Thyrotoxicosis or hyperthyroidism is the clinical syndrome caused by an excess of circulating free thyroxine and free triiodothyronine or both. It is not an uncommon disease, affecting about 2% of women and 0.2% of men. Grave’s disease occurs in about 3-5% of the patients with myasthenia gravis and about 1% of the patients with Grave’s disease developed myasthenia gravis. These associations are of interest in view of the frequent association of thymic enlargement with Grave’s disease. Furthermore, antibodies and T-cells specific for receptors are involved in the pathogenesis of the two diseases. Myopathy affects men with thyrotoxicosis more commonly than women. The effect of both thyrotoxicosis and its alleviation on the course of myasthenia gravis is variable, but in the majority of instances, myasthenia is accentuated during the thyrotoxicosis state and improves when a normal metabolic state is restored. Abnormalities of the thyroid function (hyper/hypo thyroidism) may increase myasthenia weakness (1).

A 34-year-old male, presented to the neurological OPD with complaints of diminution of vision and weakness of all the four limbs – 1 year (Fig-1). Patient was apparently well a year before, when he started with diminution of vision during the day and increased vision during the night and weakness of all the four limbs, initially upper limbs and later the lower limbs, predominantly proximal muscle weakness. Patient had weakness while getting up from sitting position and raising arm above head. On examination, patient had prominent eyes (proptosis), wide palpebral fissure, tachycardia (110/mt) and diffusely enlarged thyroid and tremors of the outstretched hands. On CNS examination, patient had normal higher functions, cranial nerves were intact and fundus examination were also normal. Motor power was Grade II in proximal muscles and Grade IV in distal muscles with intact DTR. Sensory system was normal. Various lab investigations were done. Haemogram and routine biochemical tests were normal. Thyroid function test were done, wherein T₃ was 305.7 (60-181 ng/dl normal); T₄ 23.6 (4.5-10.9 mg/dl) and TSH < 0.03 (0.5-4.7 mU/ml). FNAC thyroid was suggestive of hyperplastic goitre. Muscle biopsy was normal. EMG of deltoid, biceps, quadriceps muscles were performed and showed a myopathic pattern. A final diagnosis of thyrotoxic myopathy with myasthenia gravis was made and patient was put on Tab. Carbimazole 10 mg TDS and Tab. Neostigmine 15 mg TDS. Patient developed respiratory muscle weakness and was put on ventilator in ICU for more than 1½ months. Over this period patient improved, kept off ventilator and could breath normally and carry on his daily activities.
Clinically detectable chronic muscle weakness is described in up to 50% cases of thyrotoxicosis (2). Weakness as a symptom of thyrotoxicosis was reported by both Grave’s (3) and Von Basedow (4). The spectrum of muscle weakness in hyperthyroidism essentially encompasses four disorders – thyrotoxic myopathy; thyrotoxic periodic paralysis; extraocular muscular involvement (thyroid associated orbitopathy); and disorder of neuromuscular junction (myasthenia gravis). Among these disorders, the generalized form of thyrotoxic myopathy is the commonest, the disorder that is most directly linked to thyroid dysfunction and also the most responsive to the normalization of thyroid function.

Thyrotoxic myopathy is quite common, seen in about 60-80% of subjects with thyrotoxicosis (5, 6). Common presenting symptoms include fatigue, weakness and cramps (7). Very characteristically the disease involves the pectoral and girdle muscles. The iliopsoas and the quadriceps are among the commonly involved muscles and often the weakness is not extremely severe enough to cause a total paralysis (8). In these muscle groups, weakness and wasting is often noticed and this is usually symmetric. More interesting is the role of respiratory muscle involvement in thyrotoxicosis. Dyspnoea is a common complaint in thyrotoxic subjects seen in > 80% of subjects (9). Dyspnoea could arise from cardiac failure, increased ventilatory drive, airway resistance, decreased lung compliance, tracheal compression by goitre or respiratory paralysis. It has been reported that diaphragmatic muscle weakness could be an important factor contributing to breathing difficulties in these subjects. Recently also few authors (10, 11) similarly reported concurrent Graves disease and myasthenia gravis and suggested that treatment of one reveal the other (11). Carbimazole therapy led to significant improvement in muscle weakness.

Thyroid disorder may be seen in as many as 10% of patients with myasthenia gravis and symptoms of hyperthyroidism or hypothyroidism may be present. Weakness of bulbar muscle is a prominent feature of myasthenia gravis. In 20% of cases, myasthenia gravis affects bulbar muscles alone. Respiratory weakness may be present and respiratory failure occurs in 1% of patients.

References


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