CASE REPORT

Thyrotoxic Neuropathy- An Under Diagnosed Condition

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SUMMARY

Many neurological diseases like myopathy, periodic paralysis, ophthalmoplegia, and myasthenia gravis are known associations of thyrotoxicosis. However the association of neuropathy with thyrotoxicosis is not frequently recognized. First described by Charcot in 1889, thyrotoxic neuropathy or 'Basedow's Paraplegia' is a rarely reported entity. We describe here a case of a young woman with subacute distal neuropathy as the presenting manifestation of thyrotoxicosis. The neuropathy improved on antithyroid treatment. A careful literature search leads us to believe that peripheral neuropathy in thyrotoxicosis is under recognised. Thyroid function tests can be helpful in the diagnosis of this treatable neuropathy and should be included in the routine work up.

KEY WORDS:
Thyrotoxic neuropathy, Basedow's Paraplegia, Neuromuscular complications of hyperthyroidism, Idiopathic peripheral neuropathy.

INTRODUCTION

A number of neurological diseases like acute and chronic myopathy, periodic paralysis, ophthalmoplegia, and rarely myasthenia gravis are known associations of thyrotoxicosis. However, neuropathy in thyrotoxicosis is not frequently recognized. First described by Charcot [1] in 1889, thyrotoxic neuropathy or Basedow's Paraplegia is a rarely reported entity. Its existence has often been questioned. We report a young woman with subacute motor-sensory neuropathy as the main presenting manifestation of thyrotoxicosis. Both thyrotoxicosis and neuropathy improved on antithyroid therapy.

CASE REPORT

A 45-year-old woman presented with progressive weakness of both lower limbs and slowly increasing swelling on the anterior aspect of the neck for three months. Other than loss of 3 kg of weight and mild fatigue, she did not have any signs of hypo or hyperthyroidism. Past medical, family, occupational and personal histories were non-contributory. Examination revealed a diffuse goiter of 5cm diameter, marked lower limb weakness (3/5, distal > proximal), mild distal weakness in upper limbs (4/5), moderate muscle atrophy, and absent deep tendon reflexes in lower limbs with mild sensory loss to touch, vibration and pain below the knees. There was no evidence of vitamin deficiency, malnutrition, skin or eye changes, or signs of hypo or hyperthyroidism.

Investigations including blood counts, ESR, electrolytes, glucose, calcium, phosphate, liver and renal functions,

Table I: Nerve Conduction Studies

	Patients values	Normal Values	
		Admission	4 weeks later
Motor NCV			
1. Lt CP			
• Velocity	38-65 (m/s)	43.6	52.7
• Amplitude	3.5-10 (mV)	1.22	4.2
2. Lt PT			
• Velocity	>41(m/s)	42.2	45
• Amplitude	>3 (mV)	5	5
3. Lt Ulnar			
• Velocity	45-75 (m/s)	58.2	64.8
• Amplitude	6-15 (mV)	6.32	9.68
4. Lt Median			
• Velocity	48-70(m/s)	57.4	62.6
• Amplitude	8-15 (mV)	8.33	12.25
Sensory NCV			
1. Lt Sural			
• Velocity	38-55 (m/s)	38.5	43.7
• Amplitude	>6mcV	20.4	22.6
2. Lt median			
• Velocity	42-75 (m/s)	41.7	52.7
• Amplitude	> 20mcV	17.6	28.9

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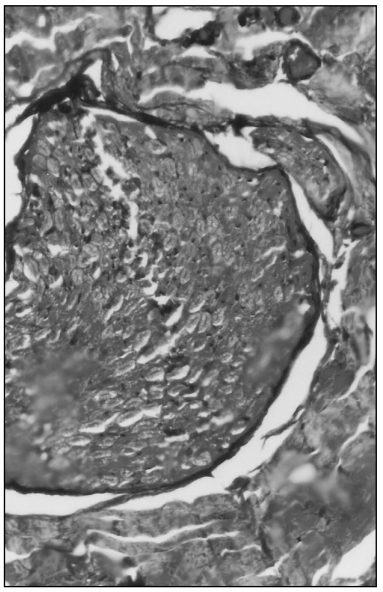

Fig. 1: Nerve biopsy showing diffuse decrease in small myelinated axons with focal increase in endoneural collagen without any inflammatory infiltrate or vasculitis or loss of myelin. These findings are suggestive of small axon axonopathy.

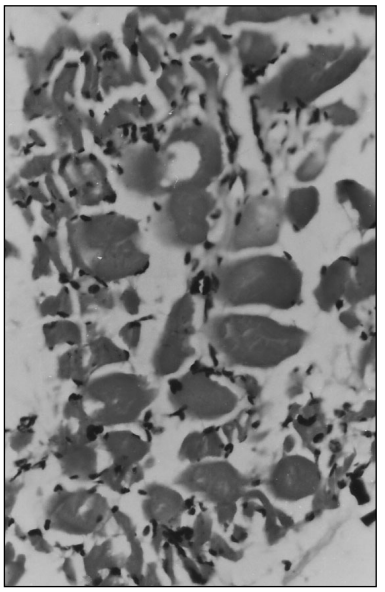

Fig. 2: Biopsy specimen from tibialis anterior muscle. This shows maintained fascicular architecture with variation in size of muscle fibres with a few type1 hypertrophic fibres. There are Type 1 and 2 atrophic fibres. No ragged red fibres are seen. Findings suggestive of neurogenic atrophy.

creatinine phosphokinase levels, urine analysis were normal. Nerve conduction studies revealed distal symmetrical axonal neuropathy (Table I). Electromyography showed fibrillations and occasional fasciculations with normal insertional activity suggestive of neurogenic pattern. Nerve biopsy showed diffuse decrease in small myelinated axons with focal increase in endoneural collagen without inflammatory infiltrate or vasculitis or loss of myelin suggestive of small axon axonopathy (Figure 1). Biopsy from tibialis anterior muscle revealed maintained fascicular architecture with variation in size of muscle fibers with a few type 1 hypertrophic fibers and atrophic fibers of both types without ragged red fibers suggestive of neurogenic atrophy (Figure 2). Vitamin B12, angiotensin converting enzyme levels and serum protein electrophoresis were normal. Antinuclear antibodies, C & pANCA, anti SSA, and anti topoisomerase were negative. Urine was negative for porphobilinogens. Thyroid function tests revealed thyrotoxicosis (TSH= 0.024 mU/L, Free T4 = 7.18 ng/dl, Free T3 = 9.6 pg/ml). USG thyroid and thyroid scan showed toxic multinodular goitre.

Antithyroid therapy (carbimazole and propranolol) was started and within four weeks a biochemical improvement (TSH= 0.5mU/L, Free T4 = 1.7ng/dl) and significant improvement in muscle strength was noted. Deep tendon reflexes became elicitable and response to sensory stimuli improved. Repeat nerve conduction studies after one month showed improvement, thus confirming our diagnosis of thyrotoxic neuropathy.

DISCUSSION

The patient here presented with subacute polyneuropathy of three months duration. The only other clinical abnormality was goiter. The absence of any other identifiable cause of neuropathy except thyrotoxicosis, and the response to anti thyroid treatment strongly suggests the diagnosis of thyrotoxic neuropathy.

Though it is not a well-recognized entity, we believe that peripheral neuropathy due to thyrotoxicosis is not very rare. Literature supports this hypothesis. In studies done on hyperthyroid subjects by Ludin *et al*[2] (13 patients), Sozay *et al*[3] (17 patients), and Duyff *et al*[4] (prospective study with 21 patients) neuropathy was seen on electrophysiological testing in 65.5%, 35.5%, and 19% of patients respectively. Most of the patients with neuropathy were asymptomatic. Sozay *et al*[3] and Ludin *et al*[2] concluded that electrophysiological studies could be useful in diagnosing asymptomatic polyneuropathy in hyperthyroid patients. Duyff *et al* [4] observed that neuropathy had a very good response to antithyroid treatment. It has also been suggested that 'thyrotoxic myopathy' is actually a neuropathic disorder in its early stages of denervation[5]. This has been shown by McComas *et al* who found neuropathic changes in 20 patients with 'thyrotoxic myopathy'[5].

The pathogenesis is unclear. It may be due to the direct effect of thyroid hormone, immune mediated, or due to the hypermetabolic state depleting nerve of essential substances. Our patient had goiter as an indication of possible hyperthyroidism. Not all patients with hyperthyroidism would have that. The patients may present only with neuropathy. Thyroid function tests can be helpful in the diagnosis of this treatable neuropathy and should be included in the routine work up.

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