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CASE REPORT

Myositis as a presenting manifestation of severe hypothyroidism: A case report and review of the literature

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Introduction

Hypothyroidism is a common disease and is associated with symptoms from several organ systems. This includes musculoskeletal manifestations, however, the precise mechanism of muscular disease is unclear, although cross reactivity of anti-thyroid autoantibodies or auto-reactive T cells with other organs may play a significant role^[1, 2].

William M. Ord described the first case of muscle weakness with hypothyroidism in 1880 and subsequently it has been reported in several cases^[3]. Hypothyroid induced myositis is an inflammatory myopathy characterized by musculoskeletal symptoms which can be confirmed through increased serum level of muscle enzymes such as creatine kinase (CK) (up to 10 times normal), nonspecific electromyographic (EMG) abnormalities and pathological findings in muscle biopsies^[1-4]. Frequent symptoms include fatigue, proximal muscle weakness, myalgia, tenderness, and stiffness^[3-6]. Necrosis, inflammatory infiltrates, type II fiber atrophy, increased numbers of internal nuclei, and “core-like” structures in type I fibers are common findings in muscle biopsies^[3, 7]. Treatment of these patients consists of administration of thyroid hormone and in some cases also

corticosteroids. It can take several months for the muscle symptoms to resolve^[1, 2].

The authors report a case of severe hypothyroidism in a 70-year-old man with myositis as the initial feature.

Case report

A 70-year-old man was presented to the department of rheumatology in June 2014 with a history of 4 months fatigue, dry skin, myalgia, and muscle weakness especially in hip and knee joints. He had previously been able to run 10-15 km a day and do 100 pushups exercise without any problem, but during the last few weeks he could only walk 200 m and would stop walking due to excessive fatigue. The patient was diagnosed with Waldenström macroglobulinemia, a low-grade B-cell lymphoproliferative disorder^[8], in 2009 and treated with Fludarabine, Cyclophosphamide and Rituximab. He had not received diuretics, tyrosine kinase inhibitors and any other medications except laxatives due to constipation problem. On physical examination, the patient was alert and had normal temperature. His blood pressure was 171/86 mmHg, pulse and respiratory rates were 62/min and 12/min respectively.

The patient had muscular pain on palpation. There was a marked periorbital edema, as well as edema of upper extremities and repealed tendon reflexes. Examination of thyroid showed an enlarged thyroid gland. The laboratory evaluations results identified: hemoglobin: 8.1mmol/L (normal range, 8.3-10.5mmol/L), lymphocyte: $0.67 \times 10^9/L$ (normal range, $1.00-4.00 \times 10^9/L$), Potassium: 3.7 mmol/L (normal range, 3.5-4.4mmol/L), alanin aminotransferase (ALAT): 72U/L (normal range, 10-70U/L), creatine kinase: 4560U/L (normal range, 40-280U/L), myoglobin: 735 μ g/L (normal range, <77 μ g/L), lactate dehydrogenase (LDH): 1198U/L (normal range, 115-255U/L), thyroid stimulating hormone (TSH): $31.7 \times 10^{-3}U/I$ (normal range, $0.30-4.0 \times 10^{-3}U/I$), T4: <6 nmol/L (normal range: 65-140 nmol/L), and T3: <0.5nmol/L (normal range, 1.45-2.5 nmol/L). Patient was negative for anti-thyroid (anti TPO) antibodies and TSH-receptor antibodies (TRAbs). CT scan of pelvis, thorax, abdomen and throat were performed due to suspicion of another malignancy or activation of Waldenstrom macroglobulinemia. These investigations revealed no abnormal findings. Ultrasonographic evaluation of the thyroid gland demonstrated reduced echogenicity and a 17 mm node with calcifications. Subsequent cytology from fine needle aspiration was benign. Immunological analyses were negative for SS-A and SS-B (Sjögren syndrome) antibodies, Smith's antibody, Ribonucleoprotein antibody, Topoisomerase 1 antibody, Histidine-tRNA-ligase antibody and Histon antibody. Biopsy of quadriceps was performed under ultrasonographic guidance. Histological examination demonstrated chronic changes as a sign of atrophy and presence of minimal phagocytes infiltration. Fiber typing showed both type 1 and 2 fibers, however with a marked atrophy of type 2 fibers. Staining for neural cell adhesion molecules (N-CAM) and neonatal myosin were highly positive for N-CAM and weakly positive for neonatal myosin. There was no sign of myofibrils disorganization in the oxidative staining. Nerve conduction velocity (NCV) and EMG was normal. Treatment with levothyroxine 100-150 μ g/d gradually lead to resolution of symptoms and after three months follow-up the patient could perform his habitual level of exercise. Reducing levothyroxine to 75 μ g/d resulted in recurrence of musculoskeletal symptoms. Thus, levothyroxine dosage was increased to 100 μ g/d again whereupon symptoms disappeared.

Discussion

Myositis or inflammatory myopathies are categorized into four types: dermatomyositis (DM), polymyositis (PM), necrotizing myopathy (NM) and inclusion body myositis (IBM). In all groups muscle weakness is a typical sign. Diagnosis can be achieved by thorough clinical examination, laboratory evaluation such as CK and autoantibodies, EMG

and muscle biopsy^[9].

Several musculoskeletal symptoms such as pyrophosphate arthritis^[10], carpal tunnel syndrome^[11], rotator cuff tears^[12], muscle pseudohypertrophy (Hoffman's syndrome)^[13], and myositis^[2, 3, 5, 14] have previously been related to hypothyroidism. A report by Cabili *et al*^[14] described several cases having increased muscle enzymes and proximal muscle weakness. These patients were diagnosed as having polymyositis and interestingly, one patient was treated with prednisone 40mg/day for a long time. Additional laboratory testing however, disclosed patients were hypothyroid and the authors concluded that hypothyroidism should be considered in all patients with unexplained myopathy and clinicians should always perform thyroid function test in such cases.

In another study, Madariaga *et al*^[15] reviewed all patients with Polymyositis-like syndrome from 1975 to 2000. Thirty two cases were included in the study. Common clinical manifestations were: weakness (100% of the patients), delayed tendon reflexes with slow relaxation phase (41%), muscle tenderness (25%), and muscle induration (9%). The mean of CK and TSH were 2164 ± 1954 U/L (± 1 SD) and 114.8 ± 85.6 mIU/L (± 1 SD) respectively. EMG showed non-specific myopathic changes in a half of the patients. Eighty percent of the patients had undergone muscle biopsy. The most frequent findings in muscle biopsies were: necrosis, inflammatory infiltrate, atrophy of type II fibers, hypertrophy of type I fibers, alteration in the amount of type I and type II fibers, central nuclei disposition and core-like structures. The authors suggested that in the presence of increased serum CK and myopathy serum TSH level should be checked routinely.

This case story illustrates a patient, used to conduct relatively heavy muscular exercise diagnosed with hypothyroidism. The primary symptoms of the hypothyroidism were two musculoskeletal syndromes namely myositis and rhabdomyolysis. His symptoms of muscle weakness and pain disappeared completely after treatment with levothyroxine. After recovery the patient was able to perform his regular muscular exercise without problems. To the best of our knowledge, this is the first case of hypothyroidism presenting simultaneously with myositis and rhabdomyolysis in the English literature. We suggest that thyroid function should always be tested in patients with muscle weakness.

Conflicting interest

The authors declare that there are no conflicts of interest.

In the preparation of this paper all authors fulfilled the

criteria of authorship. All were involved in the study design, gathering the data, writing the paper, etc. all authors read and approved the final manuscript.

Abbreviation

CK: creatine kinase; EMG: electromyography.

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