A CASE OF PRIMARY HYPOTHYROIDISM PRESENTING AS MYOPATHY

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Abstract:
In hypothyroidism, symptoms such as muscle cramps, pain, and stiffness are common but myopathy as a predominant feature is an uncommon presentation. We describe a patient with primary hypothyroidism who presented with a clinical picture of myopathy that normalized after 20 weeks of therapy with levothyroxine. Keyword: Hypothyroidism, hypothyroid myopathy, levothyroxine, electromyography

INTRODUCTION:
Myopathies may be secondary to electrolyte disturbances, endocrinopathies or drugs such as steroids, lipid lowering agents, colchicine, chloroquine, zidovudine, alcohol etc. Hypothyroidism may manifest as muscle weakness with or without pain. The severity of hypothyroid myopathy ranges from an asymptomatic disease with elevation of muscle enzymes to a disease with prominent muscle weakness. Neuromuscular manifestations are seen in 30–80% of hypothyroidism cases during the course of the disease, but myopathy as the predominant manifestation of hypothyroidism is a rare presentation. We hereby present a 45 year old female with hypothyroidism who presented with muscle weakness.

CASE REPORT:
A 45 year old female presented with the complaints of difficulty in walking and lifting her upper limbs for the past 6 months and inability to hold her head erect for the past 2 months. These symptoms were progressively worsening. She had mild cramps in her limbs. She did not have any distal weakness of upper and lower limbs. There was no history of sensory or autonomic involvement. There was no diurnal variation in her symptoms. She gave history of facial puffiness for the past 6 months. She also gave a history of constipation. There was no history of fever, joint pain or skin rash. There was no past history of any chronic illness, drug intake or surgeries. She had attained menopause 2 years back.

On examination, the patient had a hoarse voice, puffy face, pallor, macroglossia (figure 1) and ichthyosis in the lower limbs (figure 2). There was no neck...
swelling. Her pulse rate was 70 beats/minute and blood pressure 110/70 mm Hg in the upper limb. Examination of cardiorespiratory system was normal. Per abdomen examination revealed divarication of recti. Nervous system examination revealed normal bulk and decreased tone of her limbs. There was neck muscle weakness along with proximal muscle weakness of both her upper and lower limbs. Ankle jerk showed delayed relaxation (positive Woltman’s sign) while other reflexes were normal. Rest of

Investigations revealed: hemoglobin 10.7 g/dl, total count 6600, platelets 1 lakh, ESR 12 mm at 1 hour, RBS 96 mg/dl, blood urea 22 mg/dl, s.creatinine 0.8 mg/dl, sodium 134 meq/l, potassium 4.4 meq/l, ELISA for HIV 1,2 non reactive, T.cholesterol 224 mg/dl, TGL 171 mg/dl, HDL 46 mg/dl, LDL 150 mg/dl, VLDL 28 mg/dl. Peripheral smear revealed normocytic normochromic anemia with normal WBCs and platelets. Urine routine was normal, ANA and RF were negative. S.calcium was 9 mg/dl. Electrocardiogram and ultrasound abdomen were normal. Chest xray revealed left sided eventration of diaphragm (figure 3). Echo showed normal LV systolic function with mild pericardial effusion without any evidence of pericardial tamponade. Her muscle enzymes were markedly elevated, CPK was 1014 u/l (39-238). Her thyroid profile implied primary hypothyroidism: T3 < 10 ng/dl (60-200), T4 < 0.3 g/dl (4.5-12), TSH > 150 IU/ml (0.3–5.5). Anti TPO antibodies were within normal limits – 13.8 IU/ml (0-34).

Electromyography showed the following: normal insertional activity, no abnormal waves in spontaneous activity (figure 4), low amplitude motor unit potentials (37 V) (figure 5) and interference pattern showing complete recruitment with low amplitude (figure 6). These features were suggestive of a myopathic pattern. Her nerve conduction study was normal.
She was started on levothyroxine therapy 100 g/day. After around 4 weeks of therapy, patient noticed improvement in her muscle power. She was able to hold her head steady. At this stage, CPK was 417 u/l, TSH – 67.43 lu/l. The dose of levothyroxine was stepped up to 150 g/day. Over the next 4 months, she gradually regained her full muscle power. Repeat investigations revealed normal enzymes (CPK 152 u/l) and a normal thyroid profile: TSH – 10.2 lu/l, T3-141 ng/dl, T4- 10.74 g/dl. Muscle biopsy was deferred because patient did not give her consent as her symptoms started improving.

DISCUSSION:
Patients with hypothyroidism have frequent muscle complaints such as pain, stiffness and cramps. These manifestations can occur at any time in the disease process. Hypothyroid myopathy typically manifests as polymyositis-like myopathy with proximal muscle weakness and an increased creatine kinase level. Likewise, our patient also presented with a proximal muscle weakness with elevated creatine kinase levels. However, it sometimes manifests as muscle enlargement (pseudohypertrophy); in adults, this condition is called Hoffman syndrome and in children, Kocher-Debre-Semelaigne syndrome. Other neuromuscular manifestations such as delayed relaxation of deep tendon reflexes (Woltman's sign) can be present in 25% of patients; carpal tunnel syndrome and pseudomyotonia may be seen.

The prevalence of hypothyroidism in patients with myopathy is difficult to estimate but in a study group among 53 patients with acquired muscle diseases, the incidence of hypothyroidism was found to be 5.6 per cent. Muscle involvement may be caused by (1) changes in muscle fibres from fast twitching type II to slow twitching type I fibres, (2) deposition of glycosaminoglycans, (3) poor contractility of actin–myosin units, (4) low myosin ATPase activity, (5) low ATP turnover in skeletal muscle,
6an autoimmune reaction causing chronic thyroiditis, hypothyroidism, and polymyositis, (7) or an involvement of the muscle membrane.

The serum CPK level is often elevated (up to 10 times normal), even when there is minimal clinical evidence of muscle disease. Marked elevation up to the range of 20,000-25,000 U/L may be seen5. The serum enzymes may rise to very high levels, presumably because thyroid deficiency permits leakage across muscle membranes and possibly due to actual muscle necrosis6.

EMG findings in hypothyroid myopathy may be normal or myopathic. The motor unit action potentials (MUAP) are generally polyphasic with reduced amplitude and duration. Early recruitment may also be observed. Our patient's EMG showed low amplitude potentials with complete recruitment. However, there is no correlation between the clinical weakness, electric myopathic pattern, CPK levels and thyroid hormones7. NCS is usually normal or may show features suggestive of mild neuropathy. Our patient had a normal nerve conduction study. Biopsy of the muscle usually shows nonspecific changes. Fiber atrophy is noted with increased numbers of internal nuclei, glycogen aggregates, and deposition of mucopolysaccharides in the connective tissue.

Treatment of myopathy associated with hypothyroidism is levothyroxine replacement. Myopathy improves within 2-3 weeks, but may take months to resolve completely. Our patient took approximately 20 weeks to normalize. The creatine kinase levels return to normal levels and the EMG findings too revert with treatment8.

Hypothyroid myopathy is an uncommon presentation of hypothyroidism, which is reversible with levothyroxine. Hence, hypothyroidism and similar treatable conditions should always be considered in the differential diagnosis of patients presenting with myopathic complaints.

REFERENCES:
1. Harrison’s Principles of Internal Medicine, 18th edition.


