CASE REPORT

Reversible and unilateral corticospinal tract disease secondary to autoimmune free-T3-thyrotoxicosis

Carlo Canepa,¹ Ramalingam Srinivasan,² Abdul Muhith³

¹Stroke and Neurology, James Paget University Hospital, Norwich, UK

²Department of Endocrinology and Diabetes, James Paget University Hospital, Norwich, UK

³Department of Gastroenterology, James Paget University Hospitals NHS Foundation Trust, Great Yarmouth, UK

Correspondence to

Dr Carlo Canepa, Neurocanepa@gmail.com

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SUMMARY

68-year-old female patient with no significant medical history presents with a 3-month history of progressive neurological symptoms, which began with left eye ptosis, blurred vision and non-painful jaw discomfort, followed by left spastic weakness and hyper-reflexia with positive Babinski and Hoffman signs. An elevated T3 level, a positive peroxidase and an antigraves antibody level led to an ultrasound, which confirmed a sub acute-chronic autoimmune thyroiditis. A nerve conduction studies/electromyogram showed normal motor and sensory velocity conduction with a small amplitude compound motor action potential, indicative of likely axonal damage. Following treatment with carbimazole, the neurological symptoms greatly improved. The authors concluded that the left pyramidal syndrome was secondary to autoimmune free T3-thyrotoxicosis.

BACKGROUND

- It describes a very rare case of unilateral and reversible corticospinal tract (pyramidal tract) involvement secondary to autoimmune, free T3-thyrotoxicosis.
- To our knowledge, this is the first case report of unilateral pyramidal involvement secondary to T3 without T4 involvement. All previously reported cases described bilateral involvement with free T3 and T4 elevations.
- It exemplifies the importance of thinking of possible metabolic/hormonal causes of neurological manifestations.

CASE PRESENTATION

This is a 68-year-old female patient with no significant medical history and no regular medication use, who was admitted to our hospital following a 3-month history of progressive neurological symptoms, which began with left eye ptosis, blurred vision and persistent non-painful discomfort on the left side of her face, involving the forehead, ear, nasal groove and the left side of her jaw. There was neither right eye ptosis nor facial asymmetry. Approximately 1 week later, she began to notice weakness in her left upper and lower limb, more noticeable when trying to use small objects and when walking. She felt a tendency to veer towards the left when accelerating her stride. Ten days later, she began to present horizontal diplopia and to feel a 'pressure-like' sensation in the left eye on forced gaze towards the left. Soon after, she began to present dysphonia without dysphagia or

odynophagia. These symptoms led to an evaluation by her general practitioner who diagnosed her with viral conjunctivitis and rhinosinusitis. Treatment with nasal sprays and ocular drops were prescribed for 7 days without any improvement.

As time progressed, the weakness of both the upper and lower left limb intensified, yet no weakness was noted on the right side. Non-painful paresthesias were noted on the left side of her face and body, without involvement of the trunk. These symptoms led her to feel unstable when walking and tripping over on several occasions. She also began to notice a subtle resting and intentional tremor in both hands, associated with episodes of light-headedness, feeling sweaty and cold and having palpitations (without angina). No loss of consciousness was reported. These symptoms finally led to her admission.

On admission, sitting-down blood pressure was 160/74 mm Hg and her standing blood pressure (after 3 min) dropped to 118/59 mm Hg. Heart rate was consistently in sinus tachycardia (between 90–110 bpm), respiratory rate 18–20 and oxygen saturation between 93% and 95% consistently. Blood glucose was normal throughout admission. On physical examination, she was awake, alert in time, space and person. There were no signs of acute delirium, aphasia or amnesia. A subtle resting and intention tremor was noted in hands. No goitre or lymph nodes were noted on palpation of the neck. Left ptosis was noted, and both pupils were equal and reactive. There was no right-sided ptosis. Voluntary, conjugate horizontal gaze was limited due to pain on left conjugate eye movement with associated inability to abduct the left eye. No conjunctiva erythema was noted in either eye. No facial muscle weakness was noted. No lower cranial nerves were affected. Mild pain on palpation of the left sternocleidomastoid muscle was noted; however, no pain in the posterior aspect of the neck was observed. Strength in the upper limb was 3/5 proximally and 2/5 distally; the lower left limb was 4/5 throughout. Strength in upper and lower limb was 5/5. There was also ipsilateral hypertonicity. Perception for pinprick, temperature and pain was preserved in all four limbs. Reflexes were brisk (+++/++) in left acromioclavicular, brachial, radial, cubital and patella, with ipsilateral Hoffman and Babinski sign. No clonus was noted. Reflexes were normal on the right, except for a dubious extensor plantar reflex. There were no signs of cerebellar dysfunction; Romberg sign was negative. Gait was ataxic with left lateropulsion. No fasciculation were noted.

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Table 1 Complete blood count

Test	10 February 2017	10 February 2017	6 June 2017	19 April 2017	25 April 2017	Normal range
White cell count	11.5×10 ⁹ /L		10.2	9.3	9.6	4–10
Haemoglobin	151.0 g/L		144	145	146	120–150
Platelet count	239×10 ⁹ /L		211	249	272	150–410
Haematocrit	0.480		0.452	0.451	0.451	0.36–0.46
MCV	92.2 fL		95	95.8	95.3	83–101
ESR	33 mm/h					0–30
CRP	4 mg/L	4	7			0–10
Neutrophils	5.52×10 ⁹ /L		6.16	4.46	4.45	2–7
Basophils	0.07×10 ⁹ /L		0.5	0.07	0.07	0.0–0.1
Eosinophil	0.42×10 ⁹ /L		0.29	0.48	0.46	0.02–0.5
Lymphocytes	4.57×10 ⁹ /L		2.29	3.35	3.97	1.00–3.00
Monocytes	0.95×10 ⁹ /L		0.79	0.76	0.60	0.2–1.0
Sodium	141 mmol/L		143	142	139	134–145
Potassium	4.2 mmol/L		4.1	4.8	4.6	3.6–5.0
Urea	4.7 mmol/L		4.6	4.8	4.2	1.7–7.1
Creatinine	63 µmol/L		67	69	79	45–84
GFR		84		77	65	
Bilirubin	11 µmol/L	6	11	6	9	0–22
Alkaline phosphatase	110 U/L	121	130	167	177	38–126
Alkaline transaminase	10 U/L	13	11	14	17	0–50
GGT				91 U/L	124	0–60
Total protein	63 g/L	74	62	77		63–82
Albumin	31 g/L	33		36	33	35–50
Globulin	32 g/L	41		41	39	23–35
Calcium	2.18 mmol/L					
Adjusted calcium	2.34 mmol/L					
Phosphate			1.02 mmol/L			
Folate				>40 µg/L		2.7–15.0
Vitamin B12				564 ng/L		130–1100
Ferritin				109.4 µg/L		23.0–300.0
Prothrombin time			13.0			10.7–14.1
Prothrombin time control			11.5			
APTT			35			22–36
APTT control			29			
APTT ratio			1.2			
Fibrinogen			5.57			2.0–4.0
INR			1.11			
Cholesterol				4.0 mmol/L		3.6–5.0
HDL cholesterol				1.1 mmol/L		0.9–2.0
Cholesterol:HDL ratio				3.6		
Non-HDL cholesterol				2.9 mmol/L		

APTT, activated partial thromboplastin time; CRP, C reactive protein; ESR, erythrocyte sedimentation rate; GFR, glomerular filtration rate; GGT, gamma-glutamyl transferase; HDL, high-density lipoprotein; INR, international normalized ratio; MCV, mean corpuscular volume.

A CT angiogram done due to the history and presenting symptoms ruled out dissection or aneurysm. A brain MRI was performed with a dedicated study of the trigeminal nerve, which did not reveal infarction, haemorrhage or intrinsic trigeminal nerve pathology. A cervical spine MRI did not reveal disc protrusions or nerve compression. Subsequently, blood investigations were requested to rule out metabolic or systemic, autoimmune or infectious causes. The complete blood count was normal (table 1). No renal or hepatic abnormalities were noted. Sodium, potassium, magnesium, phosphate and calcium were also normal. All blood and urine cultures were negative on two occasions. The thyroid results (table 2), however, were abnormal: thyroid-stimulating hormone (TSH) was low (<0.01 mU/L), T3 was high (7.8 pmol/L) and free T4 was

normal (20.7 pmol/L). Antithyroid peroxidase antibody was very high (306.4 kU/L). All other antibodies (N-methyl-D-aspartate receptor antibody (NMDA), Lgi-1, voltage gated potassium channel (VGKC), anti-Hu, La, Ro and anticholinesterase) were normal. A lumbar puncture was performed, which did not reveal any cerebrospinal fluid abnormality (no protein: cytology dissociation was noted). Based on these results, the patient was started on carbimazole 10 mg once daily and discharged with physiotherapy and a follow-up in endocrinology and neurology.

1 week after her discharge, a nerve conduction study was performed showing normal motor sensory velocity and reduced compound motor action potential (CMAP), indicative of an axonal lesion.

Table 2 Immunology and thyroid function

Test	7 March 2017	19 April 2017	25 April 2017	Normal range
TSH	<0.01 mU/L	<0.01	<0.01	0–10
Free T3	7.8 pmol/L	4.7	4.6	3.8–6.0
Free T4	20.7 pmol/L	10.7	10.2	7.5–21.1
Antithyroid peroxidase antibody	306.4 kU/L		33.8.2	0.0–34.0
TSH receptor antibody	6.10 IU/L			0.00–1.00
Acetyl choline receptor antibody	<0.23 nmol/L			<0.5
Antineuronal nuclear antibodies (Hu/Ri)	Negative			
Antipurkinje cell antibodies (Yo)	Negative			
NMDA receptor antibodies	Negative			
Anti-Lgi-1 antibodies	Negative			
Tissue transglutaminase			2.0 U/mL	0–10
Antinuclear antibodies (ANA)			0.3	0–1.0

NMDA, N-methyl-D-aspartate; TSH, thyroid-stimulating hormone.

Approximately 1 month after her discharge, a thyroid ultrasound was performed, showing a normal-sized thyroid gland with a diffusely hypoechoic and a mildly heterogeneous echotexture. These findings were in keeping with sub acute-to-chronic thyroiditis. A repeat blood test (table 2) showed a mild improvement of her thyroid function with a normal free-T3 (4.7 pmol/L), a normal free-T4 (10.7 pmol/L) and a suppressed THS (<0.01 mU/L). Week after, a third test was conducted, revealing a persistently elevated antiperoxidase (338.2); lymphocytes were mildly elevated ($3.97 \times 10^9/L$) as were the alkaline phosphatase (177 U/L) and the gamma-glutamyl transferase (124 U/L).

Four weeks later, she was re-evaluated in the neurology clinic. Her physical examination revealed mildly brisk reflexes (+++/++) in upper limbs and normal patellar and Achilles tendon reflexes with a flexor planter reflex, and no Hoffman sign on either side. There was no spasticity in either upper or lower left limbs and strength was 5/5 throughout. No sensory disturbance or cerebellar dysfunction was noted. Both eyes had complete range of movement without pupillary abnormalities. There was a mild left ptosis. The dose of Carbimazole had been reduced to 5 mg once daily because of the mild abnormality in her liver function tests. Her blood pressure was 110/65 mm Hg and her heart rate was 91 bpm. There were no clinical signs of hyperthyroidism and finally, she did not report any new symptoms. She declared that she was feeling very well and that she had noticed a marked improvement since her discharge. She was given a follow-up appointment for neurology in 6 months and another for endocrinology in 3–4 months.

DIFFERENTIAL DIAGNOSIS

- ▶ Motor neuron disease (amyotrophic lateral sclerosis, in particular);
- ▶ Primary lateral sclerosis;
- ▶ Cervical spine stenosis +/- with medullary involvement;
- ▶ Neoplastic and paraneoplastic syndromes;
- ▶ Medullary thyroid carcinoma with spinal metastasis.

TREATMENT

- ▶ Carbimazole 10 mg once daily, following by a reduction to 5 mg once daily due to mild increase in liver function tests;
- ▶ Physiotherapy as inpatient and as an outpatient;
- ▶ Baclofen 10 mg twice daily for muscle spasticity and pregabalin 75 mg per reasons necessary (PRN) for neuropathic pain.

OUTCOME AND FOLLOW-UP

She was on follow-up in our neurology and endocrinology clinics, demonstrating great improvement following treatment with carbimazole: her pyramidal syndrome practically disappeared entirely and her thyroid function test improved as well. She will continue to be seen in our clinics for follow-ups and we will also be requesting a second nerve conduction study in 6–8 months to evaluate for neurophysiological improvements. Furthermore, we will also be requesting peroxidase levels in 3–4 months.

DISCUSSION

Undoubtedly, thyroid dysfunction (both hyperthyroidism and hypothyroidism) can cause a wide range of neurological complications,¹ including, among others, myopathies,² periodic paralysis,³ neuromuscular diseases^{4,5} and peripheral neuropathies.⁶ As early as 1889,⁷ Charcot recognised the thyroid–peripheral nervous system link, as he described a case of paraplegia associated with thyrotoxicosis, consistent of areflexia and flaccid paraplegia without signs of upper motor neuron involvement. Joffroy,⁸ in 1894, labelled the condition as Basedow's Paraplegia. More recently, Sattler⁹ and Feibel¹⁰ described patients developing paraparesis/paraplegia induced by thyrotoxic-polyneuropathies.

On the other hand, however, upper motor neuron involvement, manifested as a combination of hyper-reflexia, spasticity, clonus and Babinski reflex (pyramidal syndrome) in thyroid disease has only been scarcely reported. One of the earliest reports comes from Mussio-Fournier *et al*¹¹ and Muxi *et al*¹² who described two cases of simultaneous development of typical hyperthyroidism and a bilateral pyramidal tract syndrome. The former reported a patient with paraparesis, generalised hyper-reflexia, bilateral Babinski signs and atrophy of the quadriceps. The former presented a patient who in addition to a pyramidal syndrome developed severe muscular wasting, dysphagia and pronounced asthenia. Both authors claimed that their patients completely recovered following treatment of hyperthyroidism.

Ravera *et al*¹³ also presented two cases of Graves's disease with signs of pyramidal lesion and improvement following anti-thyroid treatment. The first case had typical signs of Graves disease consistent of exophthalmos, bilateral Graefe's sign, diffuse goitre, tachycardia, cardiovascular instability, palpitation and tremor of the fingers. Neurologically, there was hypotonus in lower limbs, increased reflexogenic area for the knee jerks and brief clonus of both patellae and bilateral Hoffman and Babinski

signs. The ankle jerks were brisk and there was prolonged bilateral ankle clonus. There were no signs of peripheral neuropathy or of ataxic gait. All cranial nerves were intact. Two months after starting Tapazole, the patient had only partially improved. After 1 year of treatment, when the patient was euthyroid, strength returned to normal, reflexes improved (ankle jerks still brisk and no clonus of the foot or the patella). The Babinski and Hoffman signs were normal. The second case presented bilateral nystagmus and exophthalmos with a firm, painless, diffuse goitre, with an intense bruit and thrill. There was generalised loss of strength, no fasciculation or atrophy, incomplete patellar clonus on the left and a Babinski response bilaterally. Barre sign could be elicited. Propylthiouracil (PTU) was prescribed. Only a few days after starting treatment, there were signs of improvement. Approximately 3 months later the dysphagia had resolved, reflexes had improved and the strength had greatly resolved. The EMG revealed an increase in the number of polyphasic motor units, suggesting the possibility of thyrotoxic dystrophy of the muscles.

Garcia and Fleming¹⁴ described a 42-year-old man with spastic gait, wasting and weakness of lower limb muscles, generalised hyper-reflexia (except for a normal jaw jerk) with bilateral extensor plantar reflexes, bilateral Hoffman and ankle clonus; no fasciculation or sensory abnormalities were noted. The thyroid gland showed an enlarged gland with diffuse, uniform increase of tracer activity (including a region of the thyroglossal duct). After diagnosing Graves's disease treatment with methimazole was started. Six months later, the thyroid function returned to normal and the patient's neurological condition had improved greatly.

All of the previous cases presented symptoms in common: bilateral corticospinal tract malfunction, Babinski sign, absent fasciculation/fibrillations on EMG, response to treatment of hyperthyroidism with almost complete neurological recovery. No patient presented involvement of posterior columns, peripheral nerves or muscles.

Our patient presents similar features with all of the previous cases; however, it is (to our knowledge) the first description of unilateral corticospinal tract involvement secondary to T3-thyrotoxicosis. More so, although our patient presented abnormal T3/T4/TSH results, there was telltale clinical signs of typical Graves's disease: no exophthalmos, lid retraction or palpable goitre.

It is also important to mention the similarity between thyroid-related cortico-spinal tract (CST) symptoms and motor neuron disease. Differentiating both conditions is paramount, mainly for prognostic outcomes. Fisher *et al*¹⁵ published a patient with a 4 month-history of progressive tremor in hands, generalised weakness (mainly on the right upper and lower limb), normal radial, cubital, patellar and Achilles reflexes, bilateral Babinski and Hoffman signs and dysarthria. There was evidence of pyramidal tract and corticobulbar tract involvement (causative of dysarthria). The lack of hyper-reflexia in the lower extremities, despite the presence of Babinski and Hoffman signs, reflected the concomitant presence of a neuropathy. The EMG demonstrated minor slowing of nerve conduction, reduction in the CMAP, lack of impulse dispersion following proximal stimulation and absence of sensory nerve action potentials. These findings were indicative of an anterior horn/dorsal root ganglion cells or distal axons rather than one involving peripheral myelin. Further investigation revealed abnormal thyroid function and antithyroid treatment was given. There was complete resolution of symptoms after approximately 2–3 months. The authors favoured a thyrotoxicity-induced physiological and structural

Table 3 Comparative table between axonal/anterior horn and peripheral neuropathies

	Evoked potentials: CMAP/SNAP	Nerve conduction velocity
Axonal/Anterior horn cell	Small CMAP	Normal
Peripheral neuropathy	Small CMAP+SNAP	Slow

CMAP, compound motor action potential; SNAP, sensory nerve action potential.

dysfunction at the anterior horn/dorsal root ganglion cellular level to explain some of these features rather than a purely peripheral process.

Hyperthyroidism/thyrotoxicosis can affect the neuromuscular system at different levels. The precise mechanism has still not been elucidated. It is not clear if thyroxine causes direct cellular dysfunction,¹⁶ if there is also enzymatic and water electrolyte influence,¹⁷ or if there are underlying mitochondrial abnormalities.¹⁸ In a mature rat, brain hyperthyroidism causes a substantial increase in striatal beta-adrenoreceptors, striatal dopaminergic neuronal activity and presynaptic alpha-2-adrenoreceptor function.¹⁹ Other studies have demonstrated changes in the concentration of intracerebral substances such as serotonin, 5-HIAA and substance P.²⁰ More so, hyperthyroidism can alter the brain concentrations of glutamate dehydrogenase and pyruvate dehydrogenase,²¹ and this can be directly linked to differential neurological symptoms. All these findings support the causative association between thyrotoxicosis and neurological complications.²²

Whatever the final cause might be, we know however that the anterior horn cells are most likely to be the underlying anatomical site for the neuropathy,²³ and therefore, the use of EMG/NCS is very important for the final diagnosis.^{24–26} The NCS in an axonal lesion will present typical findings. First, there are small CMAP amplitudes because there are fewer (than normal) axons that are able to 'drive' or 'thrust' action potentials in the muscle. Second, since there is a residual population of normal axons from other anterior horn cells (non-damaged axons), nerve conduction is normal. Third, the sensory nerve conduction. Fibrillations, fasciculation, large-amplitude polyphasic units and a reduced recruitment pattern are also consistent with a subacute to chronic process involving either anterior horn cells or motor axons. Below, the comparative table (table 3) shows a simple differentiation between peripheral neuropathies and axonal horn damage.

Our patient presents a left pyramidal syndrome secondary to T3-thyrotoxicosis. There are some interesting and atypical features worth mentioning. First, no obvious clinical features of hyperthyroidism were noted, mainly no exophthalmos or palpable goitre. She only presented tachycardia, palpitations and subtle tremor of the hands, which are usually present in hyperthyroidism, however not specific to the disease. Second, the limitation to abduct the left eye and the 'pressure-like' feeling on movement was attributed to thyroid-associated ophthalmopathy; however, no other typical features were noted such as exophthalmos, lid retraction or proptosis. Third, the left hemiparesis without right-sided weakness is another atypical manifestation. Every case previously published regarding corticospinal tract involvement in thyroid disease describes patients with bilateral involvement, an expected manifestation in systemic diseases.

ⁱ Gustafsson R, Tata JR, Lindenberg O, et al. The relationship between the structure and activity of rat skeletal muscle mitochondria after thyroidectomy and thyroid hormone treatment, *Cell Biol* 26:555–578, 1965.

The abnormal thyroid function test was useful in narrowing the differential diagnosis (Canepa and Venu's patient²⁷ was taken into consideration, assisting in the diagnostic process). Furthermore, the thyroid ultrasound done approximately 1 month after her discharge showed signs consistent with subacute chronic thyroiditis.

Despite the atypical features previously mentioned, the authors conclude that the causal relationship between pyramidal tract signs and T3-thyrotoxicosis is likely for two reasons: (1) all investigations for any other intracerebral or spinal cause were negative and (2) the patient presented a parallel improvement of the thyroid function tests and neurological examination; there was undoubtedly an improvement of the neurological status secondary to antithyroid treatment.^{28–30} These two reasons were further reinforced by the EMG study results, which showed axonal/anterior horn lesion.

Learning points

- Hyperthyroidism/thyrotoxicosis can directly cause axonal/anterior horn damage, which can be described by electromyogram (EMG) and treated with antithyroid medication.
- Hyperthyroid-related neuropathy can present as a unilateral pyramidal syndrome
- Barre and Mingazzini signs are highly indicative of pyramidal tract (CST) involvement.
- Axonal or anterior horn cell disease on EMG is suggested by the following findings: (1) compound muscle action potentials are small (but not slow); (2) motor and sensory nerve conductions are normal.

Contributors CC obtained the patient after seeing her as an inpatient with his junior AM. We then requested the pertinent investigations and obtained the antiperoxidase antibody and antigraves antibody. Once positive, we requested RS opinion and management. He then requested the USA of thyroid and started treatment with carbimazole. We all collaborated to obtain the pertinent papers to use as bibliography. CC made the main discussion with added comments from RS and AM and we all followed up the patient in clinic. We reached our conclusions together after meeting on a couple of occasions to discuss our findings and investigation results.

Competing interests None declared.

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