

Hypothyroidism simulating as polymyositis

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Abstract

Polymyositis-like syndrome in hypothyroidism is a rare condition characterised by proximal muscle weakness and elevated muscle enzymes. Patients with this condition can initially be misdiagnosed as having polymyositis due to similar characteristics of both diseases; however a response to thyroxine is the main differentiating feature. This report highlights the case of a 30-year-old male who had severe myalgia and proximal muscle weakness. In addition to raised creatinine phosphokinase (CPK) levels, his biochemical profile showed hypothyroidism. Initially thought to be suffering from polymyositis, improvement in both clinical and biochemical profile with thyroxine led to the diagnosis of polymyositis-like syndrome associated with hypothyroidism.

Keywords: Polymyositis-like syndrome, Hypothyroidism.

Introduction

Hypothyroidism is a common medical condition that classically presents as weight gain, cold intolerance, hoarseness of voice and bradycardia. Neuromuscular manifestations ranging from delayed relaxation of deep tendon reflexes to full-blown myopathy usually occur in conjunction with other more common systemic features of the disease but in some cases they may be the presenting feature.¹

Hypothyroid myopathy typically presents as progressive symmetric weakness of shoulder and hip girdle muscles.² However in few cases it can follow a more fulminant course characterised by marked serum muscle enzyme elevations that virtually resembles polymyositis.³⁻⁵ Coexistence of clinical signs and symptoms of hypothyroidism, absence of spontaneous fibrillation potentials along with normal motor units on electromyography (EMG) studies, lack of inflammatory changes on muscle biopsy and clinical as well as biochemical response to thyroxine help to distinguish polymyositis-like syndrome from classic polymyositis.

Case Report

A 30-year-old male presented in May 2014 with complaints of gradually progressing muscle weakness and myalgia for one year, with the muscle weakness symmetrically involving

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Figure-1: Before treatment.

the proximal muscles. He had no history to suggest any hereditary, infectious or drug-related cause. On examination, he had generalised body swelling with a pulse of 65 beats per minute. There were no skin lesions or thyromegaly. Tenderness was present on musculoskeletal examination, and powers were 3/5 in all proximal muscles. He had no atrophy, fasciculations or sensory abnormality. His reflexes were difficult to elicit because of generalized body swelling but were found to be grossly normal. Laboratory investigations results were as follows: haemoglobin 10 gm/dl; mean corpuscular volume (MCV) 100.5fl; aspartate transaminase (AST) 59; creatinine phosphokinase (CPK) 11,000 IU/L (10-190); EMG studies showed myopathic pattern; meanwhile biopsy of his left deltoid showed



Figure-2: After treatment.

Table: Comparison of BMI, TSH and CPK levels before and after treatment.

	Before Treatment	After Treatment
BMI (kg/cm ²)	27.5	23.9
TSH (mIU/L)	50	10
CPK (IU/L)	11,000	116

mononuclear cellular infiltrate. Anti-Jo-1 antibodies and anti-signal reduction particle (SRP) antibodies surprisingly came out to be negative. Thyroid profile showed thyroid stimulating hormone (TSH) levels to be 50 mIU/L (0.5-4.7); free thyroxine (FT4) to be 2.4pmol/L (10-23); and free triiodothyronine (FT3) to be 1.3 pmol/L (3.5-6.5). Anti-thyroglobulin and anti-thyroperoxidase antibodies were also positive. The patient was started on thyroxine (50µg once daily). After six weeks of therapy, dramatic improvement in clinical, biochemical and histological profile was noted with normalization of CPK and resolving phase of polymyositis on re-biopsy (Figure-1 and 2), (Table).

Discussion

Painful proximal weakness with extremely high levels of CPK, and absence of delayed reflexes were the factors pointing away from endocrine myopathy giving an initial impression of polymyositis. However generalised body swelling and bradycardia provided a suspicion of hypothyroidism which was later on proved by deranged thyroid function tests.

Wide spectrum of musculoskeletal abnormalities can occur in hypothyroidism including myalgias, Hoffman syndrome and polymyositis-like syndrome.³⁻⁶ Polymyositis-like syndrome is a rare form of hypothyroid myopathy in which there is marked elevation of muscle enzymes. Although a two to six-fold rise in muscle enzymes have been seen in hypothyroid myopathy, they may also rise to very high levels in polymyositis-like syndrome, making it difficult to differentiate it from inflammatory myositis. EMG studies may be normal in half of the patients or may show non-specific myopathic changes.³ Muscle biopsy may show variation in size of muscle fibers, mild focal necrosis and occasionally mild inflammatory infiltrates in contrast to polymyositis, in which inflammation, necrosis and regeneration predominates.³ Although EMG studies and muscle biopsy may be helpful, clinical and biochemical response to thyroxine is the ultimate clue to diagnosis.

Conclusion

Thyroid function should be routinely evaluated in patients with painful proximal muscle weakness and raised CPK levels. Failing to do so may result in misdiagnosis and treatment of these patients as polymyositis instead of polymyositis-like syndrome associated with hypothyroidism, due to similar characteristics of both diseases.

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