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ANKYLOSING SPONDYLITIS ASSOCIATED WITH KENNEDY'S DISEASE - CASE REPORT

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BACKGROUND

Ankylosing Spondylitis (EA) is an incurable chronic systemic inflammatory disease of unknown etiology and strong genetic predisposition. It is part of the spondyloarthrites group, diseases that mainly compromise the axial skeleton, being an important cause of chronic low back pain. Kennedy's disease (DK), also known as bulb-spinal muscular atrophy, is a rare, autosomal recessive, X-linked autosomal recessive motor neuron caused by an expansion of more than 39 CAG repeats in the androgen receptor gene, leading to a pattern of weakness, atrophy and fasciculations of the appendicular and bulbar muscles, as well as endocrine manifestations (gynecomastia, testicular atrophy and oligopermia). There is no description in the literature of the association between the two diseases. We aim to report the unusual clinical case and the therapeutic approach proposed in a patient with AD and DK.

CASE REPORT

A 40-year-old male patient with gynecomastia and polyiminimioclonias since childhood, evolving after 30 years of age with pain and proximal muscle weakness in limbs, being diagnosed in 2016 through the DK gene test. She presented siblings and maternal cousins with the same pathology.He used Growth Hormone, Testosterone and Leuprorelin for endocrine complaints, suspended due to myositis. Concomitant to the symptoms had pains in the region of sacroiliac and lumbar spine of inflammatory character, with x-ray and magnetic resonance demonstrating sacroiliitis and serum leukocyte antigen test B27 serum positive. Diagnosis of AD started, with non-hormonal anti-inflammatory and sulfasalazine, but there was no clinical improvement or scores of disease activity. After an interdisciplinary discussion, the option was made for the use of anti-TNF-etanercept therapy, but after 6 months of therapy the medication was suspended due to primary failure. Interleukin 17 blockade was then chosen, when significant clinical improvement was observed after 3 months of treatment with seciquinumab, although high scores were maintained (BASDAI dropped from 7.3 to 6.2 and BASFI from 8.2 to 7.3).

CONCLUSION

We present the unusual association of DK and AE, which for the patient showed adequate response perception after the use of seciquinumab, but maintenance of high values of disease activity and functional scores. This may have happened because of the short treatment time and the fact that DK causes muscle weakness and fatigue that can "mimic" inflammatory disease activity. We believe that with the continuity of the treatment the clinical improvement can be accompanied by a significant improvement of the activity scores, even if a high score of functionality is maintained