% with corticosteroid therapy, 45.4% with azathioprine, and 8.2% with cyclosporine A. The second-line therapy was also applied in a smaller percentage of patients – therapeutic plasma

Table 2. Main clinical characteristics of MG patients (n= 97)

History	**First examination** (n (%))	**Examination after 6 months** (n (%))
Drooping eyelids*	77 (79.4%)	54 (55.7%)
Double image*	26 (26.8%)	5 (5.2%)
Difficulty speaking*	51 (52.6%)	18 (18.6%)
Difficulty chewing*	57 (58.8%)	33 (34%)
Difficulty swallowing*	39 (40.2%)	20 (20.6%)
Neck weakness*	44 (45.4%)	18 (18.6%)
Heavy breathing*	2 (2.1%)	1 (1%)
Upper extremity pain	7 (7.2%)	7 (7.2%)
Arm weakness	49 (50.5%)	34 (35.1%)
Proximal arm weakness	45 (46.4%)	29 (29.9%)
Distal arm weakness	32 (33%)	19 (19.6%)
Lower extremity pain	9 (9.3%)	12 (12.2%)
Leg weakness	41 (42.3%)	32 (33%)
Proximal leg weakness	39 (40.2%)	30 (30.9%)
Distal leg weakness	28 (28.9%)	14 (14.4%)
Significant fatigue*	10 (10.3%)	10 (10.3%)
Autoimmune diseases	8 (8.2%)	8 (8.2%)
Other comorbidities	51 (52.6%)	46 (47.4%)
Hyperlipidemia	8 (8.2%)	3 (3.1%)

*Symptoms mainly characteristic of myasthenia gravis;

Table 3. Main neurological findings of our patients with MG (n= 97)

Clinical findings	First examination (n (%))	Examination after six months (n (%))
Ptosis	77 (79.4%)	53 (54.6%)
Double vision	26 (26.8%)	5 (5.2%)
Masticatory muscles weakness	57 (58.8%)	34 (35.1%)
Masticatory muscles fatigue	57 (58.8%)	34 (35.1%)
Mimic muscles weakness	67 (69.1%)	48 (49.5%)
Mimic muscles fatigue	67 (69.1%)	47 (48.5%)
Soft palate weakness	45 (46.4%)	19 (19.6%)
Soft palate fatigue	44 (45.4%)	19 (19.6%)
Tongue weakness	38 (39.2%)	14 (14.4%)
Tongue fatigue	38 (39.2%)	14 (14.4%)
Neck anteflexion weakness	36 (37.1%)	18 (18.6%)
Neck anteflexion fatigue	35 (36.1%)	18 (18.6%)
Neck retroflexion weakness	35 (36.1%)	11 (11.3%)
Neck retroflexion fatigue	35 (36.1%)	11 (11.3%)
Arm weakness	49 (50.5%)	35 (36.1%)
Proximal arm weakness	45 (46.4%)	29 (29.9%)
Distal arm weakness	32 (33%)	18 (18.6%)
Arm fatigue	50 (51.5%)	39 (40.2%)
Proximal arm weakness	47 (48.5%)	33 (34%)
Distal arm weakness	32 (33%)	21 (21.6%)
Leg weakness	41 (42.3%)	32 (33%)
Proximal leg weakness	39 (40.2%)	30 (30.9%)
Distal leg weakness	28 (28.9%)	14 (14.4%)
Leg fatigue	41 (42.3%)	34 (35.1%)
Proximal leg fatigue	39 (40.2%)	33 (34%)
Distal leg fatigue	28 (28.9%)	17 (17.5%)

exchange (PLEx) was applied in 18 (18.6%) patients and intravenous immunoglobulins (IVIg) in two (2.1%) patients. As mentioned above, no patient included in this study was treated with immunosuppressive or immunomodulatory therapy before the onset of MG.

A brief presentation of the case

A 71-year-old patient, previously treated solely for the diagnosis of essential arterial hypertension, clinically presented with generalized seropositive (anti-AchR positive) myasthenia gravis. The spectrum of neuromuscular complaints in our patient was comprised of predominantly proximal muscle weakness of the upper and lower extremities and a slight difficulty swallowing, which was accompanied by bilateral semi-ptosis, with clear fatigability (MGFA IIIA). Laboratory analyses showed the presence of elevated creatine kinase (8,071 U/l), serum potassium levels (5.5 mmol/l) and LDH (590 U/L), thrombocytopenia (68 10^9/L), and increased erythrocyte sedimentation (24 mm/h). After RNS conduction, the patient fulfilled the electrophysiological criteria for the presence of MG (consistent decrement of 13%). As part of the

Table 4. Main laboratory and electrophysiological parameters of MG patients at the time of diagnosis and the follow-up examination (n= 97)

Laboratory findings	First examination (n (%))	Examination after 6 months (n (%))
Decreased RBC	6 (6.2%)	-
Elevated WBC	23 (23.7%)	-
Decreased WBC	1 (1%)	-
Decreased PLT	3 (3.1%)	-
Decreased HGB	4 (4.1%)	-
Elevated ESR	26 (26.8%)	-
Elevated CRP	14 (14.4%)	-
Elevated fibrinogen	1 (1%)	-
Elevated K^+	1 (1%)	-
Elevated CK	3 (3.1%)	-
Elevated LDH	9 (9.1%)	-
Elevated AST	2 (2.1%)	0 (0%)
Elevated ALT	10 (10.3%)	2 (2.1%)
Elevated GGT	3 (3.1%)	4 (4.1%)
Elevated fT4	5 (5.2%)	0 (0%)
Decreased fT4	17 (17.5%)	0 (0%)
Elevated TSH	5 (5.2%)	2 (2.1%)
Elevated Anti-TPO antibodies	7 (7.2%)	2 (2.1%)
Elevated anti-Tg antibodies	7 (7.2%)	1 (1%)
Elevated ANA	9 (9.3%)	4 (4.1%)
Elevated ANCA	2 (2.1%)	1 (1%)
Positive myositis profile	1 (1%)	1 (1%)
Positive Anti-AchR antibodies	68 (70.1%)	-
Positive Anti-MuSK antibodies	2 (2.1%)	-
RNS test	62 (63.9%)	-

AchR- acetylcholine receptor; ALT- alanine transaminase; AST- aspartate transferase; ANA- antinuclear antibodies; ANCA- antineutrophil cytoplasmic antibodies; CK- creatine kinase; CRP- c-reactive protein; ESR- erythrocyte sedimentation rate; fT4- free thyroxine; GGT- gamma-glutamyl transferase; HGB- haemoglobin; K^+-kalium; LDH- lactate dehydrogenase; MuSK- muscle-specific tyrosine kinase; PLT- platelet count; RBC – red blood cell count; RNS- repetitive nerve stimulation; TSH- thyroid stimulating hormone; TPO- thyroid peroxidase; Tg- thyroglobulin; WBC – white blood cell count.

performed immunoserological analyses, the presence of anti-Jo-1 antibodies was observed, and the patient was diagnosed with the co-occurrence of myasthenia gravis and antisynthetase syndrome (symmetrical proximal and painful weakness of the arms and legs, elevated serum CK and LDH values, elevated erythrocyte sedimentation rate (ESR), positive myositis panel). The patient was treated with anticholinesterase and corticosteroid therapy according to therapeutic protocols for both MG and antisynthetase syndrome, respectively. The control neurological examination verified the improvement of

Table 5. Therapeutic modalities used to treat our patients with MG

Symptomatic and immunosuppressive therapy	Percent of treated cases (%)
First-Line treatment options	
Acetylcholinesterase inhibitors	97%
Steroids	92.8%
Azathioprine	45.4%
Cyclosporine A	8.2%
Second-Line treatment options	
Therapeutic plasma exchange	18.6%
Intravenous immunoglobulins	2.1%

the neurological findings (MGFA IIA), with persistence of mild but not painful weakness and fatigue of the mimic muscles, as well as the normalization of the laboratory biochemical parameters.

DISCUSSION

Similar clinical presentations of both diseases are frequently the reason for a diagnostic delay, which is important from the therapeutic point of view, bearing in mind the frequent need for more aggressive immunosuppressive therapy in these patients (7). In the broadest sense, MG and IIM can be considered part of the autoimmune neuromuscular disorder spectrum. The clinical presentation of both entities can be similar, with a range of possible overlapping symptoms and signs, which represents a significant diagnostic challenge. Studies have shown that patients with IIM, unlike patients with MG, usually do not have ocular symptoms, such as diplopia, ptosis, and/or ophthalmoparesis. However, bulbar involvement is often found in both diseases (8). Suspicion of the possible association of IIM with MG can also be raised when there is non-fatigable weakness or a continuous increase in CK before starting immunosuppressive therapy (7). Moreover, the possibility that prescribing immunosuppressive or immunomodulatory therapy in patients diagnosed with one of these two diseases could "mask" or modify the clinical manifestation of the other disease should not be ignored. The co-occurrence of these two chronic disorders has been found only in a small number of patients so far (14). Namely, the incidence of myasthenia gravis ranges from 1.7 to 30 per million individuals per year, while the average incidence of inflammatory myopathy is 5 per million individuals (15,16). Therefore, it is not surprising that the concomitant appearance of myasthenia gravis and inflammatory myopathies is extremely rare. In the most extensive publications to date, Garibaldi et al. and Huang et al. describe no more than 50 cases of coexistence of these entities (7, 8). In the largest observed Italian cohort of 441 MG patients, 2.9% of patients with MG and IIM were detected, of which 10 patients were diagnosed with both entities simultaneously. Although in a smaller cohort of patients, the frequency of *de novo* coexistence was confirmed in 1.03% of patients in our study, which does not differ significantly from the data in the available literature. Furthermore, our cohort of MG patients did not differ from the aforementioned cohorts of MG patients in terms of their clinical and sociodemographic characteristics (7).

When the frequency of MG and individual forms of inflammatory myopathies were analyzed, it was observed that individual cases of overlap of MG with polymyositis (now anti-synthase syndrome), inclusion body myositis (IBM), autoimmune necrotizing myopathy, and dermatomyositis have been described so far (8, 17–19). Compared to the findings in our patient with a positive titer of anti-Jo-1 antibodies, only three case reports so far have reported a case of an antisynthetase syndrome associated with MG, of which only one had positive anti-Jo-1 antibodies. In the remaining two patients, the presence of anti-PL7 (anti-threonyl-tRNA synthetase) and anti-Ej (anti-glycyl-tRNA synthetase) antibodies was detected (20, 21). According to the available literature at the time of writing this paper, our results represent the first data on the coexistence of IIM and MG in the area of Southeastern Europe.

Our patient met all currently valid criteria for the diagnosis of MG. The predominance of pain and muscle weakness, and uncharacteristically slow response to therapy, were the reasons for further examination, which was also the most common reason for re-examining the diagnosis in the previous literature. Inflammatory myopathy was suspected based on significantly elevated serum CK values and then confirmed by a combination of clinical presentation and positive antibody findings. The finding of anti-Jo-1 antibodies in patients with idiopathic inflammatory myopathies is rare and ranges from 1-20% of all patients (22). On the other hand, CK elevation is considered the main laboratory indicator of myocyte damage, along with an increase in LDH and potassium, but it can be associated with other pathological conditions and is still a completely asymptomatic laboratory-isolated entity. In our patient, there were no associated symptoms and signs of other manifestations otherwise described in the literature (23, 24) which could be explained by the short duration of the disease in this case.

Certain authors report an association of the coexistence of IIM and MG with an increased frequency of malignancy, which was also not the case with our patient (25). Uchio et al. showed that the prevalence of thymoma in patients with IIM and MG was as high as 70%, compared to 10% in patients with MG (9). Some authors propose an explanation according to which the occurrence of MG with IIM is not a coincidence, but the association with thymoma may indicate the presence of complex pathogenetic mechanisms between these two autoimmune disorders. However, using chest CT examination we excluded any abnormalities of the thymus and signs of intestinal lung disease in our patient.

Spanish authors have described an MG patient with

similar sociodemographic and treatment characteristics compared to the data of our patient, but his anti-synthetase syndrome was complicated with the finding of alveolitis and other pulmonary manifestations (26). After the diagnosis of IIM, the patient was treated with rituximab, while in our patient, the treatment with classic immunosuppressants proved to be sufficient to suppress the inflammatory process, regulate laboratory indicators of myositis, and reduce the clinical picture.

CONCLUSION

The concomitant occurrence of MG and IIM is rarely observed. We have described the case of co-occurrence of antisynthetase syndrome and myasthenia gravis in our patient, underlining that MG patients with atypical clinical and diagnostic features should be screened for the presence of IIM. Thus, neurologists should think about the possibility of the combined occurrence of these two rare but treatable diseases, with the aim of early recognition and more adequate treatment, and therefore a better prognosis for such patients.

Study limitations

The shortcomings of our study are the small number of included patients and the short follow-up period. Data on further rheumatological, pulmonological, or dermatological treatment were not available at the moment of study conduction. Also, a major shortcoming of the study is the fact that a definitive diagnosis of myositis was not established because a muscle biopsy was not performed. To obtain solid evidence that explains the reasons for the co-occurrence of inflammatory myopathies with myasthenia gravis, basic research is needed for the definitive exploration of the autoimmune basis of both conditions, as well as multicentric research, which could solve the problem of a small number of patients in most of the previous studies.

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POVEZANOST MIJASTENIJE GRAVIS I IMUNSKI-POSREDOVANIH MIOPATIJA

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Sažetak

Uvod/Cilj rada: Mijastenija gravis (MG) je hronično autoimuno oboljenje neuromišićne spojnice, koje se karakteriše slabošću dominantno proksimalne muskulature svih ekstremiteta, uz zamorljivost. Inflamatorne miopatije (IM) su imunski posredovana, heterogena grupa oboljenja koje karakteriše postojanje progresivne bolne slabosti proksimalne muskulature ekstremiteta. Iako se obe bolesti smatraju delom autoimunog spektra neuromišićnih bolesti, one su klinički, elektrofiziološki, ali i patofiziološki različiti entiteti. Koegzistencija ova dva oboljenja je izuzetno retka i do sada je širom sveta zabeleženo samo pedesetak slučajeva. Cilj ovog istraživanja bila je analiza učestalosti postojanja koegzistencije IM i pacijenata sa de novo MG.

Metode: Studija je retrospektivnog karaktera i sprovedena je na Klinici za neurologiju Univerzitetskog kliničkog centra Srbije. U studiju je bilo uključeno 97 pacijenata kod kojih je dijagnoza mijastenije gravis postavljena u periodu od 1. januara 2014. godine do 31. decembra 2018. godine.

Rezultati: Prosečna starost navedenih ispitanika je iznosila 54,1±18,9 godina. Od svih analiziranih pacijenata, u trenutku postavljanja dijagnoze njih 19 (19,6%) je imalo barem jedan od anamnestičkih podataka posmatranih kao potencijalni indikator za postojanje imunski posredovane miopatije. Finalno, kod jednog pacijenta je klinički prezentovano postojanje generalizovane seropozitivne (*anti-AchR* pozitivne) mijastenije gravis udruženo sa postojanjem dijagnoze antisintentaza sindroma. U daljem teksu su prikazane ključne karakteristike pacijenta sa udruženom pojavom MG i antisintentaza sindroma.

Zaključak: Premda je istovremena pojava MG i IM veoma redak fenomen, neophodno je imati na umu mogućnost udruženog javljanja ova dva autoimuna oboljenja, sa ciljem što ranijeg prepoznavanja i adekvatnijeg lečenja, a samim tim i bolje prognoze obe bolesti.

Ključne reči: Mijastenija gravis, inflamatorna miopatija, antisintetaza sindrom, koegzistencija

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