Heliyon 10 (2024) e33974

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Case report

Hypoglossal nerve involvement and sternocleidomastoid muscle atrophy in chronic inflammatory demyelinating polyneuropathy with Hashimoto's thyroiditis: A case report and literature review

Lixia Chen [a,b], Huan Wang [a,b], Ting Zheng [a,b,*]

[a] Department of Neurology, The Second Hospital & Clinical Medical School.Lanzhou University, Lanzhou, 730030, China
[b] Academician Workstation of The Second Hospital & Clinical Medical School .Lanzhou University, Lanzhou, 730030, China

ARTICLE INFO

Keywords:
Chronic inflammatory demyelinating polyneuropathy
Hypoglossal nerve
Sternocleidomastoid muscles
Hypothyroidism
Case report

ABSTRACT

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an immune-mediated neuropathy. While CIDP typically affects the peripheral nerves in the limbs, involvement of cranial nerves is atypical, and cases of muscle atrophy secondary to cranial nerve involvement are exceptionally rare. A 30-year-old female patient, who complained of numbness and weakness in her limbs, was diagnosed with CIDP after experiencing atrophy of the tongue and sternocleidomastoid muscles, along with tongue muscle fibrillation during a neurological examination. Additionally, the patient had hypothyroidism caused by Hashimoto's thyroiditis. Cerebrospinal fluid tests indicated albumincytological dissociation. Electrophysiological examination results confirmed the diagnosis of typical CIDP. Glucocorticoid treatment, a standard therapy for CIDP, led to a significant improvement in the patient's symptoms, including the regeneration of her tongue muscles. A literature review revealed only eight cases of CIDP with hypoglossal nerve involvement, and this case represents the first documentation of concurrent sternocleidomastoid muscle atrophy. Although muscle atrophy from cranial nerve involvement is infrequent in CIDP, the positive response to treatment is encouraging.

1. Background

Chronic inflammatory demyelinating polyneuropathy (CIDP) is an immune-mediated neuropathy characterized by a chronic course, which may manifest as relapses and remissions or a continuous progression. Common symptoms of CIDP include symmetrical limb numbness and weakness, absent tendon reflexes, sensory ataxia, and electrophysiological evidence of peripheral nerve demyelination [1]. Cranial nerve involvement is infrequent in CIDP, and associated muscle atrophy is even rarer. When cranial nerves are affected, it typically includes the oculomotor, facial, vagus, and glossopharyngeal nerves. Less commonly involved are the vestibular, abducens, accessory, and hypoglossal nerves [2,3].

CIDP is frequently comorbid with other autoimmune disorders, such as Sjögren's syndrome, systemic lupus erythematosus, and Hashimoto's thyroiditis [4–8]. Measuring thyroid hormone levels is crucial for differentiating CIDP. Case reports have indicated an association between Hashimoto's thyroiditis, a common etiology of hypothyroidism, and the development of CIDP [4,9].

We present the case of a 30-year-old female with CIDP who exhibited physical examination findings including tongue and

* Corresponding author. No.82, Cuiyingmen, Chengguan District, Lanzhou, 730030, Gansu Province, China.
E-mail address: zt618@163.com (T. Zheng).

https://doi.org/10.1016/j.heliyon.2024.e33974
Received 25 February 2024; Received in revised form 27 June 2024; Accepted 1 July 2024
Available online 2 July 2024

sternocleidomastoid muscle atrophy, along with tongue muscle fasciculation. Concurrent with the progression of her CIDP, she developed hypothyroidism due to Hashimoto's thyroiditis. Her clinical symptoms were effectively managed with hormone therapy. This case highlights a rare manifestation of CIDP and provides valuable insights for diagnosis and treatment. To further elucidate this complex condition, we conducted a comprehensive literature review.

1.1. Case presentation

In August 2020, a 30-year-old Asian woman presented to a community hospital's outpatient clinic with one month of distal extremity numbness, fatigue, and hair loss. Initial blood and thyroid function tests revealed hypothyroidism, prompting treatment with 50 μg of levothyroxine sodium daily. After over a month of treatment, her fatigue improved significantly. However, she subsequently stopped the medication and experienced persistent mild limb numbness and weakness, particularly in the lower limbs. This gradually progressed from her feet to her thighs. In September 2021, the patient was admitted to our neurology department due to the severe motor and sensory disturbances in her limbs, which severely impaired her ability to perform daily tasks and work. She experienced difficulties in holding chopsticks and maintaining balance while walking. Additionally, slurred speech was observed. Prior to her admission to our neurology department, she had no history of hospitalization or specialized medication, excluding her prior

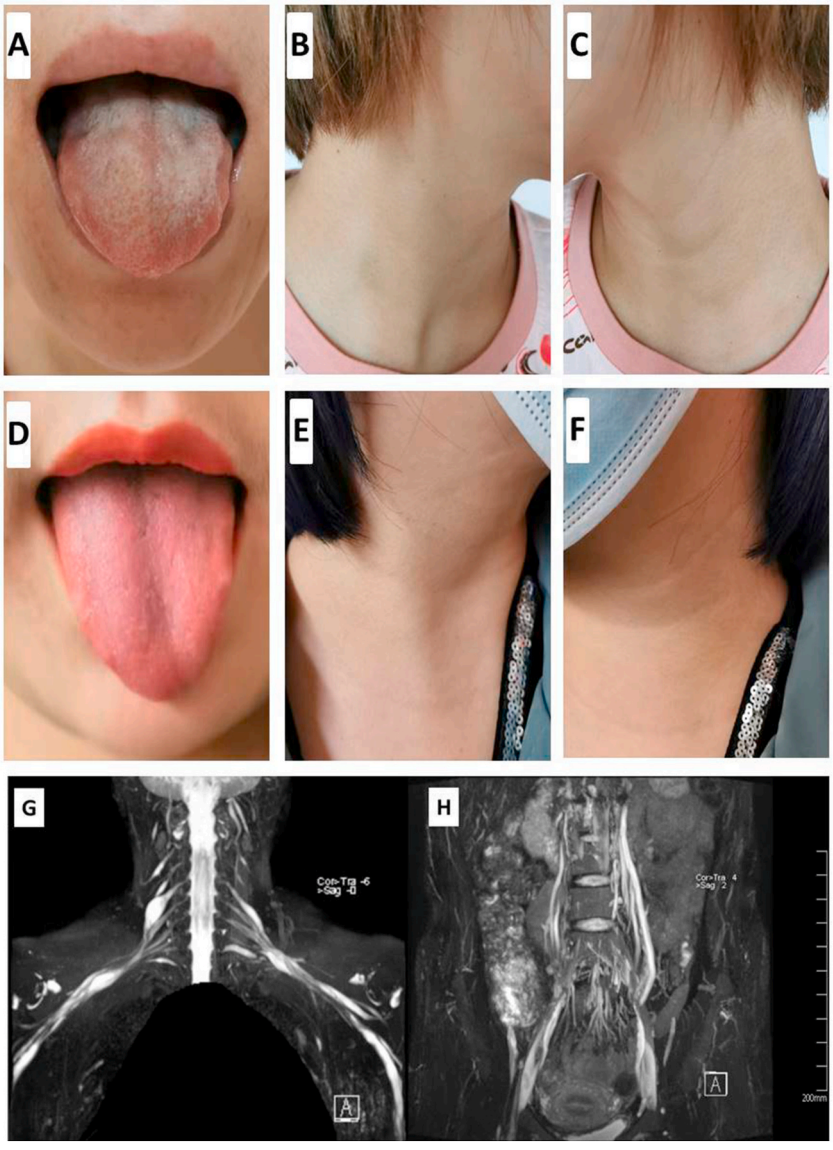

Fig. 1. Comparison of tongue muscle and sternocleidomastoid muscle atrophy in patient before and after treatment: tongue muscle in A and D, right sternocleidomastoid muscle in B and E, left sternocleidomastoid muscle in C and F, before treatment in A, B and C, and after treatment in D, E and F. Magnetic resonance imaging of brachial plexus (G) and lumbosacral plexus (H) showed edema, thickening and enhancement of nerve roots.

hypothyroidism treatment.

A neurological examination revealed dysarthria; asymmetric atrophy of the tongue with fasciculation, more severe on the left side; and atrophy of the left sternocleidomastoid(Fig. 1A–C). Upon assessing the sternocleidomastoid muscle strength, the patient demonstrated partial resistance against the manual resistance during right cervical rotation, while achieving complete resistance during left cervical rotation. The pharyngeal reflex is intact, and there is no dysphagia. Flaccid tetraparesis is present, with muscle strength graded 4 on the MRC scale for proximal muscles and 3 for distal muscles in both upper and lower limbs(Table 3). Mild atrophy of the dorsal interosseous muscles is noted in both hands. There is generalized areflexia, a negative Babinski sign, and significantly reduced pinprick and vibration sensation in the wrists and ankles. The patient fails bilateral finger-to-nose and heel-knee-tibia tests, exhibiting a sensory ataxic gait without assistance. A positive Romberg sign is observed.

Negative results were found in the complete blood count, C-reactive protein, erythrocyte sedimentation rate, biochemistry, liver function, kidney function, rheumatoid antibody, fasting serum glucose, serum and urine protein electrophoresis and immunofixation, and communicable disease test (Hepatitis A, B, C, human immunodeficiency virus, syphilis). Thyroid function testing revealed significantly elevated TSH, thyroglobulin (TG), anti-thyroid peroxidase antibodies (anti-TPO-Ab), and anti-thyroglobulin antibodies (anti-ATG-Ab), with a slight decrease in free thyroxine (FT4)TSH = 121.641 uIU/mL (normal range 0.38–4.34), TG = 111.00 ng/mL (normal range 0.83–68), anti-TPOAb >1300 U/mL (normal range < 60 U/mL), anti-TGAb = 244.10 U/mL (normal range < 60 U/mL), and FT4 = 6.71 pmol/L (normal range 10.44–24.38 pmol/L). Ultrasound of thyroid suggested a diffuse lesion. Lumbar puncture yielded CSF with albumincytological dissociation (white blood cell count (WBC), 0 cells/mm^3 [0–8 cells/mm^3]; protein level, 1.02 g/L [0.2–0.4 g/L]). No organisms were detected on gram and acid-fast bacillus stain, and CSF cultures were negative for bacteria and fungus. Needle electromyography and nerve conduction velocity studies confirmed severe, diffuse, symmetric, sensorimotor, predominantly primary segmental demyelinating polyradiculoneuropathy, meeting the electrophysiologic criteria for CIDP. Sensory nerve conduction responses of the ulnar, median, and sural nerves were unresponsive. Motor nerve conduction revealed decreased velocity and amplitude in bilateral ulnar, median, left tibial, and left peroneal nerves, with abnormal temporal dispersion and distal latency prolongation (Table 1). Spontaneous potentials such as fibrillation and positive sharp waves were observed in bilateral tongue muscles on needle electromyography. Magnetic resonance imaging (MRI) of the brain and spinal cord was normal. MRI of the brachial and lumbosacral plexus showed multiple nerve root edema and thickening (Fig. 1G.H). Anti-ganglioside antibodies, myelin-associated glycoprotein, neurofascin 155 and 186 autoantibodies, and anti-contactin 1 were all negative.

Based on the patient's symptoms, signs, and auxiliary examination, CIDP was suspected. The patient declined further nerve biopsy. Treatment with methylprednisolone pulse therapy (500 mg ivgtt qd) was administered for 5 days, followed by a gradual tapering regimen (250 mg for 3 days, 120 mg for 3 days). An endocrinologist diagnosed hypothyroidism due to Hashimoto's thyroiditis and prescribed levothyroxine (100 μg orally daily).Physical examination revealed gradual improvement in limb muscle strength and a decrease in paresthesia, corroborating the patient's self-report. Twenty-four days after the last thyroid function test, repeat testing was performed, revealing a significant decrease in thyroid-stimulating hormone (TSH) levels and anti-thyroid peroxidase antibody (anti-

Table 1
Nerve conduction velocity studies.

Nerve SNC	Left			Right		
	Distal latency(ms)	SNAP(μv)	SCV(m/s)	Distal latency(ms)	SNAP(μv)	SCV(m/s)
median		NR			NR	
ulnar		NR			NR	
Radial		NR			NR	
superficial peroneal	1.89	7.0	47.6	2.19	6.3	41.1
sural		NR			NR	
Nerve **MNC**	**Left**			**Right**		
	Distal latency(ms)	CMAP(mv)	MCV(m/s)	Distal latency(ms)	CMAP(mv)	MCV(m/s)
median						
wrist-APB	8.85	6.9		8.79	4.5	
elbow-wrist	17.1	2.8	22.4	13.6	1.67	37.4
axilla–elbow	25.4	0.92	14.5	17.4	1.15	31.6
F wave	NR			NR		
Ulnar						
wrist—ADM	5.19	7.7		4.35	9.1	
Below elbow—wrist	8.04	6.7	43.9	7.77	7.8	39.5
Above elbow—below elbow	11.7	5.5	30.1	12.0	5.9	23.6
Tibial						
ankle-AH	8.85	4.2		7.27	2.2	
popliteal fossa—Ankle	17.6	3.4	37.7	13.6	2.1	49
F wave	NR			NR		
peroneal						
ankle—EDB	10.4	3.2		6.62	5.5	
CF—Ankle	18.2	2.9	34.6	13.0	4.9	42.3

SNC Sensory nerve conduction NR no response MNC Motor nerve conduction APB abductor pollicis brevis ADM abductor digiti minimi AH abductor halluces EDB Extensor digitorum brevis CF capitulum fibula.

Table 2
Patients with hypoglossal neuropathy in chronic inflammatory demyelinating polyneuropathy.

Author year	Age gender	Clinical manifestation	CSF finding	EMG and NCV	Image	Treatment	Outcome
Zhao, H et al., 2021	28 male	Distal-predominant limb weakness and numbness at the age of 18,relapsed at the age of 28,mild bilateral facial paresis(7th), tongue atrophy and fasciculations(12th), and inverted Beevor's sign.	A normal protein content, cell count, and glucose leve. Anti-ganglioside antibodies, myelin-associated glycoprotein, neurofascin 155 and 186, and contactin 1 were all negative.	Prolonged distal motor latencies, delay and disappearance of F waves, and conduction block in the radial and ulnar nerves.	The nerve ultrasonography showed segmental hypertrophy in the bilateral median and ulnar nerves. Magnetic resonance imaging of the lumbosacral plexus revealed that the L5 and S1 spinal roots were mildly enlarged	IVIg at 0.4 g/kg/day for 5 days, intravenous methylprednisolone at 500 mg per day for 3 days, and then oral prednisolone at 60 mg/day for 1 month before it was gradually tapered off.	Numbness relieved completely, limb strength recovered 70 %. tongue atrophy not mentioned.
Roberto, K. T. et al., 2020	31 male	A year history of symmetrical weakness and paresthesias in both lower and upper extremities, bilateral facial paresis(7th) and tongue fasciculation with no dysarthria or dysphagia (12th).	High cell count, high protein content(>540 mg/dL), normal glucose level (61 mg/dL).	Fibrillation and positive sharp wave in the right tibialis anterior, left medial gastrocnemius, left rectus femoris, and left first dorsal interosseous muscles Complete conduction block.	Not reported.	IVIg at a dose of 2 g/kg over two days.	Persistence of symptoms but with no further neurologic deterioration.
Jha, S. et al., 2011	14 boy	Weakness from upper limbs to lower limbs. breathlessness and nasal twang in voice along with nasal regurgitation of food and fluids. Paralysis of 9th, 10th, 11th, and 12th cranial nerves. The signs and symptoms of accessory nerve involvement were not described.	Elevated proteins (120 mg/dL) and five lymphocytes.	Prolonged distal latencies, conduction block, decreased conduction velocities, prolonged F-wave, and decreased motor action potentials of the median, ulnar and common peroneal nerves.	Not reported	IVIg, Oral corticosteroids (1 mg/kg body weight).	Breathe normally and perform all activities of daily living independently
Mehndiratta et al., 2010	62 male	Bilaterally symmetrical weakness with distal and proximal numbness and burning paresthesias (mainly distal) in the lower limbs for 2.5 years, and similar complaints of 4 months duration in both upper limbs. Fasciculations in his masseter muscles and tongue. Atrophy of tongue (5th, 12th).	No cells, increased protein concentration (139 mg/dL) and normal sugar level.	Conduction block, temporal dispersion, prolonged distal latencies, absent F-wave latencies and reduced nerve conduction velocities in the motor nerves of all four extremities. active denervation in the form of fibrillations and fasciculations from the masseter and tongue muscles.	Not reported	Not reported	Not reported
Hemmi et al., 2008	55 male	Weakness and numbness of limbs, fasciculation and atrophy of the tongue (12th)	Acellular with a protein level of 41 mg/dL	Prolonged terminal latencies and marked slowing in motor nerve conduction velocity.	Not reported	IVIg therapy (400 mg/kg for 5 days).	Improvement of the limbs,no improvement of hypoglossal neuropathy.

(*continued on next page*)

Table 2 (*continued*)

Author year	Age gender	Clinical manifestation	CSF finding	EMG and NCV	Image	Treatment	Outcome
				Profuse fibrillation potentials and positive sharp waves were present in the left tongue.			
Yamashiro et al., 2004	49 male	Weakness of the distal upper extremities, atrophy of the tongue without fasciculation, speech disturbance, difficulty in swallowing, bilateral facial weakness, Moderate dysphagia and dysarthria(9th, 10th and 12th).	An elevated protein content (76 mg/dL) with a normal white blood cell count.	Slowing of nerve conduction velocities and reduced sensory and motor action potential amplitude. conduction block	Not reported	IVIg at 0.4 g/kg/day for 5 days another 5-day course of intravenous immunoglobulin 4 months later.	Dysarthria and dysphagia had improved, atrophy remained in the tongue.
Inoue et al., 2004	61 male	Sensorimotor impairment(13 years) painful dysesthesia(3 months) extremity atrophy bilateral ophthalmoparesis, facial palsy, and tongue atrophy (7th,12th)	Not reported	Not reported	MRI demonstrated bilateral nerve root hypertrophy of the oculomotor, trigeminal (figure), abductor, and facial nerves.	Not reported	Not reported
Waddy et al., 1989	27 female	General weakness, facial numbness, deviation of her tongue to the left vertical diplopia(5th, 12th).	Elevated proteins (100 mg/dL) and 30 WBCs.	Reduced motor conduction velocity and increased distal motor latency consistent with demyelination. multifocal conduction block Sensory action potentials were absent or markedly reduced in amplitude.	No abnormality was detected	Corticosteroids(The specific dose was not mentioned)	Slow improvement has occurred. Details were not described.

TPOAb) levels, although anti-thyroid globulin antibody (anti-TGAb) levels remained unchanged(TSH = 6.914 uIU/mL, anti-TPOAb >1300 U/mL, anti-TGAb = 279.40 U/mL). At discharge the patient was recommended to continue oral therapy with prednisone acetate (60 mg/day, tapered to a maintenance dose of 20 mg/day) and levothyroxine (50 μg qd).

Three months after discharge, the patient reported significant improvement in symptoms of limb weakness and numbness. Her speech had clarity and was nearly normal. Muscle strength had recovered to MRC scale grade 4–5 in both proximal and distal muscles of the upper and lower limbs. Notably, her tongue muscles were almost fully recovered. However, atrophy of the left sternocleidomastoid muscle had not changed. This did not affect her neck movement, however, as it did 3 months before (Fig. 1D–F). Thyroid hormone tests revealed substantial improvement (TSH = 0.386 uIU/mL, anti-TPOAb = 442.6 U/mL, anti-TGAb = 45.2 U/mL). Based on these findings, the patient was advised to continue a lower maintenance dose of steroids (20 mg/day) and to reduce levothyroxine dosage to 25 μg once daily. One year later, all symptoms have resolved, except for the persistent atrophy of the left sternocleidomastoid muscle. The patient's prednisone regimen was tapered to 5 mg orally every other day, and levothyroxine was discontinued as recommended by an endocrinologist. (The timeline for the Case presentation is shown in Fig. 2)

2. Discussion and conclusions

This report presents an unprecedented clinical case of CIDP co-occurring with hypothyroidism and presenting with tongue and sternocleidomastoid muscle atrophy. Extensive literature searches have confirmed this as the first documented case of such a unique combination. The case is particularly remarkable for the patient's successful recovery and return to normal functioning following standardized treatment. The limitations of this case study, as is typical for such reports, include the small sample size, which restricts the generalizability of the findings.

Table 3
Muscle test evaluations.

Muscles	Muscle strength	Muscle volume
Head and neck muscles		
Muscles of tongue	No deviation	Bilateral atrophy
Sternocleidomastoid	Right cervical rotation: 4; Left cervical rotation: 5	Left atrophy
Trapezius	Normal	Normal
Upper limbs		
Supraspinatus	4	Normal
Pectoralis major	4	Normal
Deltoid	4	Normal
Biceps brachii	4	Normal
Triceps brachii	4	Normal
Etensor carpi ulnaris	3	Normal
Extensor digitorum communis	3	Normal
Flexor carpi ulnaris	3	Normal
Dorsal interossei	3	Atrophy
Palmar interossei	3	Normal
Lower limbs		
Iliopsoas	4	Normal
Quadriceps femoris muscle	4	Normal
Gluteus medius	4	Normal
Gluteus minimus	4	Normal
Biceps Femoris Muscle	4	Normal
Tibialis anterior	3	Normal
Gastrocnemius	3	Normal

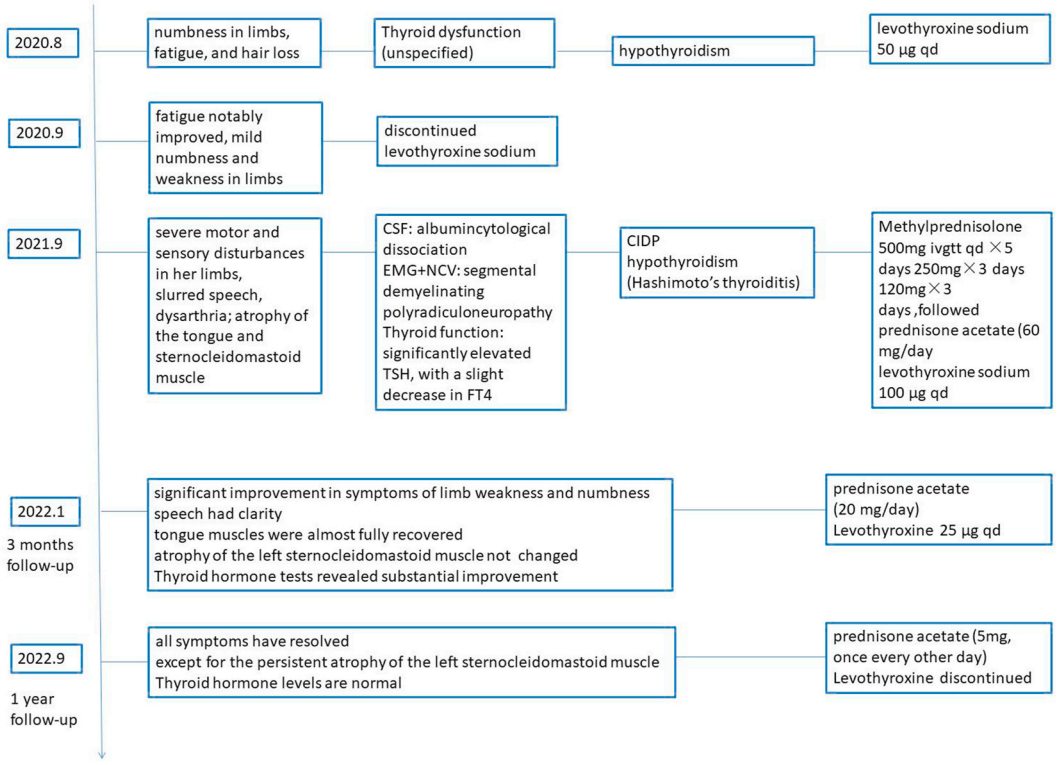

Fig. 2. The timeline for the Case presentation.

2.1. CIDP and thyroid disease

The relationship between CIDP and thyroid disease has been suggested in various case reports and studies, likely due to shared immune-mediated nature and genetic predispositions [9–11]. Cytokines such as interferon-γ, tumor necrosis factor α (TNF-α), and interleukin 17 (IL-17), as well as oxidative stress mediated by reactive oxygen species (ROS), play a role in both autoimmune thyroid

diseases and demyelination process in CIDP [12,13]. Hypothyroidism can exacerbate symptoms, like limb weakness and delayed relaxation of deep tendon reflexes, which are easily underestimated by doctors according to the principle of monism [14]. In the described case, the initial presentation of limb weakness and numbness was misdiagnosed as hypothyroidism, and EMG was not promptly conducted to investigate peripheral nerve involvement, resulting in a delayed CIDP diagnosis. The coexistence of thyroid disorders and peripheral neuropathy presents a complex scenario that warrants careful evaluation and management. The progression of peripheral neuropathy symptoms during hypothyroidism treatment, followed by significant improvement with hormone therapy, and the fulfillment of CIDP diagnostic criteria in our patient, suggest that CIDP was the primary condition. However, a dynamic electrophysiological examination at the disease's outset could have provided clearer evidence. The interplay between hypothyroidism and the rare manifestations of CIDP merits further investigation to clarify the extent of their association.

2.2. CIDP with tongue and sternocleidomastoid muscle atrophy

Our search of PubMed did not identify any existing case reports of CIDP patients exhibiting both tongue muscle atrophy and sternocleidomastoid muscle atrophy. Consequently, we consider this case to be novel and to have significant clinical teaching value. Notably, PubMed also did not list any cases of sternocleidomastoid atrophy in CIDP patients. Given that sternocleidomastoid atrophy suggests involvement of the accessory nerve, we conducted an additional review focusing on accessory nerve involvement in CIDP. We found only one article addressing this issue, but it provided insufficient description of the symptoms and signs associated with accessory nerve involvement [15]. However, we noted eight case reports of CIDP patients with hypoglossal neuropathy (Table 2) [15–22]. Despite potential publication bias, seven of these patients were male and seven were from Asian countries, including Japan, China, and India; one patient was from Europe. But there were no discernible characteristics or patterns in terms of age distribution. Hypoglossal neuropathy occurred in combination with other cranial nerves, the most common being the facial nerve (4/8) [16,17,20,21], followed by the vagus and glossopharyngeal nerves (2/8) [15,20], the oculomotor nerve (1/8) [21], and the trigeminal nerve (1/8) [18]. Two cases involved only the hypoglossal nerve [19,22]. Tongue muscle atrophy was explicitly mentioned in six cases [16–21]., yet no improvement in tongue atrophy was reported in three patients following treatment with intravenous immunoglobulins or steroids [17,19,20], and the recovery of symptoms in the remaining three patients was not described [16,18,21].

2.3. CIDP with cranial nerve involvement

A separate analysis of adult and pediatric CIDP patients revealed that cranial nerve involvement was relatively rare in both groups:4 out of 69 adults (5.8 %) and 2 out of 15 children (13 %) experienced such involvement [23]. A 6-year retrospective clinical study conducted at a Greek hospital included 25 patients, with 9 (36 %) exhibiting cranial nerve involvement [24]. The incidence of cranial nerve involvement varies across different populations, although the facial and oculomotor nerves are consistently found to be the most vulnerable in CIDP research [3,24]. Anti-NF155 antibody-positive CIDP patients are more likely to present with cranial nerve involvement [25]. A separate study indicated that patients who had experienced a prodromal infection were more likely to have cranial nerve involvement compared to those without such an event (42 % versus 18 %) [26]. Additionally, researchers observed that patients whose disease onset was less than 2 months were at a higher risk for cranial nerve involvement [27].

In conclusion, we report a unique case of CIDP in which the patient exhibited hypothyroidism and associated atrophy of the tongue and sternocleidomastoid muscle. Following standard treatment, the patient experienced a favorable recovery. A literature review confirmed that this is the first documented case of CIDP presenting with these specific symptoms. This case study emphasizes the critical need for clinicians to be vigilant about the diverse manifestations of CIDP. It underscores the value of early recognition and effective management in achieving favorable outcomes, particularly for patients presenting with atypical symptoms.

Ethics approval and consent to participate

Informed consent was obtained from the patient to publish this case.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Funding

This work was supported by the Cuiying Scientific and Technological Innovation Program of the Second Hospital & Clinical Medical School, Lanzhou University, with grant numbers CY2020-MS18 and CY2023-QN-B02. The funders had no role in study design; collection, analysis, and interpretation of data; in the writing of the report; and in the decision to submit this article for publication.

Data availability statement

Data will be made available on request.

CRediT authorship contribution statement

Lixia Chen: Writing – original draft, Investigation, Formal analysis, Data curation. **Huan Wang:** Writing – review & editing, Supervision, Formal analysis, Data curation, Conceptualization. **Ting Zheng:** Writing – review & editing, Writing – original draft, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

The research in this article was supported by the Cuiying Scientific and Technological Innovation Program of the Second Hospital & Clinical Medical School, Lanzhou University, with grant numbers CY2020-MS18 and CY2023-QN-B02.

List of abbreviations

CIDP	Chronic inflammatory demyelinating polyneuropathy
MRC	Medical Research Council
CSF	Cerebrospinal fluid
WBC	white blood cell
MRI	magnetic resonance imaging
TSH	thyroid stimulating hormone
TG	thyroglobulin
anti-TPO-Ab	anti-thyroid peroxidase antibodies
anti-ATG-Ab	anti-thyroglobulin antibodies
FT4	free thyroxine.

References

[1] M.C. Dalakas, Advances in the diagnosis, pathogenesis and treatment of CIDP, Nat. Rev. Neurol. 7 (9) (2011) 507–517.
[2] G. Said, Chronic inflammatory demyelinating polyneuropathy, Neuromuscul. Disord. 16 (5) (2006) 293–303.
[3] F.T. Rotta, et al., The spectrum of chronic inflammatory demyelinating polyneuropathy, J. Neurol. Sci. 173 (2) (2000) 129–139.
[4] S. Raghavendra, et al., CIDP, Hashimoto's thyroiditis and nephropathy: autoimmune syndrome complex? Can. J. Neurol. Sci. 36 (3) (2009) 382–384.
[5] S. Bregante, et al., Sjögren's syndrome associated chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) treated with autologous and subsequently allogeneic haematopoietic SCT (HSCT), Bone Marrow Transplant. 48 (8) (2013) 1139–1140.
[6] C.S. Simonsen, et al., Chronic inflammatory demyelinating polyradiculoneuropathy occurring after autologous haematopoietic stem cell transplantation for multiple sclerosis, Mult Scler J Exp Transl Clin 2 (2016) 2055217316658304.
[7] J.P. Domingos, et al., Chronic inflammatory demyelinating polyneuropathy associated with autoimmune hepatitis, Pediatr. Neurol. 51 (3) (2014) e13–e14.
[8] M. Alabdali, et al., Clinical characteristics, and impairment and disability scale scores for different CIDP Disease Activity Status classes, J. Neurol. Sci. 372 (2017) 223–227.
[9] C. Bairactaris, et al., Early neurophysiological evolution of chronic inflammatory demyelinating polyneuropathy in a patient with Hashimoto's thyroiditis, Muscle Nerve 38 (5) (2008) 1518–1522.
[10] B. Sheng, et al., A case of Hashimoto's encephalopathy with demyelinating peripheral neuropathy, Eur. Neurol. 53 (2) (2005) 84–85.
[11] Y. Tu, et al., Differences in thyroid function and autoantibodies in the development of Guillain-Barre syndrome vs. Chronic inflammatory demyelinating polyradiculoneuropathy, Front. Neurol. 11 (2020) 1018.
[12] G. Karanikas, et al., Relation of anti-TPO autoantibody titre and T-lymphocyte cytokine production patterns in Hashimoto's thyroiditis, Clin. Endocrinol. 63 (2) (2005) 191–196.
[13] G. Bona, et al., Defective function of Fas in T cells from paediatric patients with autoimmune thyroid diseases, Clin. Exp. Immunol. 133 (3) (2003) 430–437.
[14] D.Y. Gaitonde, K.D. Rowley, L.B. Sweeney, Hypothyroidism: an update, Am. Fam. Physician 86 (3) (2012) 244–251.
[15] S. Jha, et al., Unusual features in chronic inflammatory demyelinating polyneuropathy: good outcome after prolonged ventilatory support, J. Neurosci. Rural Pract. 2 (2) (2011) 171–173.
[16] H. Zhao, et al., Chronic inflammatory demyelinating polyneuropathy with hypoglossal nerve involvement and inverted Beevor's sign: case report, BMC Neurol. 21 (1) (2021) 244.
[17] K.T. Roberto, et al., Chronic inflammatory demyelinating polyneuropathy with tongue fasciculation: a case report, J. Clin. Neurosci. 71 (2020) 297–299.
[18] M.M. Mehndiratta, Trigeminal motor neuropathy with tongue fasciculations in a patient with chronic inflammatory demyelinating polyradiculoneuropathy, J. Clin. Neurosci. 17 (10) (2010) 1355–1356.
[19] S. Hemmi, et al., Tongue fasciculations in chronic inflammatory demyelinating polyradiculoneuropathy, Muscle Nerve 38 (4) (2008) 1341–1343.
[20] K. Yamashiro, et al., Bilateral hypoglossal nerve involvement in chronic inflammatory demyelinating polyneuropathy, Eur. Neurol. 51 (1) (2004) 45–46.
[21] H. Inoue, et al., Hypertrophic cranial nerve roots in CIDP, Neurology 63 (8) (2004) 1481.
[22] H.M. Waddy, et al., Focal cranial nerve involvement in chronic inflammatory demyelinating polyneuropathy: clinical and MRI evidence of peripheral and central lesions, J. Neurol. 236 (7) (1989) 400–405.
[23] Z. Simmons, J.J. Wald, J.W. Albers, Chronic inflammatory demyelinating polyradiculoneuropathy in children: I. Presentation, electrodiagnostic studies, and initial clinical course, with comparison to adults, Muscle Nerve 20 (8) (1997) 1008–1015.
[24] M. Rentzos, et al., Chronic inflammatory demyelinating polyneuropathy: a 6-year retrospective clinical study of a hospital-based population, J. Clin. Neurosci. 14 (3) (2007) 229–235.

[25] G. Liberatore, et al., Frequency and clinical correlates of anti-nerve antibodies in a large population of CIDP patients included in the Italian database, Neurol. Sci. 43 (6) (2022) 3939–3947.
[26] P.E. Doneddu, et al., Risk factors for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP): antecedent events, lifestyle and dietary habits. Data from the Italian CIDP Database, Eur. J. Neurol. 27 (1) (2019) 136–143.
[27] S. Cabasson, et al., Childhood CIDP: study of 31 patients and comparison between slow and rapid-onset groups, Brain Dev. 37 (10) (2015) 943–951.