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Transient myasthenia gravis as a complication of COVID-19 in a 1.5-year-old boy: a case report and literature review

Przejściowa miastenia jako powikłanie COVID-19 u 1,5-rocznego chłopca: opis przypadku i przegląd piśmiennictwa

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Abstract

SARS-CoV-2 infection often causes neurological symptoms and complications. Those associated with the production of anti-acetylcholine receptor antibodies are rare. The aim of the study was to present a case of transient myasthenia gravis as a possible complication of COVID-19. A 1.5-year-old boy was admitted on day 7 of varicella due to poor general condition and anuria. On examination, he presented with dehydration, fatigue, sleepiness, and bilateral ptosis. High titre of serum anti-SARS-CoV-2 antibodies was revealed with a history of viral infection 2 weeks prior. An initial diagnosis of encephalitis was made and treatment was started. Despite clinical improvement, gait disturbances and ptosis persisted and the boy was sent for further neurological evaluation. High titre of anti-acetylcholine receptor antibodies (2.98 nmol/L; normal <0.50 nmol/L) confirmed myasthenia gravis, but no treatment was started. Symptoms and antibodies resolved after 3 and 4 months, respectively. A follow-up after one year showed no recurrences. **Conclusion:** Transient, self-limiting myasthenia gravis may develop in a child as a complication of viral infection, including COVID-19.

Keywords: children, COVID-19, neurological complications, myasthenia gravis

Streszczenie

Zakażenie SARS-CoV-2 często powoduje objawy i powikłania neurologiczne, z których te związane z wytwarzaniem przeciwciał przeciwko receptorom acetylocholiny należą do najrzadszych. Celem pracy był opis przypadku przejściowej miastenii jako prawdopodobnego powikłania COVID-19. Półtoraroczny chłopiec został przyjęty do szpitala w 7. dobie ospy wietrznej z powodu złego stanu ogólnego i bezmoczu. W badaniu stwierdzono odwodnienie, osłabienie uniemożliwiające chodzenie, patologiczną senność i opadanie powiek, a w badaniach dodatkowych – wysokie miano przeciwciał przeciwko SARS-CoV-2 (w wywiadzie infekcja wirusowa 2 tygodnie wcześniej). Rozpoznano wstępnie zapalenie mózgu i włączono leczenie. Pomimo poprawy zaburzenia chodu i opadanie powiek utrzymywały się. Pacjenta skierowano na oddział neurologiczny, gdzie wykazano wysokie miano przeciwciał przeciwko receptorom acetylocholiny w surowicy (2,98 nmol/l; norma <0,50 nmol/l). Rozpoznano miastenię, nie podjęto leczenia. Objawy ustąpiły po trzech miesiącach, a przeciwciała zniknęły z surowicy po czterech. W trakcie rocznej obserwacji nie stwierdzono nawrotów. **Wniosek:** Miastenia po zakażeniu wirusowym, w tym SARS-CoV-2, może mieć charakter przejściowy.

Słowa kluczowe: dzieci, COVID-19, powikłania neurologiczne, miastenia

INTRODUCTION

Infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) can cause multiple neurological complications, such as persistent olfactory and taste dysfunction, headaches, muscle weakness of the limbs, neuropathies, seizures, coma, encephalopathy, ischaemic and haemorrhagic stroke, encephalitis, aseptic meningitis, acute disseminated encephalomyelitis (ADEM), posterior reversible encephalopathy syndrome (PRES), Guillain–Barré syndrome, Miller-Fisher syndrome, ataxia, demyelinating diseases and psychiatric disorders. Although these are usually transient, they can also lead to serious sequelae and even death. In their meta-analysis of 28 studies, Pousa et al. estimated the incidence of COVID-19 neurological complications in children to be around 9%[1]. This incidence is higher, up to 32%, in children with multisystem inflammatory syndrome (MIS-C)[2]. The largest group of paediatric patients with nervous system disorders has so far been described by LaRovere et al., who found them in 22% of 1,695 patients treated in 61 hospitals across the USA[3]. Damage to neural and vascular endothelium caused by viruses, inflammatory reaction and autoimmunisation with production of autoantibodies are considered causative mechanisms[4].
The aim of this study was to present myasthenia gravis (MG) as a very rare complication of COVID-19 in a child.

CASE REPORT

A 1.5-year-old boy was transported to hospital by an emergency medical team on day 7 of varicella infection due to poor general condition and anuria. Because of the complicated course of the illness with prolonged fever, agitation and insomnia, he had previously been consulted three times by his general practitioner, who recommended treatment with promethazine followed by an antibiotic for "pharyngitis". The patient had no history of serious illness. The perinatal, family, and developmental history was unremarkable. Two weeks earlier, the child and his family members developed symptoms of viral respiratory tract infection, the cause of which had not been identified.
On admission, the patient's condition was severe, with features of dehydration and significant weakness preventing walking, pathological somnolence and ptosis on physical examination (Fig. 1 A). Laboratory workup showed high titres of antibodies against SARS-CoV-2 and low inflammatory markers (Tab. 1). Initially, the patient's poor condition was linked to dehydration and several days of promethazine therapy. Since there was no improvement after intravenous fluid therapy, a suspicion of viral encephalitis was raised. Cerebrospinal fluid (CSF) analysis was normal and the sample was sent for polymerase chain reaction (PCR) and culture, while treatment with cefotaxime, acyclovir and steroids was implemented. Improvement was observed after four days, and the therapy was continued for one week until negative CSF microbiological and molecular tests were

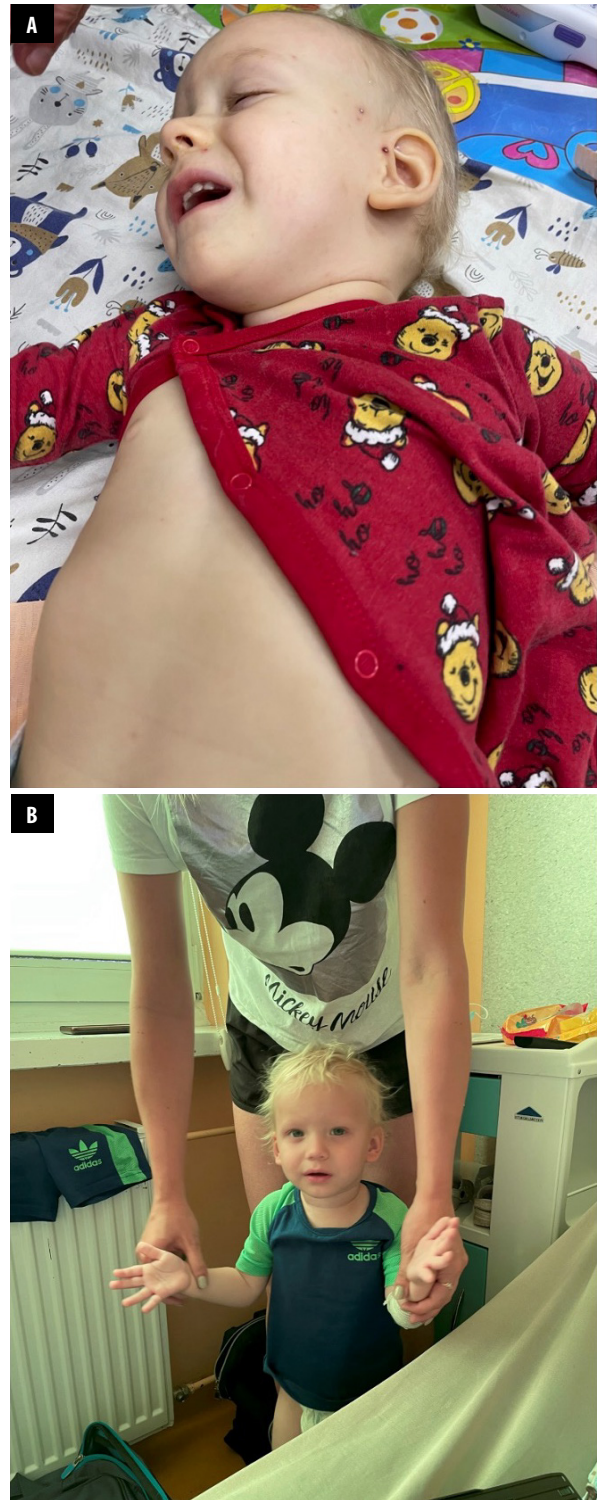

*Fig. 1. Patient with MG symptoms on admission (***A***) and at 1-year follow-up (***B***)*

obtained. Since gait disturbances and ptosis persisted, the patient was transferred to the Department of Paediatric Neurology for further diagnosis.
Neurological examination showed reduced limb muscle tone, diminished knee and Achilles tendon reflexes, Gowers sign, ptosis and convergent strabismus of the left eye.

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• Blood count: Hb – 11.6 g/L, Ht – 35.7%, RBC – 4.7 T/L, WBC – 6.82 G/L, Plt – 196 G/L • Differential: segmented neutrophils – 45%, lymphocytes – 45%, monocytes – 9%, eosinophils – 1% • CRP – 1.92 mg/L (N < 5 mg/L), procalcitonin – 0.08 ng/mL (N < 0.5 ng/mL) • Serum creatinine – 0.2 mg/dL, serum urea – 22 mg/dL, serum uric acid – 6.1 mg/dL • Whole blood glucose – 69 mg/dL, serum sodium – 138 mmol/L, serum potassium – 4.5 mmol/L • Plasma fibrinogen – 297 mg/dL, APTT – 24.4 s, INR – 1.08, plasma D-dimers – 836 ng/mL (N < 500 ng/mL) • AlAT – 10 U/L, AspAT – 28 U/L • Troponin T – 4.41 ng/L (N < 14 ng/L), NT-proBNP – 132 pg/mL • SARS-CoV-2 in nasopharyngeal swab: negative antigen • Serum anti-SARS-CoV-2 antibodies – >250 U/mL • General urine test: SG – 1,015 g/L, pH – 7.0, glucose – negative, protein – negative, ketones – 25 mg/dL, RBC – normal, WBC – normal • Cerebrospinal fluid analysis: colorless, clear, protein – 38.6 mg/dL, glucose – 58.0 mg/dL, chloride – 127 mmol/L, cytosis – 1/µL, Pandy and Nonne–Apelt reactions – negative
AlAT – alanine aminotransferase; **APTT** – activated partial thromboplastin time; **AspAT** – aspartate aminotransferase; **CRP** – C-reactive protein; **Hb** – haemoglobin; **Ht** – haematocrit; **INR** – international normalised ratio; **NT-proBNP** – N-terminal pro-B-type natriuretic peptide; **RBC** – red blood cells; **SG** – specific gravity; **WBC** – white blood cells.

Tab. 1. Laboratory workup on admission

Nuclear magnetic resonance (NMR) of the head showed small hyperintense areas on T2-weighted and FLAIR images in the region of the frontal horns of the lateral ventricles; electroencephalography showed no abnormalities. Serological investigations showed high serum acetylcholine receptor antibody (AChRAb) titres (2.98 nmol/L; positive >0.50 nmol/L); onconeuronal and antineuronal antibodies (anti-AMPA, anti-GABA B, anti-NMDA, anti-DPPX, anti-CASPR 2, anti-LGI 1) were absent. MG secondary to SARS-CoV-2 and varicella infections was diagnosed. The patient's neurological condition gradually improved, and therefore no treatment was initiated. The AChRAb titre decreased to 0.80 nmol/L after one month, ptosis resolved after three months, and there were no detectable serum AChRAbs after four months. At one-year follow-up, the boy had no recurrence of symptoms or other neurological sequelae of COVID-19, and showed normal psychomotor development (Fig. 1 B).

DISCUSSION

Myasthenia gravis is a chronic autoimmune disorder involving the neuromuscular junction, the symptoms of which arise from the blockade of postsynaptic membrane proteins, which consequently impairs contractility and increases fatigability of striated muscles. In the classical form, nicotinic acetylcholine receptors are targeted; AChRAbs are detected in 85% of patients with generalised MG and in about 50% of those with the ocular form. In the so-called seronegative form (without AChRAb), antibodies may be present against other components of the postsynaptic membrane: muscle skeletal receptor tyrosine kinase (MuSKAb), and low-density lipoprotein receptor-related protein 4 (LRP4Ab), as well as intracellular myocyte proteins – titin and ryanodine. Although the causes of autoantibody production remain unknown, a genetic predisposition to the disease is suspected, as indicated by a higher prevalence of certain human leukocyte antigens (HLAs) in patients and co-occurrence of other autoimmune diseases with MG. In most cases, it is not possible to identify a single trigger (sometimes a viral infection). To date, MG has been described following infection with HBV, HCV, HSV, HIV, EBV and varicella, Zika and West Nile viruses. Affected patients experience periods of remission and exacerbations. The clinical picture is dominated by weakness and fatiguability of the external ocular, bulbar, limb and respiratory muscles, with ocular symptoms usually developing first. Thymoma is found in approximately 10% of patients. The incidence of MG is 5–10 people per 100,000 population, with mainly young women and men over 60 years of age being affected. MG is rare in children, with paediatric patients accounting for only about 10–15% of cases. In this age group, apart from juvenile MG with a classical course, transient neonatal myasthenia associated with the transfer of maternal antibodies through the placenta and congenital myasthenic syndromes caused by defects in various proteins of the neuromuscular junction have been distinguished. The diagnosis of MG, in addition to clinical symptoms, requires one of three additional criteria to be met: the presence of typical serum autoantibodies, increased fluctuations in single-fibre electromyography (SFEMG) and a positive repetitive nerve stimulation (RNS) test. Treatment includes cholinesterase inhibitors (mainly pyridostigmine), immunosuppressants, intravenous immunoglobulin infusions, plasmapheresis and thymectomy. Recently, biologic drugs (rituximab and eculizumab) have also been utilised in cases refractory to standard therapy. This combination treatment allows for achieving positive outcomes in more than 80% of patients[5,6].

Among the 365 children with COVID-19 neurological complications described by LaRovere et al., 43 were affected by serious life-threatening conditions, such as severe encephalopathy (n = 15), ischaemic or haemorrhagic stroke (n = 12), encephalomyelitis (n = 8), acute cerebral oedema (n = 4) and Guillain–Barré syndrome (n = 4). Among milder complications, seizures predominated in younger children, whereas loss of smell and taste, headaches, fatigue and muscle weakness were most common in older children. There were no patients with a clinical picture of MG in this population[3]. Three groups of authors: Restivo et al.[7], Huber et al.[8] and Sriwastava et al.[9], who described patients with newly diagnosed MG after COVID-19 in 2020 and 2021, presented them as the first cases in the world; however, given the publication dates, priority should be given to Italian researchers from Catania, Rome and Padua[7]. To date, 23 patients (11 females and 12 males) aged 6–83 years (median 61 years) have been described, in whom the diagnosis of MG had a temporal relationship with SARS-CoV-2 infection, and whose diagnosis was confirmed (with the exception of one patient) by antibody testing[7–25] (Tab. 2). One patient developed symptoms of both

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diseases simultaneously; in the others, the time from the first COVID-19 symptoms to the onset of MG symptoms ranged from 5 days to 5 months (median 18 days). Five patients had a history or a family history of other autoimmune disorders. Most patients developed generalised muscle weakness (n = 17), and six patients experienced respiratory failure. The ocular form of MG, in which symptoms were limited to the muscles of the eye region, was less common (n = 5); additionally, one patient presented with both ocular and bulbar symptoms. No treatment was used in only one patient, a 48-year-old male with ocular MG, whose condition improved spontaneously[10]. The remaining patients received acetylcholinesterase inhibitors (n = 20), glucocorticoids (n = 15), intravenous immunoglobulins (n = 8), azathioprine (n = 4) and methotrexate (n = 1). Additionally, plasmapheresis and thymectomy were performed in 2 and 3 patents (including two patients with thymoma and one patient with thymic hyperplasia), respectively. Although improvement was achieved in all patients, the follow-up was not sufficiently long to draw conclusions about the further course of the disease.

Literature reports to date include only two cases of children. A seven-year-old girl was treated for 10 days in a paediatric intensive care unit (ICU) in Cape Town, South Africa, for MIS-C manifesting with shock and respiratory failure. Her parents had developed pharyngitis two weeks earlier and their daughter had serum anti-SARS-CoV-2 antibodies. Immediately after discharge from the ICU, she was found to experience muscle pain and weakness in the distal limbs, which was considered to be associated with myositis and corticosteroid therapy. After further 3 days, ptosis and double vision with Cogans sign and curtain sign on neurological examination developed. The diagnosis of ocular MG was confirmed by AChRAb testing, with titres of 0.51 nmol/L (normal <0.39 nmol/L). Treatment included pyridostigmine, prednisone and methotrexate. After one month, resolution of ocular symptoms and improvement in limb strength was achieved, and serum AChRAbs were no longer detected. Pharmacotherapy was gradually discontinued with no relapse observed[16]. The second patient, a six-year-old girl, was admitted to hospital in Ankara, Turkey, for MIS-C with a similar clinical course. Three weeks earlier, she had developed a cough for 2 days, with the presence of SARS-CoV-2 in nasopharyngeal swab as tested by PCR. When assisted breathing treatment was discontinued after 6 days, generalised flaccid paresis was found, which gradually resolved with intensive rehabilitation. The patient was discharged home after 16 days. Six weeks later, she presented with fatigue, which exacerbated in the evening, and intermittent ptosis. A fatigue test confirmed ptosis and serum AChRAb titres of 6.42 nmol/L (normal <0.25 nmol/L) were detected. Ocular MG was diagnosed and treatment with pyridostigmine was initiated. The symptoms resolved after 4 months, but antibody titres remained high[20].

The aetiopathogenesis of COVID-19-associated MG primarily considers the activation of autoimmune mechanisms in genetically predisposed individuals, in whom SARS-CoV-2 infection acts as a trigger for the disease, which then progresses independently. It has been suggested that the generation of autoantibodies may be linked to virus-induced inflammatory changes in the thymus, immune dysregulation associated with the cytokine storm in COVID-19, as well as a break in immune tolerance to self-antigens. The so-called molecular mimicry, i.e. the similarity between the epitopes of SARS-CoV-2 virus and the nicotinic acetylcholine receptor and MuSK on the neuromuscular junction, leading to the production of antibodies that react with the components of the muscle cell membrane following coronavirus infection, may also play an important role. Other immunological phenomena associated with the initiation of autoimmunity include epitope spreading and bystander activation[12,15,16,20,24,26]. Furthermore, some medications previously used for the treatment of COVID-19, such as hydroxychloroquine and azithromycin, may contribute to neuromuscular junction dysfunction[15]. All these mechanisms may also play an important role in patients with MG diagnosed prior to COVID-19, in whom the infection may increase both disease severity and the risk of respiratory failure[27]. Analysis of the clinical course in the boy admitted to our Department and the patient described by Pérez Álvarez et al.[10] suggests that these processes are reversible up to a certain point, i.e. antibody synthesis may cease after elimination of the potential causative agent. This was also suggested by Essajee et al., although immunosuppressive treatment was used in the described patients[16]. The low number of documented cases of MG after SARS-CoV-2 infection with the high prevalence of COVID-19 also does not allow to exclude coincidence. Evidence for a causal relationship between the two diseases could be provided by detailed epidemiological analyses of the prevalence of MG in different populations during the pandemic period, but such studies are lacking to date.

Almost all patients with MG after COVID-19 were treated with different modalities, and eligibility for treatment was not related to disease severity. Considering the possibility of spontaneous cure, it seems reasonable to defer therapy and implement watchful waiting with the monitoring of the dynamics of changes in antibody levels over time in cases with non-life-threatening symptoms. In our patient, who is the youngest patient with MG symptoms after COVID-19 described so far, a decrease in symptom severity was already observed within the first weeks after the diagnosis, and there was s drop in AChRAb levels. This justified refraining from causal treatment, which proved unnecessary during further follow-up. In the differential diagnosis of MG triggers, varicella infection, previously described in the literature, was also considered[28], but the sequence of symptoms indicated the involvement of SARS-CoV-2 in the aetiopathogenesis in the first place.

Although the pandemic has subsided, sporadic cases of COVID-19 in children are still observed, and the low vaccination coverage in this age group leads us to a conclusion

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No.	Author, year of publication, country	Age [years], sex [F/M]	Autoimmune diseases/ positive family history	Time elapsed from first COVID-19 symptoms to MG	Symptoms	AChRAb titre	Treatment	Treatment outcome
1	Restivo et al., 2020, Italy[7]	64, M	No	5 days	Generalised	22.5 pmol/L (N < 0.4)	PST, GCS	Improvement
2	Ibid.	68, M	No	1 week	Generalised	27.6 pmol/L	IVIG	Improvement
3	Ibid.	71, F	No	5 days	Generalised, RF	35.6 pmol/L	PE	Improvement
4	Huber et al., 2020, Germany[8]	21, F	No/Addison's disease, Hashimoto's disease, pernicious anemia	2 weeks	Ocular	0.9 nmol/L (N < 0.25)	IVIG, PST	Improvement
5	Sriwastava et al., 2021, India[9]	65, F	No	2 weeks	Ocular	7.39 nmol/L (N < 0.02)	PST, GCS	Improvement
6	Pérez Álvarez et al., 2020, Spain[10]	48, M	Psoriasis/no	10 days	Ocular	1.10 nmol/L (N < 0.20)	None	Improvement
7	Assini et al., 2021, Italy[11]	77, M	No	8 weeks	Ocular, bulbar	Negative AChRAb, positive MuSKAb	PST, AZT	Improvement
8	Muhammed et al., 2021, UK[12]	24, F	No	4 weeks	Generalised	Negative AChRAb, positive MuSKAb	IVIG, PST, GCS	Improvement
9	Bhandarwar et al., 2021, India[13]	61, M	No	8 weeks	Generalised	11.3 nmol/L	IVIG, GCS, PST, thymectomy of a thymoma	Improvement
10	Karimi et al., 2021, Iran[14]	61, F	No	6 weeks	Generalised	10 pmol/L (N < 0.4)	PE, PST, GCS, thymectomy of a thymoma	Improvement
11	Ibid.	57, M	No	10 days	Generalised	8 pmol/L	PST, GCS	Improvement
12	Ibid.	38, F	No	4 weeks	Generalised	>8 pmol/L	PST, GCS	Improvement
13	Muralidhar Reddy et al., 2021, India[15]	65, M	No	6 weeks	Generalised, RF	4.5 nmol/L (N < 0.4)	GCS, PST, IVIG, AZT	Improvement
14	Essajee et al., 2021, RPA[16]	7, F	No	18 days (13 days after MIS-C)	Generalised	0.51 nmol/L (N < 0.39)	PST, GCS, MTX	Improvement
15	Rahimian et al., 2022, Iran[17]	31, F	No/IBD, Hashimoto's disease, Graves' disease	Simultaneous	Ocular	Brak AChRAb	PST, GCS	Improvement
16	Taheri et al., 2022, Iran[18]	35, F	No	3 weeks	Generalised	0.57 nmol/L (N < 0.45)	PST	Improvement
17	Chatterjee et al., 2022, USA[19]	83, M	No	7 weeks	Generalised, RF	0.26 nmol/L (N < 0.02)	GCS, PST, AZT	Improvement
18	Jõgi et al., 2022, Estonia[20]	65, M	No	2 weeks	Generalised	>20 nmol/L (N < 0.44)	GCS, IVIG, PST, AZT	Improvement
19	Yavuz et al., 2022, Turkey[21]	6, F	No	11 weeks (8 weeks after MIS-C)	Generalised	6.42 nmol/L (N < 0.25)	PST	Improvement
20	Croitoru et al., 2022, Romania[22]	78, M	No	9 days	Generalised, RF	19.2 nmol/L (N < 0.25)	PST, IVIG, GCS	Improvement
21	De Giglio et al., 2023, Italy[23]	74, M	Graves' disease/no	8 weeks	Ocular	3.2 mmol/L (N < 0.5)	GCS, PST	Improvement
22	Tereshko et al., 2023, Italy[24]	19, F	Hashimoto's disease, autoimmune gastritis/no	13 days	Generalised, RF	>5 nmol/L (N < 0.25)	IVIG, GCS, PST, thymectomy of hyperplastic thymus	Improvement
23	Sadiq et al., 2023, USA[25]	46, F	No	5 months	Generalised, RF	Negative AChRAb, positive LRP4Ab	PST	Improvement

AChRAb – acetylcholine receptor antibody; **AZT** – azathioprine; **F** – female; **GCS** – glucocorticoids; **IVIG** – intravenous immunoglobulin; **LRP4Ab** – low-density lipoprotein receptor-related protein 4 antibody; **M** – male; **MIS-C** – multisystem inflammatory syndrome in children; **MTX** – methotrexate; **MuSKAb** – muscle skeletal receptor tyrosine kinase antibody; **PE** – plasmapheresis; **PST** – pyridostigmine; **RF** – respiratory failure.

Tab. 2. A summary characteristics of 23 patients with post-COVID-19 MG described in literature

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that this will be a permanent situation. It can therefore be expected that the number of case reports of paediatric patients with newly diagnosed MG will also increase. Recording such cases and their in-depth clinical analysis seem necessary to determine an appropriate course of management, including avoidance of unnecessary therapy.

CONCLUSIONS

Myasthenia gravis following viral infection, including SARS-CoV-2, may be transient. Watchful waiting instead of treatment seems an optimal strategy in cases of rapid spontaneous clinical improvement with a drop in ACHR antibodies.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study: TJ, AK. Collection, recording and/or compilation of data: TJ, MC, JM, AK. Analysis and interpretation of data: TJ, MC, JM, AK. Writing of manuscript: TJ, AK. Critical review of manuscript: MC, JM. Final approval of manuscript: TJ, MC, JM, AK.

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