

Thyrotoxic neuropathy - a case report

Sayan Malakar¹, Kailash Nath Sharma², Abhinav Rana³, Katyayni Dutt¹, Tarun Sharma⁴

From ¹Junior Resident, ²Professor, ³Senior Resident, ⁴Assistant Professor, Department of Medicine, Dr. Rajendra Prasad Government Medical College and Hospital Tanda, Kangra, Himachal Pradesh, India.

Correspondence to: Dr. Tarun Sharma, Department of Medicine, Dr. Rajendra Prasad Government Medical College and Hospital Tanda, Kangra, Himachal Pradesh - 176001, India. E-mail: tarunpgi@gmail.com

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ABSTRACT

Thyrotoxic neuropathy is a rare entity in literature. The association between thyrotoxicosis and neuropathy is under-recognized. We here present a rare case report in which patient was presented with ascending sensory-motor paralysis coupled with respiratory muscle weakness which closely resembles Guillain-Barré syndrome (GBS). But relevant history suggested thyrotoxic features, thus a timely focused investigation revealed the diagnosis. It was confirmed in nerve conduction studies (NCS) and other necessary investigations ruled out other differential diagnosis. Patient was treated with anti-thyroid drugs. On follow up patient's power improved and NCS after 6 months came out to be normal which established the diagnosis. Thyrotoxic neuropathy is a close differential diagnosis of LGBS and other commonly encountered neuroparalytic illnesses. So high degree of suspicion is needed to diagnose this potentially treatable neuropathy.

Keywords: GBS, Neuropathy, Quadriplegia, Thyrotoxicosis.

Thyrotoxic neuropathy also known as Basedow's Paraplegia [1] is rarely reported entity. It was described for the 1st time by Charcot in 1889 [2] and has not been very well-recognized entity in literature. Various neurological diseases are associated with hyperthyroidism or thyrotoxicosis like myopathy, periodic paralysis with hyperthyroidism and hypokalemia, ophthalmoplegia and myasthenia gravis. The association between thyrotoxicosis and neuropathy is under-recognized. Though rare, thyrotoxic neuropathy is a treatable condition. It mimics very common diagnosis like Guillane Barre syndrome (GBS) thus a strong clinical suspicion is needed to establish the diagnosis. Here we present a case of 47-years-old female who was presented to our department with the complaint of weakness in bilateral lower limb.

CASE REPORT

A 47-year-old female patient presented in the emergency with the complaint of weakness in bilateral lower limb for 1 day followed by weakness of bilateral upper limbs for 10 hours. The patient was well before she was presented with abrupt onset of weakness in bilateral lower limbs simultaneously. For initial few hours, patient could walk with support but had a weak grip of the floaters that she wore. After 12 hours of initial weakness, the patient became completely bed-ridden. She was unable to change posture on bed as well as sit up from supine position. Over next few hours, she felt her bilateral upper limb getting weaker initially in the form of holding cups followed by inability to comb her hair within hours.

She experienced difficulty in raising head and had no drooping of eye lids.

The weakness wasn't followed by any high carbohydrate meal, fever, neck pain, diarrheal illness, vaccination, dog bite, neck trauma, nausea, vomiting or any history suggestive of raised intra cranial tension. Patient also complained of tingling sensation of bilateral feet and inability to perceive hot and cold temperature in bilateral legs upto knee. There was no history suggestive of any cranial nerve involvement, autonomic involvement, or any toxin exposure like mercury, arsenic. There was overflow incontinence. The patient didn't give any past history of similar episodes though she suffered unintentional weight loss since last 5 months (from 62 kg to 47 kg in 5 months). The patient also experienced palpitation and chronic diarrhea for last 4 months.

On examination, her vitals were normal with tachycardia (heart rate of 124 per minute, regular) and body mass index (BMI) 15.3 Kg/m². Higher mental functions were normal. Her Glasgow coma scale was 15/15 and Mini mental state examination of 30/30. Examination of cranium, spine, and speech was normal. Examination of cranial nerves and autonomic nervous system was also normal Fig. 1. On sensory examination; pain, touch and temperature were impaired in lower limbs up to knee bilaterally. There was generalized areflexia with hypotonia. Power was 0/5 bilateral lower limb, 2/5 bilateral upper limbs. There were no fasciculations and bilateral plantars were flexor. Patient was catheterized. Differential diagnosis kept was GBS, toxic neuropathy, human immunodeficiency virus (HIV) associated neuropathy, acute transverse myelitis .

Table 1: Motor Nerve Conduction Study

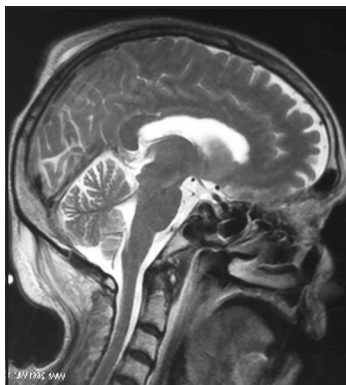
NERVE	LEFT		RIGHT	
	Wrist	elbow	Wrist	elbow
Median	3.5 (high)	7.8	4.2	8.7
Amplitude	6.0	2.6	1.4 (low)	1.5
Ulnar	2.5	7.0	3.1	7.3
Amplitude	6.2	4.8	3.5 (low)	3.0
Peroneal	8.7	13.7	4.1	9.6
Amplitude	180(low)	290	650	400
Tibial	4.5	13.4	4.0	11.8
Amplitude	1.7 (low)	900	4.3	3.8

As there was bladder involvement possibility of GBS was slightly lower though there were typical ascending quadriplegia. Complete hemogram (hemoglobin 10.5 gm%; Total leukocyte count: 11500/uL with normal differential count; platelets: 276000/uL), liver function tests (Bilirubin: 1.2 mg/dL, albumin: 4.1 g/dL, alanine transaminases: 35 U/L; aspartate transaminase: 43 U/L; alkaline phosphatase: 112 U/L) and renal function tests (urea: 18 mg/dL; creatinine: 0.9 mg/dL) tests were normal including electrolytes (Sodium 135 mEq/L and Potassium 3.9 mEq/L). Her CPK was also normal 165 U/L (normal: 51-294 U/L).

Magnetic resonance imaging (MRI) cervical spine and NCS were done. MRI cervical spine was normal while that of NCS was suggestive of motor>>>sensory predominantly axonal polyneuropathy. In view of tachycardia, unintentional weight loss and chronic diarrhea, thyroid function test were done which revealed high free T3 (64; normal - 3.7 – 6.5 pmol/L), and free T4 (88; normal - 9-16 pmol/L) with suppressed TSH levels (0.001; normal - 0.34-4.25mIU/L).

Cerebro-spinal fluid (CSF) study after a week revealed no abnormality. There was no albumino-cytological dissociation while antinuclear antibodies (ANA), HIV 1/2, HbsAg and Anti HCV were negative.

On day 2 of admission, patient had respiratory depression and her respiratory rate was 8/min and she was unable to maintain saturation. She was incubated and started on carbimazole (30

**Figure 1: normal MRI brain****Table 2: Sensory Nerve Conduction Study**

Nerve Amplitude (µV)	Right	Left
Median	12.2 (low)	35.7 (low)
Ulnar	22.5 (low)	38.4 (low)
Sural	6.7 (low)	3.2 (low)

mg twice daily), propranolol (60 mg 4 hourly). Patient clinically improved and was extubated on day five. Relevant investigations are detailed in table 1 and 2.

The patient was followed up in neurology OPD after she was discharged on anti-thyroid medications. On follow up after 6 weeks, her power improved in all the 4 arms. Repeat NCS was done after 6 months which came out to be normal which accompanied normalization of free T3, T4 and TSH. It confirmed the final diagnosis of neuropathy associated with thyrotoxicosis. Nerve biopsy wasn't done as patient didn't give the consent for it.

DISCUSSION

Thyroid hormone has an important role in early growth and development of a child. Hyperthyroidism is mainly associated with a thyroid adenoma or Grave's disease, which would be accompanied by any other autoimmune illness. In addition to the physical symptoms associated with hyperthyroidism, a heterogeneous group of neuro-psychiatric symptoms and syndromes may occur [3,4]. Thyrotoxic periodic paralysis is not a very uncommon disorder that is manifested as recurrent episodes of hypokalemia and muscle weakness lasting from hours to days. This information is important as this clinical condition is particularly relevant to us in India, the incidence of the disorder is relatively higher among Asians [5].

Neuromuscular junction disorders like myasthenia gravis may be seen in a proportion of patients with hyperthyroidism. Patients with myasthenia gravis have an increased incidence of thyroid disorders; and 5.7% of myasthenic patients are found to be hyperthyroid [6]. Apart from these, in patients with hyperthyroidism, 80% have neuromuscular related complaints and more than half patients have marked muscle weakness [7]. Women predominate at 3:1 to 4:1, and the mean age of onset is fifth decade. Weakness is primarily proximal and is usually out of proportion to the amount of muscle wasting; distal weakness develops later and is less severe than the proximal myopathy. Myalgia, fatigue, and exercise intolerance are common. Rarely inflammatory myopathy may occur in thyrotoxicosis which necessitates corticosteroid therapy [8].

Thyrotoxic neuropathy or Basedow's Paraplegia is a rarely reported entity. Its existence has often been questioned. Though cases with polyneuropathy and hyperthyroidism have been reported in literature [9,10]. The pathophysiology of neuropathy in hyperthyroidism is still obscure. It has been postulated to be either a direct effect of excessive thyroid hormones, immune-mediated or due to a hypermetabolic state depleting the nerves of essential nutrients [11].

In our patient, we considered all the possibilities of acute lower motor neuron quadriplegia along with acute transverse myelitis

with spinal shock. GBS was ruled out according Brighton criteria and patient improved after successful treatment with carbimazole without plasmapheresis or intra venous immunoglobulin and her CSF was also normal. MRI cervical spine came out to be normal, serum electrolytes were also normal. Patient was negative for HIV1, & 2, CytoMegalo virus, Epstein Barr virus, angiotensin converting enzyme and ANA. Her CPK and electromyography were normal. Finally, thyroid function test and antithyroid peroxidase antibodies and successful treatment with antithyroid drugs clinches the diagnosis.

A prospective study on hyperthyroid patients found that 14% had numbness and paresthesia and 19% had signs of distal sensory disturbances in the limbs with depressed ankle jerks. Electrophysiological findings on them confirmed predominantly sensory axonal neuropathy among 24%; moreover, with treatment of the hyperthyroidism, the sensory symptoms resolved within seven months [11]. It has also been postulated that ‘thyrotoxic myopathy’ is actually a neuropathic disorder in its early stages of denervation. This has been shown by McComas et al who showed neuropathic changes in 20 patients with ‘thyrotoxic myopathy’ [12].

CONCLUSION

Thyrotoxic neuropathy is a close differential diagnosis of LGBS and other commonly encountered neuroparalytic illnesses. Though it is rare but it can be picked up by simple history talking and thyroid function tests and can be confirmed by NCS after ruling out common diseases. A high degree of suspicion is needed to diagnose this potentially treatable neuropathy.

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