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Case Report

The Myasthenic Crisis Revealed by SARS COV-2 Viral Pneumopathy: A Case Report

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Abstract

Myasthenia is an autoimmune disease characterized by the destruction of postsynaptic acetylcholine receptors in striated skeletal muscles and is most common in young women. Myasthenia can be diagnosed through the detection of anti-acetylcholine receptor antibodies. Its treatment includes anticholinesterase drugs, thymectomy, and restrictive drugs that may aggravate the myasthenic crisis. We herein report a rare case of accidental revelation of a myasthenic crisis in a 26-year-old woman triggered by a SARS COV-2 viral pneumopathy.

Keywords: *Myasthenic Crisis; Myasthenia Gravis; SARS COV-2; Immunoglobulin; Resuscitation; Prognosis*

Introduction

Myasthenia gravis is an autoimmune disease affecting neuromuscular transmission and resulting in generalized or localized muscle weakness most often due to the presence of autoantibodies directed against acetylcholine receptors (AchR) in the postsynaptic motor endplate [1]. Myasthenic seizure is a complication of myasthenia gravis characterized by the worsening of muscle weakness, leading to respiratory failure-prone to require intubation and mechanical ventilation [2]. The disease classically affects the young female population (sex ratio 2 F/1 M) and is distributed in a bimodal manner, with the first peak of frequency during the second and third decades, affecting mostly women, and a second peak during the sixth and seventh decade, whereas it affects mostly men [3]. Diagnosis is based mainly on questioning, confirmed by therapeutic tests, biological, electrophysiological, and radiological investigations. In the perioperative setting, myasthenic crisis usually occurs in the context of thymectomy surgery [4]. The management of the Myasthenic crisis is done in an intensive care unit. The rest of the treatment consists mainly of anticholinesterase drugs, immunoglobulins, and thymectomy, and compliance with drug restrictions. The prognosis depends on early and adapted management [5]. In this document, we report a rare case of accidental revelation of a myasthenic crisis in a young woman aged 26 years without any particular pathological history triggered by a viral pneumopathy with SARS COV-2.

This article was reported using the SCARE Checklist 2020, or CARE Checklist 2016 criteria.

Case Report

We report a case of a 26-year-old female patient who had not been vaccinated against SARS-COV2 and had no specific pathological history. Seven days before her admission, she presented a respiratory discomfort associated with a cough in the context of uncalculated fever with progressive worsening of the symptoms. She was admitted to the emergency room and admitted to the intensive care unit for specialized care afterward. On admission, she was conscious with a Glasgow score of 15/15, reactive and symmetrical pupils, drooping eyelids, decreased swallowing and cough reflexes, tetraparesis. On the respiratory level, SpO2 at 88% in the open-air rose to 98% under a 4 liter per minute scope, polypneic at 24 cycles per minute. In front of this picture of respiratory distress, a thoracic scanner was carried out showing lesions compatible with a SARS COV-2 viral pneumopathy with a parenchymatous attack estimated at 10-25%. Hemodynamics were stable with a BP of 120/60 mmHg, heart rate of 80 beats per minute. In addition, the RT-PCR of the nasopharyngeal swab was revealed positive. The patient was apyretic at 37.4°C. Biology: blood ionogram was unremarkable, Interleukin 6 at 2 pg/ml, C-reactive protein at 17mg/l, Procalcitonin at 0.01g/l, Hb at 13g/dl, WBC at 6700/mm3, lymphocytes at 950/mm3, Platelets at 235000/mm3, fibrinogen at 2.3g/l, PT at 97%. A myasthenic crisis was evoked given the pre-existing symptomatology with positivity to the neostigmine test, a Myasthenic Score rated at 58% and the diagnosis of autoimmune myasthenia was retained because of the positivity of anti-acetylcholine receptor antibodies (45 nmol/l, normal range < 0.2 nmol/l) with an electroneuromyogram of four limbs in favor of a post-synaptic block of the myasthenic type. The rest of the autoimmune workup was

negative, and the CT scan of the thorax excluded thymoma. Given the rapid decompensation of the clinical condition, we retained the diagnosis of a myasthenic crisis on SARS-COV2 infection. The management was based on the introduction of intravenous immunoglobulins at a dose of 1g/kg for 48 hours, anticholinesterase drugs based on pyridostigmine 60mg every 8 hours, and the avoidance of all drugs contraindicated for myasthenia. As well as antibiotic therapy based on ceftriaxone, vitamin therapy C&D, Zinc, LMWH preventive dose, aspirin, gastric protection, nursing, and adapted physiotherapy.

The evolution was marked by an improvement of the muscular function with a Myasthenic score rated at 85% and respiratory function with a SpO2 at 100% in free air.

The patient was discharged on the tenth day of her hospitalization and referred to the neurology department for further management.

Discussion

Myasthenia gravis is an autoimmune disease affecting neuromuscular transmission and manifested by generalized or localized muscle weakness caused by autoantibodies directed against acetylcholine receptors in the postsynaptic motor plate. Myasthenic crisis is a complication of myasthenia characterized by increased muscle weakness that can potentially lead to acute respiratory failure requiring intubation and mechanical ventilation. The prevalence of myasthenia is between 5 and 150 cases per million population, with an incidence of 2.5 to 20 cases per year per million population [6]. Most patients who develop a myasthenic crisis have a triggering factor such as a respiratory infection (SARS COV-2), emotional stress, microaspirations, change in medication regimen, trauma, or surgery. However, in 30% to 40% of cases, the attack is idiopathic [2]. Many medications (Table 1) can potentially exacerbate the myasthenic seizure and therefore should be excluded or used with caution [7].

The diagnosis of myasthenia gravis is based on questioning, symptoms, and investigations. The neostigmine (anticholinesterase) test is used to complement the clinical examination. Electromyography (EMG) shows a progressive decrease in the amplitude and duration of the potentials collected during iterative supramaximal stimulation between 2 and 5 Hz. The reduction between the second and fifth potentials must be greater than 10% to confirm the diagnosis. The detection of anti-RACh antibodies by radioimmunological assay, found in 80% of cases, validates the diagnosis [2, 3, 7, 8].

In our case, the symptoms were eyelid drooping, decreased swallowing and coughing reflexes, respiratory distress, tetraparesis, and the anti-acetylcholine receptor antibody assay was positive. The EMG showed the presence of post-synaptic blocks of the myasthenic type.

Table 1: Drugs contraindicated in Myasthenia gravis

Medications formally contraindicated	Drugs to be used with caution
- Aminoside	- **Neuroleptiques**
- **Colimycine**	- **Benzodiazépines**
- **Polymyxine**	- **Phénothiazines**
- **Injection de cycline**	- **Carbamazépine**
- **Quinine - Chloroquine**	
- **Quinidine**	
- **Procaïnamide**	
- **Diphényl-hydantoïne**	
- **Triméthadione**	
- **Bêta-bloquants**	
- **Dantrolène**	
- **D -Pénicilline**	

Conclusion

We understand from our case that myasthenia gravis can be the first episode of the disease regardless of age or gender, SARS COV-2 infection can highlight under-diagnosed myasthenia. Early diagnosis and medical treatment can make the prognosis less bleak. Myasthenia and SARS COV-2 infection in a young patient without co-morbidities, if well managed, has a good prognosis and in the management of SARS COV-2 infection associated with myasthenia, the avoidance of any contraindicated drugs would allow a good evolution.

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