



EXPLORING HYPERTHYROIDISM WITH A NEUROLOGICAL OUTLOOK

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Dear Editor:

Being one of the most common endocrinal disorders, the thyroid diseases having an extended spectrum from subclinical hypothyroidism to life threatening thyroid storm. Hyperthyroidism a hypermetabolic state with excess circulating thyroid hormones present as common systemic features like weight loss, palpitation, heat intolerance as well as peripheral and central nervous system manifestations.^[1] Few of the initial neurological symptoms are often misdiagnosed as psychiatric disorders.

Cognitive impairment with features of encephalopathy like seizures, confusion & even dementia complicates or associated with hyperthyroidism at times respond dramatically to beta blockers with other specific drugs.^[2] The hyper adrenergic state secondary to hyperthyroidism is responsible for such features. Fine action tremors exhibiting on out stretched hands or even head or extremities are the most common movement disorder again responding well to beta blockers like propranolol. Chorea, choreoathosis and myoclonus may also be part of movement disorder spectrum.^[3]

Anxiety, irritability, agitation, emotional liability and even depression are the usual neuropsychiatric manifestations of increased sympatneic stimulation. Atrial Fibrillation (AF) due to underlying hyperthyroid state is responsible for cardioembolic cerebral infarction in 10-15% cases. Similarly hypercagulable state due to increased factor VIII activity with elevated fibrinogen and decreased protein C activity predispose to thyrotoxicosis induced cerebral venous thrombosis with high morbidity & mortality.^[4]

Peripheral nervous system is another site of affection by high circulating thyroid hormones and may present as sensory, distal, symmetrical polyneuropathy to more pronounced Guillian-Barre syndrome & Chronic

inflammatory demyelinating polyneuropathy in 5-10% untreated hyperthyroid individuals. An uncommon but specific "Basedow's" paraplegia with features of neuropathy and areflexia presenting as acute manifestation of severe thyrotoxicosis is also described.^[5]

Of the various sites of peripheral nervous system, the key areas of thyroid dysfunctions are muscles and myoneural junctions. Myopathy (mostly proximal) with or without myalgia is seen in 50-70% of long standing untreated hyperthyroid or thyrotoxicosis patients. This chronic progressive myopathy which is more common in males, after the age of 40 results due to imbalance between myofibril synthesis & its degradation, decreased muscle carnitine levels, increased cellular metabolism & energy utilization.^[6] Muscle atrophy and raised creatine Kinase levels are not the usual features. Similarly deep tendon jerk may even be increased due to shortened relaxation phase of muscle contraction. Electromyography may be normal or myopathic. Treatment of hyperthyroid condition and attainment of euthyroid is sufficient to treat the myopathy.^[7]

Thyrotoxic Periodic Paralysis, an acquired form of hypokalemic periodic paralysis triggered mostly by sternous exercise or high carbohydrate diet is encouraged with higher frequency in Asian males in their second to fourth decades.^[8] A symmetrical ascending weakness without respiratory, bulbar or ocular muscle involvement correlates with decrease in serum potassium levels. Increased Na^+K^+ ATPase pump activity along with high catecholamine & insulin levels result in massive shift of potassium into the cells. Adequate replacement of potassium along with beta blockers reverses the paralysis in the order of its appearance. TPP is rare in pediatric population.^[9] Due to shared auto immunity via HLAB & HLADQ3 and genetic susceptibility, an epidemiological link exists between hyperthyroidism (Particularly Grave's disease) and ocular form of

myasthenia gravis (MG). Autoimmune thyroid disease is more commonly associated with ocular MG reflecting the immunological cross reactivity with target eye muscles.^[10] The overlap semiology of Graves ophthalmopathy and ocular MG can be distinguished clinically as ptosis and orbicularis oculi weakness suggest MG while proptosis, lidlag, lid retraction, peri orbital odema with restricted ocular movements suggest grave's disease. The association of thyrotoxicosis in myesthenic patients does not affect the modalities of treatment and prognosis.^[11]

Thus new onset or deteriorating neurological states should raise a suspicion of underlying hyperthyroid state as prompt correction & attainment of euthyroid state coupled with beta blockers in many situation can resolve the issues.

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