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Case Report

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Relapsing Multiple Cranial Neuropathies as a Manifestation of Neurosyphilis: A Case Report and Literature Review

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[Abstract] Neurosyphilis is an infectious disease of the central nervous system caused by *Treponema pallidum*. Its incidence has been increasing globally. Due to variable clinical presentations, nonspecific symptoms, and diverse latency periods, neurosyphilis is frequently misdiagnosed or missed altogether. It commonly involves a single cranial nerve. We report a rare case of neurosyphilis presenting with multiple cranial nerve involvement as the initial manifestation. The patient was admitted with progressive cranial neuropathies. Comprehensive evaluations, including serologic tests, cerebrospinal fluid (CSF) analysis, and cranial MRI, were conducted. Laboratory findings supported the diagnosis of neurosyphilis, with positive serum and CSF *Treponema pallidum* antibodies, CSF pleocytosis, and elevated protein levels. MRI revealed abnormal enhancement of multiple cranial nerves. The patient was treated with high-dose intravenous penicillin, resulting in gradual clinical improvement. This case highlights the diagnostic challenges posed by atypical neurosyphilis presentations. By reviewing this rare case and relevant literature, we aim to improve clinicians' awareness of neurosyphilis with polycranial neuritis and promote timely diagnosis and effective treatment.

[Keywords] Neurosyphilis, polycranial neuritis, meningitis, cerebral vasculitis

1. Introduction

Neurosyphilis is an infectious disease caused by the invasion of *Treponema pallidum*, involving many parts of the brain, meninges, spinal cord, blood vessels, peripheral nerves, and organs.^[1] Notably, the course of the disease varies from several days to several years after infection. Most untreated syphilis patients have a chronic or subacute course, with a short incubation period of 2 months and up to 25 years^[2]. Early neurosyphilis can be divided into asymptomatic neurosyphilis, syphilis meningitis, late neurosyphilis gradually appearing as paralytic dementia, tabes dorsalis, and meningeal vascular syphilis can be seen in early or late syphilis^[3]. But, the symptoms and signs of neurosyphilis patients are unclear, such as headache, dizziness, meningitis, epilepsy, stroke, insomnia, cognitive impairment, mental symptoms, language impairment, vision loss, and other neurological-related symptoms and signs, which are the main reasons for their misdiagnosis and missed diagnosis^[4].

Cranial nerve involvement is well recognized in neurosyphilis, but involvement of several cranial nerves during the same disease course is uncommon^[5]. When this occurs, the presentation may resemble other inflammatory, demyelinating, vascular, or infectious neurological disorders, which can delay the correct diagnosis.

We describe a 54-year-old woman who developed recurrent cranial neuropathies over a two-month period, with involvement of the optic, oculomotor, abducens, facial, and vestibulocochlear nerves. Serum and cerebrospinal fluid findings supported the diagnosis of neurosyphilis, and her neurological symptoms improved after penicillin treatment. This case illustrates the importance of considering neurosyphilis in patients with unexplained recurrent or multifocal cranial nerve palsies.

2. Case presentations

(Female, 54 years old) The patient presented with left facial asymmetry two months ago and was diagnosed with cerebral infarction and intracranial aneurysm at Xi'an Gaoxin Hospital after MRI and MRA showed pons and bilateral basal ganglia softening lesions, multiple infarcts, and a suspected left internal carotid siphon segment aneurysm. She was also diagnosed with left facial nerve palsy and received unspecified treatment.

Approximately 1 month before admission, she developed diplopia and left eye ptosis. Myasthenia gravis was initially considered; however, there was no clear diurnal fluctuation, repetitive nerve stimulation was normal, and visual evoked potentials showed a normal P100 response. After hormone therapy, her ptosis resolved.

Ten days before admission, she experienced blurred vision in bright light and dizziness, with stable gait and intact limb mobility. There were no signs of nausea, vomiting, dysphagia, or typical myasthenia symptoms. She received treatment to improve circulation, nourish nerves (Mecobalamin 0.5mg po.tid), and regulate blood lipids and platelets, but visual symptoms persisted.

Two days before admission, she developed new dizziness, difficulty closing the right eye, and right-sided facial deviation. She was diagnosed with multiple neuritis.

Her medical history was notable for hypertension and a history of medication use (specifics not provided).

Physical exam findings:

- General condition: stable; no rash.
- Conscious, fluent speech.
- Pupils: 4 mm; right sluggish light reflex, left sensitive.
- Right eye: limited adduction/abduction; left eye: horizontal nystagmus.
- Bilateral peripheral facial paralysis, left > right. The left reflects a previous episode; current symptoms are on the right.
- Tongue midline, normal limb strength and tone.
- Coordination was intact and the Romberg test was negative.
- Bilateral tendon reflexes ++; no pathological reflexes.

Ophthalmology findings:

- Visual acuity (VOD/VOS): 0.05
- Fundoscopy: suspected fissure at the right eye fundus, retinal clastoid degeneration in the left.

- **OCT:** Normal bilateral macular thickness.

As shown in Table 1.

Table 1. Clinical course and sequential cranial nerve involvement

Time relative to admission	Cranial nerve involvement	Main clinical manifestations
Approximately 2 months before admission	Left facial nerve (CN VII)	Left-sided peripheral facial weakness
Approximately 1 month before admission	Left oculomotor nerve (CN III)	Diplopia and left ptosis
10 days before admission	Visual/optic nerve involvement	Blurred vision and marked reduction in visual acuity
2 days before admission	Right facial nerve (CN VII)	Incomplete right eye closure and new right-sided facial weakness
At admission	Right oculomotor nerve (CN III) and right abducens nerve (CN VI)	Impaired adduction and abduction of the right eye
During the evolving illness	Vestibular/vestibulocochlear involvement (CN VIII)	Dizziness and horizontal nystagmus

Auxiliary examination: Initial blood tests showed a white cell count of $3.34 \times 10^9/L$, neutrophil count of $1.79 \times 10^9/L$ and platelet count of $102 \times 10^9/L$. Liver and renal function tests and serum lipid concentrations were not significantly abnormal.

Serological testing for syphilis was positive, including rapid plasma reagin (RPR), toluidine red unheated serum test (TRUST) and Treponema pallidum particle agglutination assay (TPPA). Cerebrospinal fluid (CSF) examination demonstrated a white cell count of $29 \times 10^6/L$ with lymphocytic predominance and an elevated protein concentration of 0.92 g/L. CSF testing for syphilis, including RPR, TRUST and TPPA, was also positive. Tests for cryptococcal infection, tuberculosis and the tested viral pathogens in CSF were negative.

Additional immunological investigations showed positive pANCA and weakly positive anti-SSA/Ro52 antibodies.

Oligoclonal IgG bands were detected in both the cerebrospinal fluid and serum, with a greater number of bands in the cerebrospinal fluid than in serum. The cerebrospinal fluid IgG index was elevated at 1.29 (reference value ≤ 0.7), and the 24-hour intrathecal IgG synthesis rate was markedly increased at 65 mg/24 hours (reference value < 7.0 mg/24 hours), supporting intrathecal immunoglobulin synthesis. As shown in Table 2.

Main Laboratory Tests and Results

Table 2. The main laboratory tests and their respective results.

Main Laboratory Tests	Indicator	Main Test Results
Blood Routine	White Blood Cell Count	$3.34 \times 10^9/L$
	Neutrophil Count	$1.79 \times 10^9/L$
	Platelet Count	$102 \times 10^9/L$
Infectious Markers	Quantitative HBsAg	108.51 mIU/mL
	Hepatitis Be Antibody	0.04 COI

	Hepatitis B Core Antibody	6.73 COI
	Hepatitis C Antibody	13.50 COI
	Syphilis Treponemal Antibody	22.47 COI
	Kappa Light Chain	60.02 g/L
	Lambda Light Chain	2.62 g/L
Immunology	Immunoglobulin G	20.80 g/L
	Immunoglobulin M	2.25 g/L
	White Blood Routine	
	Cell Count in CSF	29×10 ⁶ /L
Cerebrospinal Fluid (CSF)	Cytology	Increased
	Cerebrospinal Fluid Syphilis Antibody	Increased ratio of monocytes and lymphocytes, suggesting inflammatory response.
	Syphilis Protein	(1:128) +
Serology	Syphilis Antibody (RPR, TPPA, TRUST)	(1:32) +
ANCA	pANCA	Positive
Brain MRI		Multiple small enhanced foci in the brain, new ventriculitis
Neurology	MMSE Score	23
	MOCA Score	12
	ADL Score	6
	AQP4	+

As shown in figure 1, important auxiliary examination: Brain MRI demonstrated a previous pontine infarction and multiple intracranial abnormalities. T2-weighted and FLAIR imaging showed an abnormal lesion in the left cerebellum. Magnetic resonance angiography suggested an aneurysmal abnormality involving the siphon segment of the left internal carotid artery. As shown in figure 2, Contrast-enhanced MRI demonstrated enhancement in the right cavernous sinus region and multiple intracranial enhancing lesions.

As shown in figure 3, Ophthalmological assessment showed markedly reduced visual acuity at presentation. Fundus examination demonstrated a suspected right fundus lesion and left retinal lattice degeneration, while optical coherence tomography showed normal bilateral macular thickness.

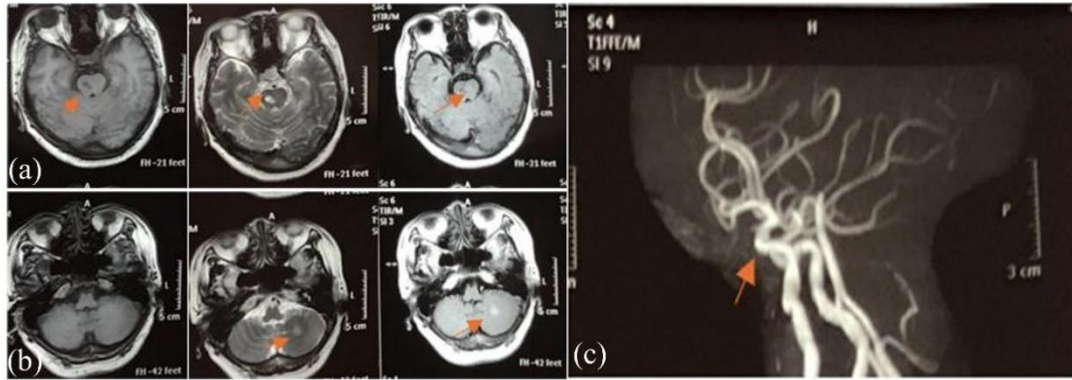


Figure 1 Brain MRI and magnetic resonance angiography. (a) Brain MRI showing a previous infarction in the right brainstem (arrow). (b) T2-weighted and FLAIR images showing an abnormal hyperintense lesion in the left cerebellum (arrow). (c) Magnetic resonance angiography showing a suspected aneurysmal abnormality involving the siphon segment of the left internal carotid artery (arrow).



Figure 2 MRI of brain enhancement: (Figure d) visible enhancement in the right cavernous sinus area compared with the left; (Figures e and f) multiple intracranial enhancement lesions (arrows), multiple invasion of the brain parenchyma, autoimmune or infection.

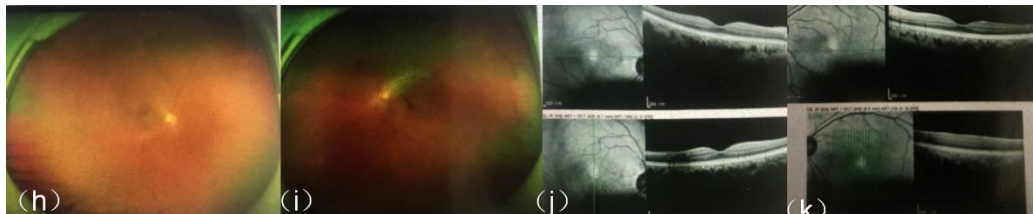


Figure 3 Ophthalmological imaging. (h,i) Fundus photographs obtained during ophthalmological assessment. The right fundus showed a suspected retinal abnormality, while lattice degeneration was noted in the left retina. (j,h) Optical coherence tomography showing no significant abnormality in bilateral macular thickness..

Diagnosis

Location diagnosis: 1. Neurosyphilis (polycranial neuritis) — Optic nerve, oculomotor nerve, facial nerve, vestibulocochlear nerve, abducens nerve;

Qualitative diagnosis: infectious lesion;

Differential diagnosis:

Several alternative diagnoses were considered because of the relapsing and multifocal cranial nerve involvement.

1. Neuromyelitis optica: Neuromyelitis optica spectrum disorder (NMOSD) was also considered because of the visual symptoms and the reported positive aquaporin-4 (AQP4) antibody result. However, the patient had no clinical evidence of myelopathy,

and neuroimaging did not demonstrate characteristic longitudinally extensive spinal cord lesions or a typical demyelinating pattern. The AQP4 antibody result therefore required cautious interpretation in the context of the overall clinical picture.

2. Multiple sclerosis: it is an immune-mediated inflammatory demyelinating disease of the central nervous system, which may be related to genetic, environmental, viral, and other factors, mainly involving the white matter of the central nervous system. It is manifested by extensive myelin demyelination and damage to a few keratinocytes, and the lesions have multiple and spatial necrosis. MRI mainly showed multispace, lateral paraventricular, cortical, and subcortical involvement. Common symptoms include optic nerve dysfunction, diplopia, limb sensory impairment, ataxia, bladder or rectal dysfunction, craniocerebral MRI, and spinal MRI. The patient only showed cranial nerve damage, no limb disorders, and no obvious multiple, multispace, cortical, and subcortical necroses caused by craniocerebral MRI (FLAIR). OB was positive in domestic studies and found that its positive rate in neurosyphilis is 95%^[6,7]. Furthermore, many studies in Europe and America have reported that the positive rate of OB in multiple sclerosis patients can be as high as 95%^[8]. However, the OB positive rate in the Asian population is only 30% to 60%^[9]. Positive OB could not be identified as MS.

3. Paraneoplastic syndrome refers to the endocrine, nerve, digestive, hematopoietic, bone, joint, kidney, and skin system lesions caused by the abnormal immune reaction of the tumor product and the corresponding clinical manifestations. It can involve any part of the nervous system, such as the brain, spinal cord, peripheral nerves, nerve-muscle joints, and muscles. Symptoms and signs can be fixed, and the characteristic symptoms include cerebellar degeneration, limbic lobe encephalitis, etc. Patients with cerebellar degeneration may have mild positive plantar reflexes except for vertigo, diplopia, and ataxia. CSF cellularity, increased protein and IgG levels, and corresponding or muscular lesions of peripheral nerves were seen on electrophysiological examination. The patients mainly had cranial nerve damage, cerebellum, and limbic lobe encephalitis, combined with tumor series and related tumor antibody tests were negative.

Treatment:

The patient's symptoms did not improve significantly after receiving nutritional nerve and circulation-improving therapy (Vitamin B6 tablets 10 mg p.o. t.i.d.; Mecobalamin injection 1 mg diluted in 250 mL of 0.9% sodium chloride injection, i.v. drip q.d.; Ginkgo diterpene lactone glucamine injection 5 mL diluted in 250 mL of 0.9% sodium chloride injection, i.v. drip q.d.) following admission. After the test and examination return, the polycranial neuritis caused by neurosyphilis was finally determined after joint consultation with the dermatology department, rheumatism immunology department, and ophthalmology department. Prednisone was subsequently administered for 3 days; (from the next day) an intravenous infusion of penicillin sodium 24 million U / d (4 million U every 4 hours) for 14 days; an intramuscular injection of 2.4 million U per week (on both hips); The patient was closely monitored for the development of a Jarisch–Herxheimer reaction during treatment. On the second day of treatment, the patient's vision improved, dizziness

resolved, right eye movement gradually recovered, and right-sided facial paralysis showed improvement. At discharge, visual acuity had improved to 1.0 in both eyes (VOD 1.0 and VOS 1.0). At approximately 3 weeks after treatment, her neurological symptoms had improved substantially. Subsequent follow-up was limited, with clinical information available for approximately 5 weeks after treatment. At telephone follow-up, visual acuity had improved to 1.2, right-sided facial weakness had further diminished and ocular movements were reported to be unrestricted.

Long-term neurological, ophthalmological and serological follow-up data were not available. Combined with the current symptoms, the hormone and penicillin treatment was effective.

It can be seen in figure 4 that the oculomotor nerve adduction distance gradually improved with time. After the start of hormone treatment, the recovery of the oculomotor nerve was slightly improved. After administering penicillin treatment (Penicillin treatment is indicated by the blue arrow), the condition improved significantly, indicating that the treatment was effective.

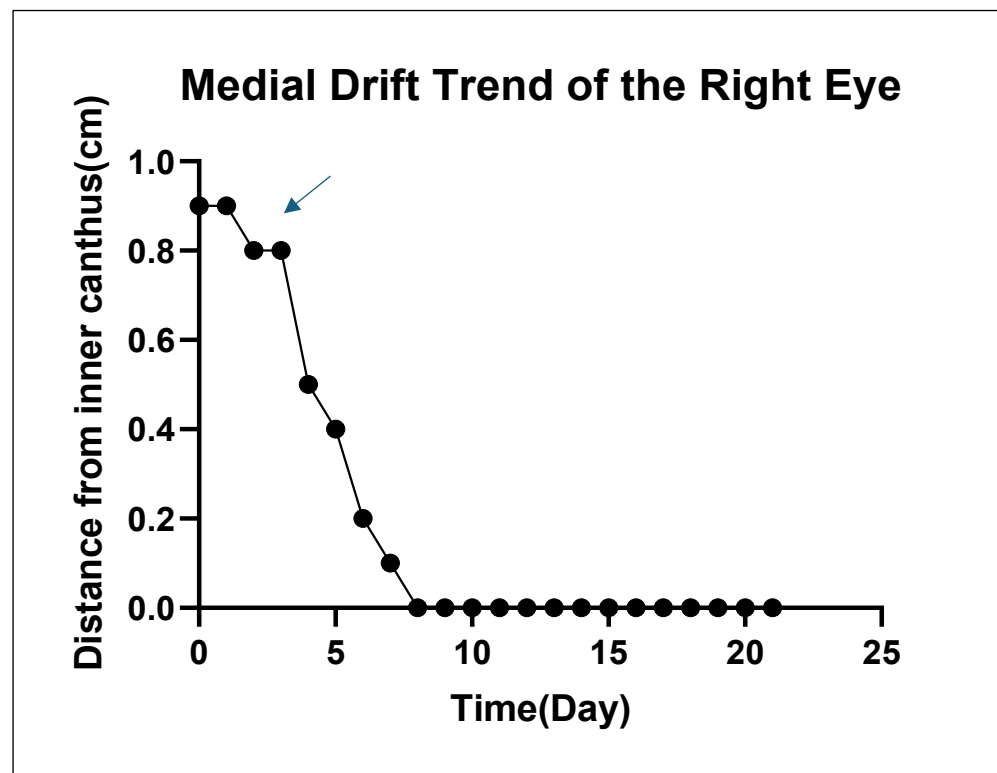


Figure 4. Medial drift trend of the right eye

It can be seen from figure 5 that the patient's condition was repeated, showing the repeated invasion of neurosyphilis. Following the treatment of penicillin, his vision was significantly improved, demonstrating the effectiveness of syphilis on penicillin, which further proved the diagnosis of neurosyphilis.

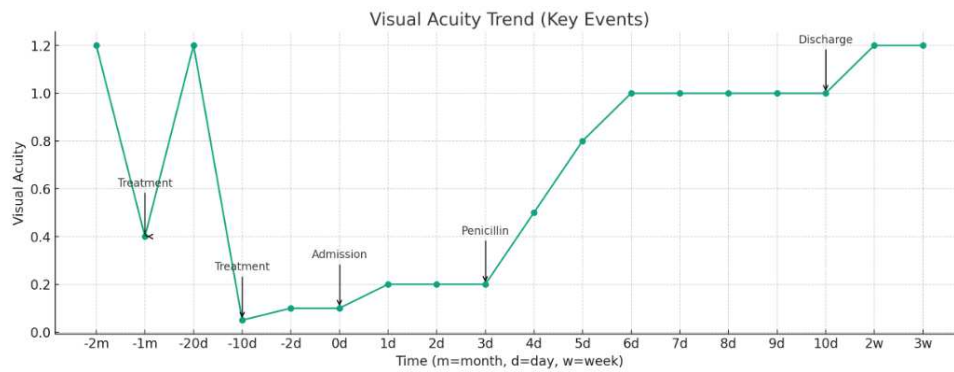


Figure 5 Visual Acuity trend (Key events)

3. Discussion

The main finding of this case is that neurosyphilis may present as a relapsing and progressive multiple cranial neuropathy, even in the absence of typical meningeal irritation. In this patient, the disease course began with facial nerve palsy, followed by oculomotor nerve involvement, and later progressed to optic, facial, oculomotor, abducens, and vestibulocochlear nerve dysfunction. This sequential and recurrent pattern, together with positive serum and cerebrospinal fluid syphilis tests, lymphocytic-predominant CSF inflammation, and contrast-enhanced MRI findings showing meningeal and cranial nerve involvement, strongly supported the diagnosis of neurosyphilis. The novelty of this case lies in the extensive involvement of five cranial nerves, which is uncommon in previously reported neurosyphilis cases, most of which involve only one to three cranial nerves. In addition, the initial partial response to corticosteroids may have temporarily masked the infectious nature of the disease, highlighting that steroid-responsive cranial neuropathy does not exclude neurosyphilis. Therefore, this case emphasizes the need to include neurosyphilis in the differential diagnosis of unexplained, recurrent, or multifocal cranial nerve palsies.

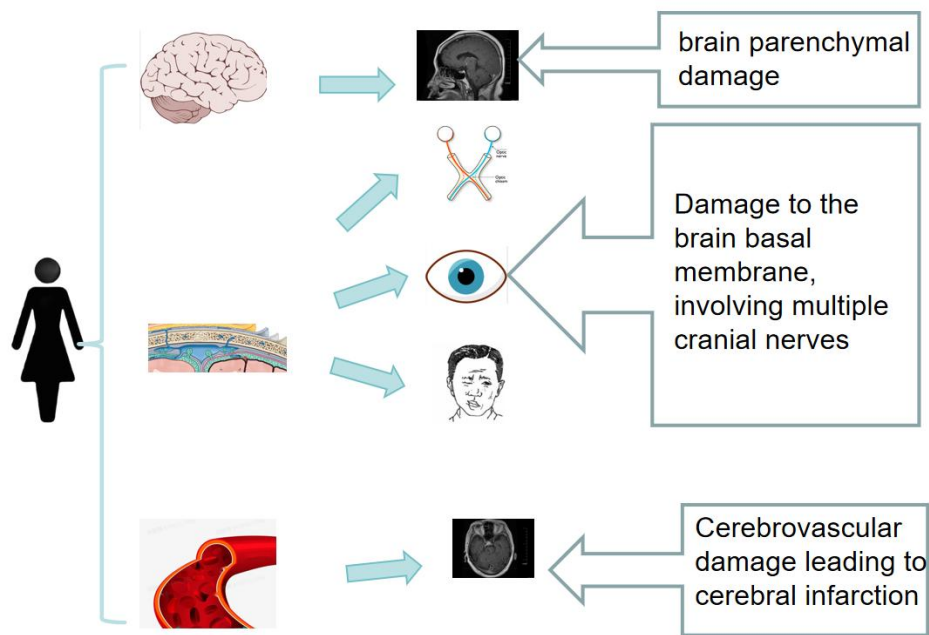
The CNS infection caused by central nervous system invasion has risen recently. After syphilis infringes on the nervous system, most of the manifestations are asymptomatic neurosyphilis in the early stage^[10]. If not actively treated, it gradually develops into symptomatic neurosyphilis, manifested as syphilis meningitis or meningeal cerebrovascular syphilis. Some studies have shown that about 13.5%~20.0% of early syphilis patients lacking standardized treatment can develop neurosyphilis, of which 4%~10% are symptomatic neurosyphilis^[11,12]. Therefore, the early diagnosis and treatment of syphilis are critical. Due to the different invasion sites of *treponema pallidum*, patients have different clinical symptoms, involving various diseases such as neurology, ophthalmology and psychiatry, so it is easy to misdiagnose or miss diagnosis in clinical practice. It has also been reported that the misdiagnosis rate of neurosyphilis can reach 47%~57.1%.^[13] In the present case, the patient initially presented with left facial nerve palsy, followed by diplopia and left ptosis, and later developed visual impairment, bilateral facial nerve neuritis, abducens nerve palsy, and vestibulocochlear symptoms. This relapsing and stepwise progression involving multiple cranial nerves was the most important clinical clue. Although each episode could initially be explained by common conditions, such as idiopathic facial nerve palsy, neuromyelitis optica spectrum disorder, viral neuritis, multiple sclerosis, or cavernous sinus-related

inflammation, the recurrent involvement of different cranial nerves could not be fully explained by a single isolated lesion. Brain MRI further showed multiple silent infarctions, microbleeding lesions, meningeal enhancement, cranial nerve enhancement, and possible vascular abnormalities, suggesting that the disease process was not limited to a peripheral nerve disorder. Instead, these findings indicated a more diffuse intracranial process involving the meninges, cranial nerves, and cerebral vessels.^[14]

Laboratory and imaging findings in this case provided the decisive evidence. The high syphilis titers in both serum and CSF supported active central nervous system infection. CSF cytology showed lymphocyte predominance, which was more consistent with chronic inflammatory or infectious meningitis than with acute bacterial infection. MRI findings, including meningeal and cranial nerve enhancement, white matter lesions, silent infarctions, and microbleeding lesions, further supported a meningovascular and basilar meningeal mechanism^[15]. Although AQP4 antibody positivity raised the possibility of neuromyelitis optica spectrum disorder, however, combined with the magnetic resonance examination, demonstrated no demyelination and inflammatory changes of the long spinal cord and brain stem. Thus absence of typical longitudinally extensive spinal cord lesions or brainstem demyelinating lesions made this diagnosis less convincing. Nevertheless, the AQP4 antibody result should be interpreted cautiously. Concurrent AQP4-positive NMOSD and neurosyphilis has previously been reported, indicating that AQP4 positivity should not automatically be attributed to neurosyphilis alone^[16]. In the absence of detailed assay information and long-term neurological follow-up, concomitant or evolving autoimmune demyelinating disease cannot be completely excluded. This case has several practical implications. Neurosyphilis should be considered in patients with unexplained recurrent or multifocal cranial neuropathies, particularly when different cranial nerves are affected sequentially or when neurological deficits are accompanied by ocular or vestibular symptoms, cerebrospinal fluid inflammation or unexplained intracranial abnormalities on MRI. In such circumstances, serum syphilis testing and, when clinically indicated^[17], cerebrospinal fluid examination may provide important diagnostic information.

The temporary improvement in ptosis after corticosteroid treatment also illustrates a potential diagnostic pitfall. A clinical response to corticosteroids does not establish an immune-mediated aetiology and should not preclude investigation for an underlying infection when the subsequent clinical course is atypical or relapsing. In the present patient, the continued emergence of deficits involving different cranial nerves prompted further investigation and ultimately led to the diagnosis of neurosyphilis.

In conclusion, neurosyphilis may present with relapsing and sequential multiple cranial neuropathies and can mimic vascular, neuromuscular and inflammatory neurological disorders. Recognition of the evolving pattern of cranial nerve involvement, together with appropriate serum and cerebrospinal fluid testing, was central to the diagnosis in this case. Early recognition is clinically important because appropriate antimicrobial treatment may result in substantial neurological improvement..



4. Learning points

- 1) Neurosyphilis should be considered in patients with unexplained, recurrent or multifocal cranial neuropathies, particularly when different cranial nerves are involved sequentially.
- 2) Positive oligoclonal bands or other evidence of intrathecal immune activation should not automatically be interpreted as evidence of a primary demyelinating disorder; infectious causes should also be considered in the appropriate clinical context.
- 3) Temporary improvement after corticosteroid treatment does not exclude an underlying infection and should not delay further investigation when neurological symptoms recur or evolve.
- 4) Appropriate penicillin treatment may lead to substantial neurological recovery, highlighting the importance of early recognition of neurosyphilis

Consent for publication

Written informed consent was obtained from the patient for publication of this case report, including the clinical information and accompanying images.

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