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*Coping with miastenia gravis*

**Sprostać miasteni gravis**

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Key words: myasthenia gravis, myasthenia crisis, thymoma, treatment

Słowa kluczowe: miastenia, przełom miasteniczny, grasiczak, leczenie

Myasthenia gravis also known as Erb-Goldflam disease, is a chronic autoimmune disease characterized by rapid fatigability of striated muscles [1, 2] Tommas Willis, a British physician, first described myasthenia gravis in 1672. Name of the disease was introduced a little later by the fusion of the Greek words "muscle" and "weakness" and adding the Latin adjective "heavy" (gravis) [1].

Myasthenia gravis is more common in women than in men [2]. This disease can be divided into two types, paraneoplastic and non-paraneoplastic. Paraneoplastic type is more common in women of age 40 years or younger, and may be associated with thymic hyperplasia. The paraneoplastic type of myasthenia gravis is more likely to have AChR antibodies or anti-MuSK, and increased chances of a thymoma. Features of a non-paraneoplastic form are different because the patients are often men older than 40 years of age, who have normal histological structure of the thymus or have thymic atrophy. The presence of antibodies AChR is also less likely in this type than the paraneoplastic type [2, 3]. Patients with late-onset of symptoms represent a group of more severe myasthenia gravis and have a greater risk of myasthenic crisis, which is not clearly understood [3].

Symptoms of the disease vary significantly between individuals. We can however, divide the severity of symptoms into classes. Class I: ptosis, diplopia and weakness of the eye muscles. Classes II-V depends on the severity of the disease, which includes difficulty swallowing, drooping of the jaw, difficulty in chewing, dysarthria, postural instability, walking, standing and as well as difficulty in breathing in advanced stage of myasthenia gravis, which is life threatening and requires immediate intervention [2, 3]. Patients notice the intensification of symptoms after muscle use to conduct day-to-day activities and resolution of them after rest. Exacerbation

of symptoms may occur as a result of infection, stress, surgery or medication. The symptoms of myasthenia gravis tend to be more severe in first 2 years of the onset of symptoms [3]. The remission of the disease is usually short lasting.

During the course of the illness, two life-threatening conditions may occur – myasthenic and cholinergic crisis. The first one affects 70 percent of patients in the first two years of the disease. The duration of the crisis varies; the average is 2 weeks [4]. Respiratory infections are one of the key factors predisposing a patient to myasthenic crisis and are responsible for about 50% of the crises. Abrupt discontinuation of treatment also plays an important role in inducing the condition [4]. Other factors affecting the development of myasthenic crisis are: major surgery, emotional stress or insufficiently and long term immunosuppressive therapy [5]. For patients with myasthenia gravis, it is crucial to treat bacterial infections as quickly and decisively as possible, even before culture results are known. Majority of antibiotics, e.g. third generation cephalosporins, do not increase the risk of the myasthenic crisis, however aminoglycosides may have a negative effect on neuromuscular conductivity and generally should be avoided [5]. The most dangerous symptom of myasthenic crisis is fatigability of breathing muscles, which makes respiratory therapy the most important part of the treatment [4, 5]. Often times the warning signs of impending crisis are not noticeable by the patients and may in fact develop within one day. Alarming symptoms include shortness of breath, blurred speech, difficulty swallowing, neck muscle weakness, orthopnea, and intense sweating. Paleness and cyanosis suggest life-threatening lowering of oxygen and elevated levels of carbon dioxide in the blood [5].

Progressive muscle weakness suggesting myasthenic crisis may occur due to a paradoxical condition – cholinergic crisis. Cholinergic crisis is induced by overdose of AChE inhibitors [5, 6] and is accompanied by cholinergic symptoms like blurred vision, miosis, excessive salivation, increased sweating, vomiting, diarrhea, and increased heart rate [6]. Additionally, difficulty in breathing, which is caused by large amounts of mucus in the bronchi leads to anxiety or even panic. Differential diagnosis between those two symptomatically similar but pathophysiologically different conditions is based on the effects of administration 10 mg of edrophonium chloride. If symptoms worsen, that indicates the occurrence of crisis associated with overdose of AChE inhibitors and not myasthenic crisis. In this situation, procedure includes decreasing the dose of AChE inhibitors and symptomatic treatment, and even respiratory therapy as needed, until the crisis is resolved [6].

The pathophysiology of myasthenia gravis is nothing short of complex as a substantial amount of indirect evidence suggests that the disorder of neuromuscular transmission may be associated with activation of the complement by the NMJ (neuromuscular junction). Factors protecting the cell surface before activation of the MAC are decay-accelerating factor (DAF), a protein cofactor membrane (MCP) and membrane inhibitor of reactive lysis (MIRL). Experiments demonstrate that DAF deficiency leads to a more elevated symptom of muscle fatigue [1]. In the development of acute myasthenic crises it is believed that the anti-AChR antibody [Ab anti-AChR] blocks the AChR which leads to myasthenic crises sometimes seen in pa-

tients being treated with pharmacotherapy [1]. The antibodies predominantly seen in myasthenia gravis, works by three mechanisms: bind and activate the complement, performs molecular modulation AChR receptor and blocks functions of Ach [1, 2]. Moreover, studies have shown a direct effect of CD4 + cells in the development of myasthenia gravis. Thus, also confirmed the role of HLA II in the pathogenesis of myasthenia gravis. Patients are likely to contain the gene products of the HLA class A1 and B8 and molecules DR3/DW3 class II molecule and DQ alleles of certain products.<sup>[1]</sup> Presence of HLA-DR3 and B8 is related to early myasthenia gravis onset with hyperplasia of thymus. Gene AIRE and CHRNA1 gene might be crucial in development of the disease [2, 3]. It was observed that pregnancy also influences the course of myasthenia gravis. Estrogen is considered to play a great role in progress of the disease due to increase in the number of antibodies and sensitive humoral immunity [2].

It is estimated that approximately 10-15% of patients with myasthenia gravis have a thymoma which can appear at any age [3]. More than 80% of patients with early onset of myasthenia gravis with antibodies against AChR have shown significant lymphocytic proliferation [3]. About 20% of myasthenia gravis patients with seronegative AChR antibodies, develop antibodies against muscle specific tyrosine-kinase (anti-MuSK). Sometimes myasthenia gravis patients lacking either of the above mentioned antibodies can have other antibodies found in patients serum that affect the neuromuscular junction, for example antibodies against titin and ryanodine [1, 2].

If there is a suspicion of myasthenia gravis in a patient due to complaints like muscular weakness and fatigue, then the first course of action is to take a blood test to detect anti-AChR antibody. Levels over 0,4 nmol/l is positive for myasthenia. In an instance where the blood test is negative and symptoms are apparent for myasthenia, then it is important to check for anti-MuSK antibodies (normal range <0,4 nmol/l) to verify the diagnosis of myasthenia gravis. Another assessment used for diagnosis of myasthenia gravis, one can use neostigmine or edrophonium test also known as the tensilon test. These drugs provide more acetylcholine to the neuromuscular junction which improves muscle tension and reduces myasthenia gravis symptoms [8, 9]. Repetitive stimulation of peripheral nerves at low frequencies (2 to 3 Hz) is also an objective diagnostic method used in myasthenia gravis. The peripheral nerve stimulation confirms whether or not there is a defect in neuromuscular junction [9]. It is also quintessential to monitor thymus activity in this disease so a CT scan is used for thymus imaging. Unfortunately, sometimes it is unclear and difficult to tell how much the thymus has enlarged and if thymus is active or if there is a thymoma [7]. Another technique used to monitor thymus changes is chemical shift MRI, which is useful in detecting fatty elements; therefore, the best way to identify thymus in young and adult patients with hyperplasia of the gland [7]. If a patient has a malignant tumor or presents with high risk of local recurrence, then invasive surgical techniques should be avoided [7].

There are many diseases that can be mistaken with myasthenia gravis, e.g. botulism, tick paralysis, snake bite with envenomation, oculopharyngeal dystrophy,

inclusion body myositis, Guillian Barre syndrom, heavy metal poisoning, motor neuron disorder, and multiple sclerosis. It is crucial to take into consideration possible differential diagnoses after a thorough analysis of anamnesis, physical examination, laboratory and electromyography tests [8].

Even though myasthenia gravis is a disease known to mankind for over three hundred years, it is still only partly understood and, unfortunately, not always diagnosed and successfully treated in every patient. There is an undisputed need to carry out studies looking for its pathophysiology, inducing and exacerbating factors as well as coexisting diseases and best treatment approaches.

### **METHODS AND MATERIAL**

We conducted a non-invasive, retrospective study of 55 medical histories of patients referred to the Department of Neurology Independent Public Clinical Hospital No. 4, Medical University of Lublin from January 2010 to October 2013 due to exacerbation of myasthenia gravis. Our patient population consisted of 41 people. Ten of them were admitted to the Department twice, other two – three times during the study period. Analysis included each-patients individual history, called “patients” below, and every hospitalization of each patient, called “cases” below. We analyzed sex, age of onset of MG symptoms, duration of myasthenia gravis, admittance symptoms, duration of hospital stay, mediastinal tumor examination, presence of myasthenia crisis, mortality, myasthenia gravis therapy, co-existence of other chronic somatic diseases and psychiatric diseases.

### **RESULTS**

Out of our 41 patients, 25 were women (60,98%) and 16 were men (39,02%). During the course of the study, 4 women and 6 men were admitted twice, 2 women were admitted thrice. 10 patients histories were lacking the information about the onset of myasthenia gravis. Out of the 31 patients, almost two third [20 patients – 15 women and 5 men] presented with the onset of myasthenia gravis before the age of 50.

Average age of each case was 50 years old (range 20-78). Main symptoms that caused patients to come to the Department included problems with eyesight, talking, swallowing, weakness of the limbs and breathing aggravation [Table 1]. Duration of hospital stay ranged from 2 to 293 days (median – 8 days).

During the study period, thirty abdominal computed tomography imaging were conducted. Eleven confirmed mediastinal tumor and eighteen were negative for mediastinal tumor presence, and one was ambiguous. Out of 41 patients, 14 were diagnosed with a mediastinal tumor at some point in their life and out of those 6 patients underwent thymectomy.

In our analysis, 4 females and 1 male were diagnosed with myasthenic crisis. Three women died in the hospital due to myasthenic crisis, the fourth one was hospitalized two more times for the same reason (myasthenic crisis) during the study period.

In 23 cases (41,82%), patients were treated with mestinon (Pyridostigmini bromidum) only, which was enough to improve patients' health. In eight cases, encor-ton was added to mestinon. Thirteen cases required IVIG, twelve – plasmapheresis, five - both procedures during the same hospitalisation [Table 2]. Four cases were only observed without any specific pharmacological treatment.

The average dose of mestinon home prescribed was 300 mg per day (5 tablets), ranged from 120 mg to 600 mg per day. In one case no mestinon was prescribed home - 77 year old patient with 6 year long history of myasthenia, negative AChR antibodies level (<0,4 nmol/l) and ptosis was the only symptom of the disease.

The most common coexisting chronic disease was hypertension in 21 patients (51,22%) followed by osteoarticular diseases in 14 patients (34,15%), thyroid diseases in 12 patients (29,27%) and other cardiovascular diseases in 9 patients (21,95%) [Table 3]. There were 7 patients with diabetes and 5 patients with pulmonary diseases. Five patients had a diagnosed psychiatric disease: schizophrenia (2 patients), depression (2 patients) and one patient with anxiety disorder.

## DISCUSSION

Lastly, there are a few different types of treatment options available for patients suffering from myasthenia gravis. The treatment varies from patient to patient depending on how mild or severe the symptoms of myasthenia gravis are. Most commonly used treatments include acetylcholinesterase inhibitors, corticosteroids, thymectomy, azathioprine, cyclophosphamide, plasmapheresis and symptomatic management of other symptoms.

Acetylcholinesterase inhibitors are typically the first line of treatment for MG. They increase the amount of acetylcholine available for neuromuscular transmission by impairing the breakdown of acetylcholine at the synaptic cleft. The first use of this group of drugs on 11 April 1934 by Dr M.B Walker represented a breakthrough in the pharmacological treatment of myasthenia gravis [9]. Pyridostigmine (Mestinon) is the main acetylcholinesterase inhibitor, which provides only symptomatic treatment, but in some patients it is the only therapy needed. Drug effects may be noticed in 2-3 hours after taking a dose and beneficial effects last about 4 hours [10]. Maximum daily dose of pyridostigmine is 360 mg. Most patients require around 180 mg/day [11]. The side effects of the cholinesterase inhibitors are relatively mild and related to the high concentrations of ACh and therefore, present with muscarinic effects like cramps, diarrhea, respiratory and gastrointestinal secretions, and bradycardia. The nicotinic effects can be muscle weakness and fasciculation. The muscarinic side effects can be reduced by the addition of muscarinic antagonists, such as propantheline (15 mg usually 15–30 minutes prior to taking pyridostigmine). For fixed deficits such as ptosis and diplopia, ophthalmic surgical intervention may be helpful [11]. Pyridostigmine is usually given orally but it can be also given intravenously, especially for hospitalized patients with myasthenic crisis. To this group of patients we can also prescribe edrophonium (Tensilon), ambenonium (Mytelase), neostigmine (Polstigminum), which can be given with Mestinon for severe bulbar

symptoms. Neostigmine injection should be given with atropine to reduce muscarinic effects.

50% of cortical thymoma patients develop myasthenia gravis (MG), while 15% of MG patients have thymomas [11]. Thymectomy is the classic long-term treatment in such cases. There are two histological abnormalities of the thymus that occur in patients with MG: thymoma and thymic carcinoma. In thymic carcinoma, surgery is generally recommended due to the risk of extension of the tumor into adjacent structures in the mediastinum [12]. The main aim of surgery is to achieve complete remission and reduction of exposure to immunosuppressive drugs. A number of studies have shown that the earlier the thymectomy is performed during the course of the disease, the more effective it is. There is mounting evidence that it is most beneficial during the first 2 years of the disease [12]. Thymectomy can be performed using several surgical approaches. Optimal surgical procedure depends on general health status, physical built and the place of a thymoma. The most invasive technique is full and partial sternotomy. The recovery period depends on the specific surgical approach used and the type of work or activity the patient performs regularly but it is usually recovery is about 3 months. The recovery time period is significantly shorter in less invasive surgical approaches and may be as little as 1-2 weeks with the transcervical thymectomy. Depending on the histological type and extent of spread, in some cases postoperative radiotherapy and/or chemotherapy is needed. In 30% of patients pharmacological treatment results in remission, whereas others might require continuous pharmacotherapy because of persistent clinical symptoms of varying severity due to the presence of ectopic thymic tissue [10].

Corticosteroids are currently the basis for the immune-directed treatment for patients with myasthenia gravis. Their major effect is anti-inflammatory by reducing expression of inflammatory cytokine, adhesion molecules and reducing trafficking of inflammatory cells. High doses may also induce apoptosis in immune cells. These medications induce remission in at least 50% of the patients [12]. Unfortunately there are many side effects so long term treatment is not advisable. Some of the side effects of corticosteroids include but are not limited to, osteoporosis, diabetes mellitus, susceptibility to infections, acne, dyspepsia, and double vision. The worsening of myasthenia symptoms associated with treatment occurs 7 to 14 days after initiation of high dose corticosteroid treatment and usually lasts less than 1 week. It's proven that gradually increasing the dose of steroids over 1 to 2 months reduces the risk of the early worsening of the disease [12]. The main objective is to carry out the treatment in a way to avoid as many side effects as possible, which can be achieved by prophylactic treatment with vitamin D and calcium for bone protection, and proton pump inhibitors for gastric protection. Prednisolone is indicated for patients with ocular myasthenia gravis with double vision, patients with severe symptoms before thymectomy to improve their condition prior to the treatment and after thymectomy if improvement is not sufficient. Prednisone therapy must be consistent, and the treatment period must not be shorter than 6 months [13]. Prednisone dose and duration of treatment depends on the patient. Some authors recommend initiating therapy with a low dose and slowly increasing it, which helps to protect the patient from

worsening symptoms of myasthenia that sometimes occurs at the beginning of steroid therapy. Starting dose of 10 mg, daily or on alternate days and increasing by 10 mg every week until target dose is achieved or symptoms and signs resolve.<sup>[11]</sup> Some studies suggest, administering the dose of 1,0 -1,5 mg /kg for about 6 months can lead to alleviation of myasthenia symptoms in a patient [13]. One should always remember to discontinue steroids very slowly to avoid rapid reduction in dosage, which can lead to recurrence of symptoms. During steroid therapy symptomatic treatment should be continued.

Results of treatment with prednisone in patients indicate the following [10]:

- 21% of patients achieve full remission after one course of treatment;
- 18% of patients require long-term therapy (usually 5-10 years);
- 41% of patients require multiple courses of treatments;
- 20% of patients do not respond to the treatment with corticosteroids.

Azathioprine and cyclophosphamide are also treatment options for patients with MG. The main indication for treatment with azathioprine is no improvement after using steroids or contraindication to steroid therapy. Azathioprine can induce leukopenia or thrombocytopenia, intractable vomiting, and hepatic dysfunction. Cyclophosphamide is an inhibitor of T helper which induce remissions as effective as corticosteroids, although there are no controlled studies of this drug for induction of remission in patients with MG [12]. According to Daniel Drachman, high doses of cyclophosphamide may be used in cases of drug resistant myasthenia gravis to yield satisfying results [14]. Authors suggested IV: 50-200 mg/kg/day, 4 infusions. The concept of such a treatment is “rebooting”, or resetting - the total destruction of the defective immune system. Unfortunately, it has a much higher cost-to-benefit ratio than azathioprine and not to mention side effects like hypertension and renal damage.

There is an established therapy for patients in myasthenic crisis. It is also often times used in patients undergoing thymectomy, and for the treatment of frail patients admitted to the hospital for initiation of corticosteroid therapy. The therapy may consist of: plasmapheresis, plasma exchange, or intravenous immunoglobulin. Plasmapheresis is a procedure in which cell mass is separated from the plasma and blood cells and fluids are re-introduced to the vasculature. By doing plasmapheresis one removes acetylcholine receptor antibodies from the circulation. It is confirmed that the plasmapheresis is more effective if the patient is treated with an immunosuppressive agent such as corticosteroids, azathioprine, or cyclophosphamide at the same time [12]. Plasma exchange usually involves exchanging a plasma volume of 2–3 l daily for 5 days. Improvement is probably more rapid than with IVIg but they have similar efficacy [11]. The effects of a course of plasmapheresis lasts only several weeks. Intravenous immunoglobulin (IVIg) is a procedure which involves 0,4 g/kg/day for 5 days and improvement occurs within 1–2 weeks, which lasts up to 12 weeks [8]. Intravenous infusions of immunoglobulin seems to be as effective as plasmapheresis. The advantages of IVIg are reduced side effects compared with immunosuppressive medications and plasmapheresis. However, there is a chance

that IVIg might cause thrombotic events, including myocardial infarction and stroke [15].

Patients in myasthenic crisis require hospitalization in the intensive care unit (ICU). Currently, mortality of myasthenic crisis is assessed at 3-10%, compared with 70% before the implementation of invasive ventilation [16].

Treatment of patients with myasthenic crisis consists of 4 parts

- treatment of life threatening condition, especially respiratory insufficiency;
- identifying and eliminating the factors causing myasthenic crisis;
- pharmacological treatment of myasthenia gravis; It includes plasmapheresis and IVIg. Rapid decision about immunomodulatory treatment allows to shorten the period depending on the respirator and reduce the risk of complications. We also continue corticosteroid treatment;
- reducing the risk of complications and their intense treatment.

It is important to keep in mind that some of myasthenia symptoms occur occasionally and can recede for some time. Symptoms can affect different muscle groups in different time, which makes this disease that much complicated to manage.

It is only natural for a person going through any illness to make temporary or permanent changes to his or her lifestyle depending on the nature of the illness. A persons lifestyle is affected by any illness but with illnesses like myasthenia gravis, the change is progressively insidious and drastic. The disease not only affects the patient but it also takes a toll on patients closest family members and maybe friends who are there tending to the patients every need. It severely hampers day-to-day physical activity in a patient and affects them mentally as well. Having no cure to this illness does not help a patients psyche or morale. Knowing that this illness will progressively get worse adds on to stress of living a life that will require implementing some serious changes and getting accustomed to those changes. In our analysis of 41 patients, five patients had a psychiatric illness: schizophrenia (2 patients), depression (2 patients) and one patient with anxiety disorder. According to Judith Schiffbauer, changes in lifestyle of patients with myasthenia gravis include but are not limited to, quitting their jobs, giving up caring for the home and children and these are all things that play a pivotal role in a persons identity so losing them piece by piece puts a patient in a state of depression and emotional distress [17].

The occurrence of major depression in myasthenia gravis patients happens to be notably elevated compared with healthy control subjects. Due to lack of enough data and studies performed solely on the psychiatric manifestations of MG, and a few studies that have focused on this issue have yielded somewhat conflicting results due many reasons like small sample size.<sup>[18]</sup> The precarious message, that physical symptoms of myasthenia gravis may falsely inflate the prevalence of depression in myasthenia gravis, is retained.<sup>[18]</sup> This issue is significant because between 20% to 30% of myasthenia gravis patients are originally misdiagnosed with a psychiatric disease.<sup>[18]</sup> More importantly, symptoms of depression among patients already diagnosed with myasthenia gravis may be dismissed as symptoms of a neurological illness which can delay the appropriate treatment of a mood disturbance problem [18]. This is not



the end of the extent of changes a person suffering from myasthenia gravis has to endure. According to Judith Schiffbauer, there are interpersonal relationship problems, dissatisfaction with ones own appearance and lack of understanding about myasthenia gravis by family and friends [17].

### CONCLUSION

It is important to not only address the physical symptoms in patients with myasthenia gravis, but also pay attention to mental and psychological symptoms in the patients to provide a better overall treatment plan and thereby, improving the quality of life substantially. There is still much work needed to be done in the analysis of myasthenia gravis to give a better understanding of the disease progression and pathophysiology in order to find a way to provide the best possible treatment (if not cure) with best possible results.

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Table 1. Admittance symptoms according to age groups

Symptoms	Number of cases	% of cases	20-35 year-olds	36-50 year-olds	51-65 year-olds	over 65 year-olds
breathing aggravation	16	29,09%	4	4	8	0
Ptosis	30	54,55%	5	8	10	7
Diplopia	18	32,73%	9	5	2	2
dysarthria	19	34,55%	4	2	11	2
dysphagia	27	49,09%	8	5	11	3
jaw drop	7	12,73%	0	1	4	2
neck muscles weakness	6	10,91%	4	1	1	0
upper limbs muscles weakness	20	36,36%	7	5	5	3
lower limbs muscles weakness	22	40,00%	9	6	3	4
number of patients in each age group			12	16	19	8

Table 2. Myasthenia gravis therapy according to cases

Treatment during hospitalisation stay	Number of cases	% of cases
mestinon only	23	41,82%
mestinon + encorton	8	14,55%
mestinon + encorton + IGIV	5	9,09%
mestinon + encorton + plasmapheresis	3	5,45%
mestinon + encorton + IGIV + plasmapheresis	3	5,45%
mestinon + IGIV	3	5,45%
mestinon + plasmapheresis	4	7,27%
mestinon + plasmapheresis + IGIV	1	1,82%
encorton + IGIV + plasmapheresis	1	1,82%
no specific drugs	4	7,27%

Table 3. Chronic diseases co-existing with myasthenia gravis according to sex

Chronic disease	Number of patients	% of all patients	Average age [years]	Number of women	% of women	Number of men	% of men
hypertension	21	51,22%	57,33	10	40,00%	11	68,75%
osteoarticular diseases	14	34,15%	52,43	8	32,00%	6	37,50%
thyroid diseases	12	29,27%	52,67	9	36,00%	3	18,75%
other cardiovascular diseases	9	21,95%	57,78	3	12,00%	6	37,50%
diabetes	7	17,07%	51,57	2	8,00%	5	31,25%
pulmonary diseases	5	12,20%	56,20	2	8,00%	3	18,75%

### ABSTRACT

Myasthenia gravis (MG) is a chronic autoimmune disease characterized by rapid fatigability of striated muscles. Its symptoms vary between individuals as well as between different exacerbations of the same patient. Improperly treated, myasthenia gravis can lead to life-threatening condition – miastenic crisis. We conducted a non-invasive, retrospective study of 55 medical histories of patients referred to the Department of Neurology Medical University of Lublin from January 2010 to October 2013 due to exacerbation of myasthenia gravis. Sex, age of onset of MG symptoms, duration of myasthenia gravis, admittance symptoms, duration of hospital stay, mediastinal tumor examination, presence of myasthenia crisis, mortality, myasthenia gravis therapy, co-existence of other chronic somatic diseases and psychiatric diseases were analyzed.

### STRESZCZENIE

Miastenia gravis (MG) jest przewlekłą chorobą autoimmunologiczną charakteryzującą się męczliwością mięśni poprzecznie prążkowanych. Jej objawy różnią się zarówno pomiędzy pacjentami, jak w poszczególnych zaostrzeniach u tego samego pacjenta. Niewłaściwie leczona może doprowadzić do zagrażającego życiu przełomu miastenicznego. Przeprowadzono nieinwazyjne, retrospektywne badanie 55

historii chorób pacjentów hospitalizowanych w Klinice Neurologii Uniwersytetu Medycznego w Lublinie w czasie od stycznia 2010 roku do października 2013 roku z powodu zaostrzenia objawów miastenii. Badaną grupę przeanalizowano pod kątem płci, wieku wystąpienia pierwszych objawów choroby, długości trwania choroby, objawów obecnych przy przyjęciu do oddziału, długości hospitalizacji, obrazowania guza śródpiersia, wystąpienia przełomu miastenicznego, śmiertelności, leczenia, współwystępowania innych przewlekłych chorób somatycznych i psychiatrycznych.

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