

COVID Infection in A Myasthenic Patient (About One Case)

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Abstract

Infection with the novel Sars-Cov2 coronavirus is responsible for a severe form of pneumonia. COVID-19 which can progress to an acute respiratory distress syndrome. However, although the criteria for hypoxemia are present, this SDRA differs from the classic forms, in particular due to pulmonary compliance which is usually normal at the initial stage. This suggests specific pathophysiological mechanisms still poorly understood, which lead to disease profiles that call for rethinking protective ventilation. Symptoms are more severe in people with neuromuscular diseases such as myasthenia gravis already predisposing them to the risk of respiratory distress.

Keywords: Covid 19, myasthenia gravis, auto immune diseases, respiratory failure.

Introduction

Patients with autoimmune diseases (AIDs) typically have an increased risk of infections, which is attributed to the disease itself, but also to immunosuppressive (IS) treatments and comorbidities, SARS cov 2 infection is not an exception. Myasthenia gravis is the prototype for damage to neuromuscular transmission It is caused by the presence of autoantibodies directed against acetylcholine receptors in the motor plate and it's characterized above all by a fluctuating muscle weakness, which is temporarily alleviated by cholinesterase inhibitors. Infections can be a trigger for myasthenic crises, the covid infection being able to be responsible for an acute respiratory distress syndrome can trigger an attack of myasthenia gravis on a weakened ground constituting an additional factor of mortality.

Observation

This is the patient L. M, 66 years old, chronically smoking at 20 packs a year, followed for autoimmune myasthenia gravis for 1 year for which he was operated on 3 months

ago. Symptoms began 10 days before admission with the onset of febrile respiratory distress with headache, cough and severe myalgia. On admission the patient was conscious 15/15 hemodynamically stable TA = 13/8 FC = 110 bpm the patient was polypneic at 20 cpm with a spo2 = 74% in the open air and 93% under MHC 15 L. The patient was Benefiting from a positive return PCR and a thoracic CT showing 40% pulmonary involvement in favor of covid, a complete biological assessment was carried out showing PNN = 8930 lymphocytes = 480 platelets = 236000 TP = 84% fibrinogen = 8.4 urea = 0.42 creatinine = 7 PCT = 7.41 anti Xa activity = 0.8 CRP = 235An echocoeur evaluation found a LV of good c9ntractility with an ejection fraction of 50% without any anomalyThe patient was put on Lovenox 0.8 * 2, decadron 6mg / d, oedes 40 mg, triaxon 2g / d and vitamin supplementation.The evolution was marked by the improvement on the clinical level by the disappearance of the polypnea with decrease in oxygen requirements and passage to O2 glasses and on the biological level by the regression of the markers of inflammation PNN = 5500 Lymphocytes = 1000 platelets = 255000 TP = 75% fibrinogen = 4.2 urea = 0.37 creatinine = 8 CRP = 54 anti Xa activity = 0.72. The patient was discharged and went home.



Thoracic CT showing 40% pulmonary involvement in favor of covid.

Discussion

Myasthenia gravis is an autoimmune reaction resulting in the production of polyclonal antibodies directed against several components of the muscle acetylcholine receptor. Circulating antibodies are found in 60 to 90% of patients with myasthenia gravis and the level of antibodies circulating does not correlate precisely with the severity of the condition. The interaction of antibodies with one or more components of the postsynaptic receptor glycoprotein, with secondary activation of complement modifies the structure of the receptor and causes a drastic decrease in the amplitude of the miniature motor plate

potentials (up to 20% of its value normal). Myasthenia gravis is characterized by muscle weakness, the distribution of which is extremely variable from patient to patient. The onset is often insidious, but the onset of signs can be precipitated by infection, surgery, pregnancy, or even emotional shock. Usually, the extrinsic eye muscles, those of the face, pharynx and neck are the first affected, but occasionally damage to the belts or respiratory musculature occurs early. Muscle weakness is accentuated during sustained effort and typically, it is reduced at rest. In more than 90% of cases, in the state phase, there is palpebral ptosis and involvement of the extrinsic eye muscles. The muscles of facial expression, chewing, swallowing and phonation are affected in about 80% of

cases. The flexors and extensors of the neck, the muscles of the shoulder girdle and the flexors of the thigh are less frequently affected. In more advanced cases, all the muscles are weak, including the diaphragm, the abdominal and intercostal muscles and more rarely the external sphincters. Respiratory abnormalities, even subclinical, can be detected by functional tests. A muscular atrophy of variable importance can be found in approximately 10% of cases, especially in undernourished patients due to oropharyngeal involvement. Despite the muscle weakness, tendon reflexes are preserved and there is no disturbance of sensitivity. Muscle masses are painless. Fasciculations are only found in an overdose of cholinergic substances. Symptomatology groups together a triad which constitutes a diagnostic combination:

- The fluctuating nature of muscle weakness varies within a day, sometimes within minutes or from day to day. Classically, weakness is more marked at the end of the day than in the morning. Longer periods of fluctuations may also occur, corresponding to phases of exacerbation and remission. When the exacerbation begins the respiratory musculature, it is called a myasthenic crisis. This occurs in 10% of patients, and the most exposed are those with oropharyngeal involvement or abnormal respiratory function tests. The attack can be precipitated by a respiratory infection, surgery, drugs that alter neuromuscular transmission (curare, quinine, aminoglycosides, etc.) or a change in treatment. Myasthenic crisis requires monitoring in an intensive care unit, often with assisted ventilation.
- The distribution of muscle involvement is typical. In 40% of patients, the initial involvement is ocular and, as mentioned above, it ends up in over 85% of patients, with a combination of diplopia and fluctuating eyelid ptosis. Mild diplopia often persists, even during periods of remission. The distribution of muscle involvement is usually stabilized within a few weeks or months after the onset of symptoms. Generally, when the damage is limited to the ocular musculature after two or three years, it will remain circumscribed there.
- The clinical response to cholinergic substances results in a dramatic improvement upon injection of neostigmine or edrophonium. In practice, the neostigmine test is carried out by injecting 1.5 to 2 mg combined with 0.4 mg of atropine by the intramuscular route. It induces a clear improvement in twenty

minutes to two hours. The edrophonium test by intravenous administration (first 2 mg followed by a bolus of 3 mg after 15 seconds, then 5 mg) induces an improvement in about 30 seconds but this is brief. This test seems more sensitive for ocular forms but edrophonium is not available in all countries.

Infections are a common trigger for myasthenic exacerbations. Hypoxemic respiratory failure secondary to the virus itself is common, but the course of the disease can also be complicated by myasthenic exacerbation and resulting neuromuscular respiratory failure. COVID-19 poses unique challenges in the assessment and management of patients with MG. Patients with COVID-19 are at risk of developing acute respiratory distress syndrome, requiring high doses of sedative and paralytic drugs for management of their failure, limiting access to neurological examination and potentially increasing the risk of myasthenic exacerbation. In intubated patients tidal volumes can be used as a proxy, with an expected normal value of 5 mL / kg and significantly lower values suggesting a contribution of neuromuscular respiratory failure. In addition to the risk of myasthenic exacerbation of COVID-19, the therapies Experiments of COVID-19 such as azithromycin and hydroxychloroquine may also trigger myasthenic exacerbation. Azithromycin and hydroxychloroquine should probably be avoided or used with caution in patients with MG who may worsen MG. In the absence of COVID-19, the use of tocilizumab has been shown to be safe and effective in the treatment of refractory MG. The management of immunosuppression in patients with MG and COVID-19 is a challenge, there is limited literature on the course and recovery in immunocompromised patients with covid.

Conclusion

Infections can be a trigger for myasthenic crises, the covid infection being able to be responsible for an acute respiratory distress syndrome can trigger an attack of myasthenia gravis on a weakened ground constituting an additional factor of mortality which impose special precautions in the matter of protective ventilation and specific treatments of covid that can cause a myasthenia crisis.

Conflict of interest