<https://doi.org/10.47660/CBR.2020.17332>

UNTREATED HYPOTHYROIDISM: A DIFFERENTIAL DIAGNOSIS ON INFLAMMATORY MYOPATHIES - A CASE REPORT

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BACKGROUND

Patients with inflammatory myopathies often present with symmetric proximal muscle weakness and increased muscle enzymes as creatine kinase (CK). As they receive proper treatment, patients experience laboratory and clinical improvement. The same may happen to patients who present with hypothyroidism. This study reports the case of a patient with polymyositis who was being treated with immunosuppressive dose of corticosteroid. She was evolving with worsening of proximal muscle weakness and persistent increase of CK. The diagnosis of hypothyroidism was confirmed. The patient initiated treatment with levothyroxine sodium while weaning corticosteroids, she had clinical improvement and normalized CK levels.

CASE REPORT

A 52-year-old woman with polymyositis, taking 60 mg of prednisone was hospitalized, evolved with hyperglycemia, intense acute thoracic pain, dyspnea and muscle weakness. The clinical picture started progressively and symmetrically. During admission of the patient, the hypothesis of acute coronary syndrome and corticosteroids-induced hyperglycemia arose. She had depressive disorder, no history of skin rashes, sicca symptoms and Raynaud' phenomenon. Physical exam showed thick and dry skin, diffuse alopecia on the scalp and on 1/3 of the eyebrows. The size of the thyroid gland was not increased and there were no palpable nodules. The muscle strength grade was, and the palpation of muscle groups caused pain. The tendon reflexes were normal. The results of the medical examinations showed elevation of creatine phosphokinase and creatine phosphokinase MB (CPK-MB), normal troponin and no suggestive alterations of ischemia noted on the ECG. The x-ray revealed bilateral pleural effusion and the echocardiogram confirmed subtle pericardial effusion without hemodynamic repercussions. Laboratory: Normocytic normochromic anemia, increased serum lipids levels with cholesterol level of 225 mg/dL and triglycerides levels of 240 mg/dL. The rheumatoid factor test results were negative, the levels of complements were normal, TSH 22, T4 0.6. The patient was diagnosed with hypothyroidism, the treatment with Levothyroxine sodium was initiated and the dosage was gradually increased, the weaning of corticosteroids was completed and after six weeks she recovered the muscle strength, the pleural effusion, the pericardial effusion and the depression symptoms disappeared, furthermore, her glycemic levels were controlled.

CONCLUSION

This case study concludes that hypothyroidism must be included in differential diagnosis of patients with inflammatory myopathies. It is extremely important to perform thyroid function analysis on these patients considering that both hypothyroidism and inflammatory myopathies result in an increase of CK and muscle weakness. The differential diagnosis changes therapeutics and prognosis.