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Case Report Open Access

Case Report: De novo Ocular Myasthenia Gravis after the mRNA Vaccine for SARS-COV2.

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Abstract

Autoimmune Myasthenia gravis is a neuromuscular junction disease caused by destruction of the acetylcholine receptor on the postsynaptic membrane, mediated by autoantibodies and clinically characterized by skeletal muscle weakness. This can be triggered by drugs, infections, and in rare cases by vaccines. During the COVID pandemic, de novo cases and exacerbations of myasthenia gravis due to viral infection have been documented. Although few cases related to COVID vaccination.

A 69-year-old female patient with blurred vision, a clinic that began 10 days later after receiving first dose of COVID vaccine, and diplopia and right palpebral ptosis after the second dose, 30 days later. In the examination, Edrophonium test was clearly positive, treatment with pyridostigmine and prednisone was started and after 14 days the clinic showed improvement until the resolution of the symptoms

There are few reports of exacerbations or triggers for the appearance of myasthenia gravis, a series of 27 cases has been published where there are outbreaks of immune-mediated diseases or the new appearance of autoimmune diseases, in which it is observed that the time of appearance of the outbreak was on average 4 days up to a maximum of 25 days.

In conclusion, Myasthenia gravis is a rare complication of the BNT162b2 COVID-19 vaccine. Its potential severity and the current lack of knowledge of the real incidence after vaccination makes it necessary to maintain an attitude of vigilance in the face of symptoms that suggest it.

Keywords: Myasthenia Gravis, Autoimmune Disease, SARS-COV2, Eyelid ptosis

Introduction

Myasthenia gravis (MG) is an autoimmune disease that affects the neuromuscular junction, mediated by circulating antibodies directed against acetylcholine receptors on the motor endplate. Clinically it is characterized by weakness in the extraocular muscles, muscles of bulbar innervation and extremities [1].

The incidence of MG is 1-2 in 100,000 and the prevalence is 5-15 in 100,000. It has a bimodal presentation, being more frequent among women in the second and third decades of life, with a second peak in the fifth decade, where the distribution by sex is equal, although in recent years the cases in the elderly have increased [1].

The disease can be exacerbated and triggered by various factors such as medication, infections and, rarely, vaccines [2-3]. During the COVID-19 pandemic, various observational studies have reported neurological affectations and complications, including MG, although with uncertain direct causality. In some patients, COVID-19 worsened the weakness, leading to a myasthenic crisis. Very infrequently, new-onset autoimmune MG has also been reported after COVID-19 [2-4].

MG patients are considered a high-risk group for the presentation of respiratory failure due to SARS-COV2 infection, both due to the entity itself and due to immunosuppressive treatments, with a higher morbidity and mortality that justifies the recommendation to vaccinate them. However, it is not clear whether the vaccine can trigger a myasthenic crisis or the de novo appearance of autoimmune MG [2,4,5].

We present the case of a woman who developed de novo autoimmune MG after vaccination against SARS-COV2.Clinical case
69-year-old women with a known beta-lactam allergy and a history of hypertension on Olmesartan treatment and no other associated pathology presented in November 2020 withfever, cough without expectoration, fatigue and arthromyalgia. Physical examination was unremarkable. A left basal interstitial infiltrate was observed in the chest X-ray, analytical inflammatory markers were normal, and arterial gasometer did not show signs respiratory failure. PCR for SARS-COV2 was positive. The duration of symptoms was two weeks, but residual fatigue persisted, for which she continued to be followed by the pneumology department. Functional respiratory tests were normal and chest X-ray showed no signs of pneumonia or fibrosis.

Subsequently, the patient received two doses of the BNT162b2 vaccine for COVID-19. The firstdose was on 4/9/21, 10 days later the patient noticed blurred vision in the evening or after reading for long periods of time, she even referred double vision. After the second dose, on 4/30/21, she reported worsening symptoms, presenting with binocular diplopia in the eveningaccompanied by predominantly right eyelid ptosis. At three weeks of evolution, she was evaluated by the neurology department. The physical examination revealed a blood pressure 120/80mmHg, heart rate 70bpm, oxygen saturation 98%, temperature 36.21^{0}C, with the rest ofthe physical examination being unremarkable. Neurologic examination showed patient was conscious and oriented to person, place, and time, no aphasia or dysarthria, no hemianopsia, normal saccades, notable right palpebral ptosis. In the sustained upward gaze diplopia appeared at 10 seconds with bilateral palpebral ptosis and paresis of both superior recti.
Remaining cranial nerves normal, muscular strength 5/5 in the four extremities, osteotendinous reflexes present and symmetrical, no sensory or gait disturbances.

Blood tests were unremarkable except for elevated PTH levels. Anti-acetylcholine receptor antibody levels were 20 mg/dl (normal value <0.50). Edrophonium test was clearly positive. Abrain CT scan found mild cortico-subcortical atrophy and a thoracic CT effectively ruled out a possible thymoma. The ENG-EMG jitter study, six weeks after our initial assessment, reportedno alteration in neuromuscular transmission.

With a diagnosis of ocular myasthenia gravis, treatment with pyridostigmine was started, observing clinical improvement, but diplopia persisted when vision was forced (for example,reading), for which prednisone was added at a dose of 30 mg daily, obtaining a total improvement after 14 days of treatment.

At the present time, after 100 days of treatment, patient remains asymptomatic, withoutcomplications and with good tolerance.

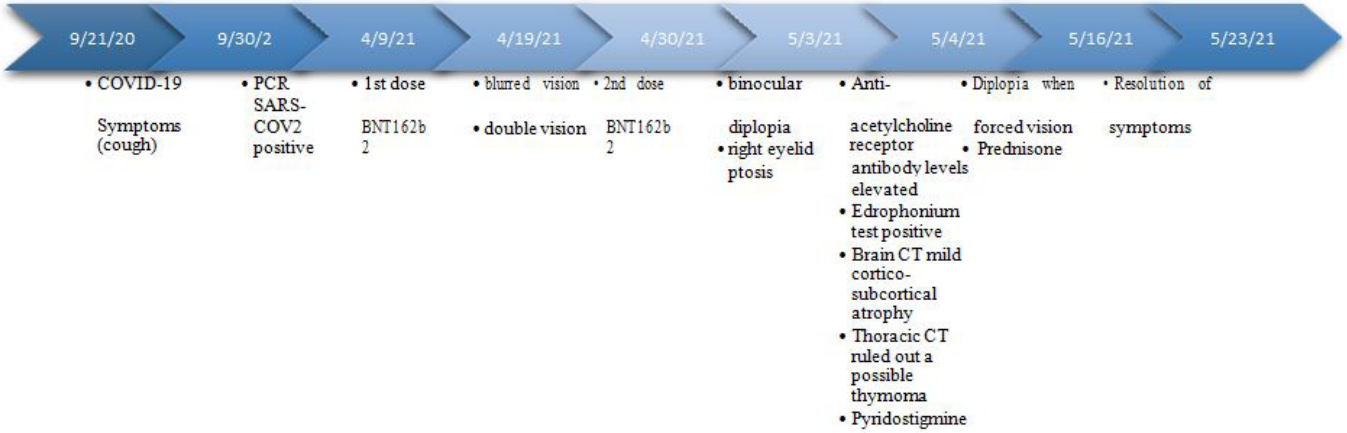

Figure 1: Timeline of evolution of symptoms, test and treatment

Discussion

Autoimmune MG is a neuromuscular junction disease caused by destruction of the acetylcholine receptor on the postsynaptic membrane, mediated by autoantibodies and clinically characterized by skeletal muscle weakness. There are few descriptions of MG debut after viral infections such as varicella-zoster, West Nile virus, and Zika virus. There are also reports of exacerbations after vaccination, Hung Youl Seok reported 1.5% of exacerbations after influenza vaccination in patients with MG [7]. Dan, He showed that the attenuated virus vaccine for Japanese encephalitis induced the presence of ACRH as well as damage neuromuscular plaque with decreased strength in mice to which said vaccine was applied. This is a reasonable hypothesis because in China the prevalence of infantile myasthenia has a higher prevalence [8].

There are reported cases of new-onset MG after SARS-COV2 infection, with a maximum latency of 56 days. In the case of our patient, 167 days had elapsed after the SARS-COV2 infection, a time delay three times longer than those described, which suggests a relationship with the administration of the two doses of vaccine, rather than with the SARS-COV2 infection [2]. Watad recently published a series of 27 cases of outbreaks of immune-mediated diseases after administration of mRNA vaccine. The delay time in the appearance of outbreaks was four days on average, reaching a maximum of 25 days. In this series, two new-onset MG cases are described, both after the second dose of the BNT162b2 vaccine, one of which was severe, the first patient with moderate symptoms had it on the first day after the second dose and the one who had severe symptoms had it on day seven [3]. Tagliaferri published a case of myasthenic crisis that required endotracheal intubation and assisted ventilation after mRNA- 1273 vaccination [5].

In this case, it is a MG that meets diagnostic criteria, except EMG-Jitter, whose normality may be due to the treatment prescribed in the previous weeks. The temporal relationship between vaccination for SARS-COV2 and the appearance of symptoms of ocular MG suggests a possible trigger for neuromuscular pathology, with a possible pauci symptomatic onset of MG after COVID-19 aggravated by vaccination being more unlikely.

The onset of the clinical symptoms exhibited by this patient is correlated with the administration of the vaccine, but causality cannot be definitively demonstrated.

The proposed mechanisms by which an autoimmune disease is triggered by a vaccine include molecular mimicry and bystander activation. In a model of molecular mimicry involving an mRNA vaccine, the immune system can recognize the antigen produced from the mRNA as similar to the host tissue antigen, resulting in the activation of T cells and the formation of antibodies against host tissue, including the acetylcholine receptor. In this case, the bystander activation model may be more likely. In which, the previously existing antigen is released due to stimulation of the innate immune system as part of the response to the vaccine, resulting in the activation of autoreactive T cells [9].

In conclusion, MG is a rare complication of the BNT162b2 COVID-19 vaccine. Its potential severity and the current lack of knowledge of the real incidence after vaccination makes it necessary to maintain an attitude of vigilance in the face of symptoms that suggest it.

Patient Perspective: Patient perspective and consent.

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