

***Thyrotoxicosis with Myasthenia Gravis-ICU Management*****Kiran N, Priyadarshini Arvind, Ravi M*****Department of Anaesthesiology and Critical care,******Sri Devaraj Urs Medical College, Kolar*****ABSTRACT**

*A 30 year old patient , known case of hyperthyroidism, developed respiratory insufficiency ,was intubated and put on ventilator . Myasthenia gravis was confirmed with tensilon test and acetylcholine receptor antibody titres positive. A final diagnosis of thyrotoxic myopathy with Myasthenia gravis was made. Anticholinesterase treatment makes it difficult to achieve euthyroid state in a patient with Myasthenia and hyperthyroidism. Cholinergic crisis should not be confused with worsening of Myasthenia gravis. If a see-saw effect is elicited before undertaking treatment of hyperthyroidism one must decide which is worse, uncontrolled hyperthyroidism or Myasthenia gravis.*

**Keywords:** *Myasthenia gravis, anti-cholinesterase, hyperthyroidism, see-saw effect*

**INTRODUCTION**

Thyrotoxicosis or hyperthyroidism is the clinical syndrome caused by an excess of circulating free thyroxine and free tri-iodothyronine or both, hyperthyroidism affects 2% of women and 0.2% of men. Graves disease occurs in about 3-5% of patients with Myasthenia Gravis and 1% of patients with Graves developed Myasthenia. Myasthenia accentuates during thyrotoxicosis and improves when a normal metabolic state is restored. <sup>[1]</sup> Symptoms of Myasthenia gravis and hyperthyroidism can be precipitated by stress.

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**CASE REPORT**

A 30 year old male patient presented to the casualty with chief complaints of difficulty in breathing since 1 month which increased since 2 days, generalised weakness since 6 months, difficulty in speaking and swallowing since 4 months, intermittent blurring of vision since 2 months. History of difficulty in breathing was gradual in onset aggravated on doing work and in the evening and relieved on taking rest, which increased since 2 days. There was generalised muscular weakness, he could not walk far without stopping and resting frequently. His arms fatigued rapidly on combing his hair. These symptoms were worse in the later part of day. He felt at his best in the morning after a night's rest. Difficulty in swallowing food was mainly to solids and he became easily fatigued when chewing or swallowing food and he would have to pause several times during a meal. The intermittent double vision recurred on and off,



frequently objects would drop out of his hands. His appetite was poor and he lost about 10 kgs weight in the last 6m. He had noticed prominence of the eyes, often felt nervous and suffered palpitations.

His condition deteriorated progressively. He had been on carbimazole therapy 10mg OD for treatment of hyperthyroidism from 6 months. There was nothing significant in the past history. Family history was negative for thyroid or muscular disease. Patient was conscious, oriented, dyspneic and emaciated. Eye signs of hyperthyroidism were positive. He was unable to close his eyes tightly against resistance, but there was no ptosis. He could not gaze upwards or sideways for long. Nystagmus was absent and pupils reacted normally. Thyroid gland was enlarged and a solitary nodule was palpable. There were fine tremors of the outstretched hand. Deep tendon reflexes were bilaterally absent and plantars were flexor. On examination : patient was afebrile ; Pulse rate=120-140/min, regular; Blood Pressure=130/80 mmHg; Respiratory rate=40-50/min; Cardio vascular system and Respiratory System were normal. Haemogram, ECG, serum electrolytes, blood sugars, renal functions, coagulation parameters were within normal limits. Thyroid function test values were as follows T3=1.17ng/ml(0.7-2); T4=3.33mg/dl(4.5-11); TSH = < 0.015 mIU/ml (0.4 - 4.2) ; Pseudocholinesterase = 5918U/L(4850-12000); Cerebro Spinal Fluid analysis was normal, was done to rule out Guillain Barre Syndrome syndrome; Muscle biopsy was normal; Electromyogram of deltoid, biceps, quadriceps showed myopathic pattern ; Acetylcholine

receptor antibody titre was 1.42nmol/l (Normal value is <0.05nmol/l). Radiological examination of chest showed no abnormality in superior mediastinum to suggest an enlarged thymus. CT chest was performed to rule out tumour of thymus. Since he had inadequate respiratory efforts he was intubated and connected to ventilator. Since TSH was low, euthyroidism was achieved clinically with increased dose of carbimazole (10mg TID).

The exophthalmos then decreased slowly with carbimazole therapy and the ptotic eye became prominent. Co-existent myasthenia was suspected and test dose of 0.5mg neostigmine was given intravenously and the extra ocular muscle power improved. A final diagnosis of Thyrotoxicosis with Myasthenia Gravis was made and patient was then started with Inj. Neostigmine i.v 2.5mg TID for 3 days with Inj. glycopyrrolate 0.4mg, Tab. Pyridostigmine 30mg QID and Tab. Carbimazole 10mg TID was continued. Gradually the respiratory muscle power improved and we were able to wean him off the ventilator.

## DISCUSSION

When thyrotoxicosis with myasthenia gravis co-exist they should be regarded as parallel phenomenon since the evidence does not suggest that either of these conditions causes the other.<sup>[3,7]</sup> When thyrotoxicosis and myasthenia prevail at the same time the prognosis is extremely poor and treatment is beset with great difficulty. Symptoms of myasthenia can be at a less prominent when hyperthyroidism was more and myasthenia symptoms can be aggravated as



hyperthyroidism subsided.

Medications used for treatment of hyperthyroidism like methimazole, corticosteroids like prednisolone, muscle relaxants and betablockers to be used with caution as they may aggravate the symptoms of Myasthenia gravis.<sup>[8]</sup> Single Fibre EMG is thought as a the gold standard for diagnosis of Myasthenia Gravis.<sup>[9]</sup>

When there is evidence of a see-saw balance between the two conditions and therefore it can be suggested that the two diseases are mutually antagonistic that is aggravation of Myasthenia following improvement of the thyrotoxicosis.<sup>[2,3,4,5]</sup> There are also reports displaying the combination of the two diseases in which the Myasthenia became worse after thyroidectomy.<sup>[2]</sup>

Thymectomy can be done first followed by thyroidectomy, because the risk of precipitating a thyroid crisis by thymectomy may be less than the risk of myasthenic crisis following thyroidectomy.<sup>[3]</sup>

Thymectomy should be considered in patients in whom an anterior mediastinal shadow can be diagnosed by X-ray or in patients who remain incapacitated by Myasthenia inspite of adequate medical management.<sup>[3]</sup>

## CONCLUSION

It is shown that anti-cholinesterase treatment makes it difficult to achieve euthyroid state in a patient with Myasthenia gravis and hyperthyroidism. Thyroid disorder may be seen in 10% of patients with Myasthenia gravis and the symptoms of hypo/hyperthyroidism may be present.<sup>[5]</sup> One must be careful not to confuse a

cholinergic crisis with worsening of Myasthenia Gravis. If a definite see-saw effect is elicited then before undertaking permanent treatment of hyperthyroidism one must decide which is worse uncontrolled hyperthyroidism or Myasthenia Gravis.

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