MYASTHENIA GRAVIS SECONDARY TO THE USE OF ANTI-TNF - A CASE REPORT

Olívia Regina Lins Leal Teles^{1,*}, Max Luan de Carvalho Aragão¹, Yure Guimarães Azevedo¹, Tatiana Barbosa Castelo Branco¹, José Caetano Macieira¹, Lina Oliveira de Carvalho¹, Regina Adalva de Lucena Couto Ocêa¹, Mônica Valéria Siqueira Santana de Vechi¹

1.Universidade Federal de Sergipe, São Cristóvão (SE), Brazil.

*Corresponding author: oliviaa.teles@gmail.com

BACKGROUND

The participation of anti-TNF in the therapeutic arsenal of immune-mediated rheumatologic diseases is increasing, being an important part in the management of patients with rheumatoid arthritis. Its use is contraindicated in patients with demyelinating diseases, and there are rare reports of myasthenic syndrome developing after its administration. The objective was to describe the case of a patient with rheumatoid arthritis who developed myasthenia gravis after anti-TNF therapy.

CASE REPORT

Female patient, 47 years old, diagnosed with seropositive rheumatoid arthritis for 9 years, made use of several drug associations with methotrexate, chloroquine diphosphate, leflunomide, glucocorticoid and nonsteroidal anti-inflammatory drugs to control the disease, but presented refractory moderate to high activity by DAS-28. In 2016, golimumab was started with a good response. However, 2 years later the patient developed secondary failure, and treatment was then modified to certolizumab pegol which was started on September 2018. Over a period of 4 to 6 months, she developed high dysphagia with nasal regurgitation, bilateral palpebral ptosis that worsened in the afternoon, fatigability of facial expression and visual blurring, without associated diplopia, and tetraparesis with several episodes of falling from her own height, progressive dysphonia, and dyspnea. Physical examination showed strength grade 3 in lower limbs and 4 in upper limbs, and eyelid drooping after external eye movement tests. Laboratory tests showed aldolase of 29 U/L; CPK 62 U/L; positive ACPA and rheumatoid factor; positive ANA 1:640 with homogeneous nuclear pattern and anti-DNA persistently positive; anti-RNP, anti-Sm and anti-SSA were negative; anti-aquaporin and anti-MUSK were negative; acetylcholine antireceptor was positive; increased acute-phase proteins. An electroneuromyography of the limbs showed post-synaptic neuromuscular junction involvement. Chest computed tomography showed absent thymus. Certolizumab was discontinued and pyridostigmine therapy was subsequently started, with progressive improvement of the neurological symptoms and worsening of the joint symptoms. Tocilizumab was introduced 4 months ago with an excellent response and low disease activity.

CONCLUSION

This is a case of rheumatoid arthritis in use of anti-TNF that evolved with myasthenia gravis.

KEYWORDS

Rheumatoid arthritis, Myasthenia gravis, Anti-TNF.