Case Reports in Neurology

Case Rep Neurol 2026;18:105–112
DOI: 10.1159/000550579
Received: September 2, 2025
Accepted: January 6, 2026
Published online: January 19, 2026

Single Case – Brain Injury

Tapia's Syndrome following Noninvasive Continuous Positive Airway Pressure Therapy: A Case Report

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Keywords

Tapia's syndrome · Continuous positive airway pressure · Cranial nerve palsy · Case report

Abstract

Background: Tapia's syndrome is a rare neurological condition defined by concurrent unilateral paralysis of the vagus (cranial nerve X) and hypoglossal (cranial nerve XII) nerves. It is most commonly reported as an iatrogenic complication of procedures involving airway manipulation, such as orotracheal intubation. This report describes a unique case of Tapia's syndrome with a temporal association to the initiation of noninvasive continuous positive airway pressure (CPAP) therapy. ***Case Presentation:*** A 66-year-old female presented with a four-day history of acute-onset dysphonia, dysphagia, and right-sided tongue deviation. Her symptoms began shortly after initiating CPAP therapy with a full-face mask for newly diagnosed obstructive sleep apnea. She had also recently received multiple vaccinations. Clinical examination revealed right-sided vagus and hypoglossal nerve palsies, and laryngoscopy confirmed right vocal cord paralysis. Extensive diagnostic evaluation, including magnetic resonance imaging and angiography of the brain and neck, effectively excluded central nervous system pathologies such as stroke, demyelinating disease, or mass lesions and diagnosis of Tapia's syndrome was made. The patient was managed by discontinuing CPAP and administering a course of oral corticosteroids, alongside speech and swallowing therapy. She experienced a near-complete resolution of her symptoms over 6 weeks. ***Conclusion:*** This case suggests that Tapia's syndrome can be a rare complication of noninvasive airway support. A multifactorial etiology involving mechanical nerve compression from the CPAP apparatus, potentially compounded by an immune-mediated nerve sensitization from recent vaccinations, should be considered in the differential diagnosis of lower cranial neuropathies.

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Introduction

Tapia's syndrome is the eponymous term for a distinct clinical entity characterized by simultaneous, ipsilateral paralysis of the vagus (cranial nerve X) and hypoglossal (cranial nerve XII) nerves [1]. First described in 1904 by Spanish otolaryngologist Antonio Garcia Tapia in a bullfighter who sustained a neck injury, the condition is also known as "matador's syndrome" [2]. The combined neural deficit results in a classic clinical triad: dysphonia due to vocal cord paralysis (CN X), dysphagia from impaired pharyngeal and laryngeal function (CN X), and deviation of the protruded tongue toward the paralyzed side due to unopposed action of the contralateral genioglossus muscle (CN XII) [3].

The syndrome is most frequently iatrogenic, arising as a complication of procedures that involve airway manipulation, particularly orotracheal intubation for general anesthesia [4, 5]. The underlying pathophysiology is believed to be a peripheral nerve injury caused by direct compression or stretching of the vagus and hypoglossal nerves in their extracranial course, where they lie in close anatomical proximity [6]. Other documented causes include cervical spine surgery, direct trauma, and compression from tumors at the skull base or in the neck [7, 8].

This report presents a novel case of Tapia's syndrome with a compelling temporal association to the initiation of noninvasive continuous positive airway pressure (CPAP) therapy, an etiology not previously documented in the medical literature. Furthermore, the patient had received multiple vaccinations in the week prior to symptom onset, raising the possibility of an immune-mediated process acting as a predisposing factor. This case aims to expand the recognized etiological spectrum of Tapia's syndrome and to underscore the importance of considering mechanical factors from noninvasive devices in the evaluation of acute cranial neuropathies. This case report was written in accordance with the CARE (Case Report) Checklist guidelines to ensure accuracy, transparency, and completeness. The CARE Checklist for this case report is available as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000550579).

Case Presentation

Patient Information

A 66-year-old female with a past medical history of hyperlipidemia, osteoporosis, and recently diagnosed obstructive sleep apnea (OSA) presented to the emergency department. She reported a 4-day history of progressively worsening hoarseness, slurred speech (dysarthria), difficulty swallowing solids and liquids (dysphagia), and a noticeable deviation of her tongue to the right upon protrusion. The patient had no prior history of neurological deficits, neck trauma, or surgical procedures involving the head or neck.

Clinical Findings

On physical examination, the patient was alert, oriented, and afebrile. The neurological examination localized the deficits to the right lower cranial nerves. Examination of cranial nerve X (vagus) revealed a hoarse, breathy voice (dysphonia), a diminished gag reflex on the right, and deviation of the uvula to the left upon phonation. Examination of cranial nerve XII (hypoglossal) demonstrated marked deviation of the tongue to the right upon protrusion, with visible fasciculations and mild atrophy of the right side of the tongue (Fig. 1). The remainder of the cranial nerve examination, as well as motor, sensory, reflex, and cerebellar testing, was entirely within normal limits. An urgent consultation with an otolaryngologist included a flexible laryngoscopy, which confirmed paralysis of the right vocal cord in the paramedian position.

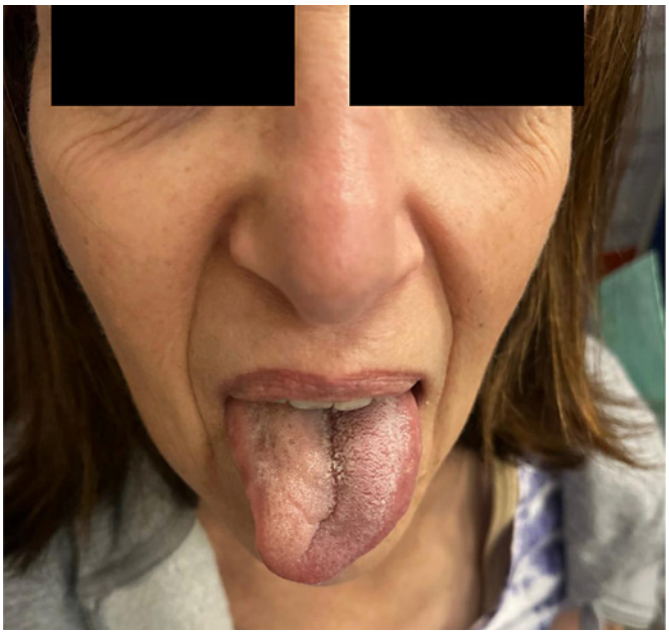

Fig. 1. Clinical photograph demonstrating right-sided tongue deviation upon protrusion. The tongue deviates toward the affected side (right) due to unopposed action of the left genioglossus muscle, consistent with right hypoglossal nerve (CN XII) palsy. Photograph obtained with written informed consent.

CPAP Device Specifications

The patient was prescribed a ResMed AirSense 11 CPAP machine with a ResMed AirTouch F20 full-face mask. The device was configured with a fixed pressure of 12 cm H_2O, with ramp time set to Auto and humidification on Climate Control Auto (maintaining approximately 85% relative humidity). The full-face mask was professionally fitted by a certified sleep technician. These represent standard therapeutic settings for moderate OSA; however, the full-face mask design, which features straps that cross the upper neck and angle of the mandible, may have contributed to external compression of the vulnerable anatomical region where cranial nerves X and XII course in close proximity.

Timeline of Events

The patient's symptoms emerged in close temporal proximity to the initiation of CPAP therapy and a series of recent vaccinations. The key events are summarized in Table 1.

Diagnostic Assessment

The diagnostic process focused on excluding central and systemic causes to confirm a peripheral nerve lesion. Laboratory investigations, including a complete blood count, comprehensive metabolic panel, and inflammatory markers (C-reactive protein and erythrocyte sedimentation rate), were all within normal limits. An extensive panel for infectious and autoimmune etiologies, including Lyme serology, was also negative.

To rule out a central nervous system cause, comprehensive neuroimaging was performed. Magnetic resonance imaging and magnetic resonance angiography of the brain and neck with gadolinium contrast were unremarkable. These studies showed no evidence of an acute brainstem ischemic or hemorrhagic event, demyelinating plaques, space-occupying lesions at the skull base, or vascular abnormalities such as carotid or vertebral artery dissection. The absence of findings on these critical imaging studies was a pivotal step in the

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Table 1. Timeline of clinical events

Time point	Event
Day 8	Patient receives influenza and Tdap vaccinations
Day 1	Patient receives pneumococcal and COVID-19 vaccinations
Day 0	Patient initiates CPAP therapy (ResMed AirSense 11) with a ResMed AirTouch F20 full-face mask at a pressure of 12 cm H_2O
Day 1	Onset of mild hoarseness noted by the patient
Day 4	Presentation to the emergency department with worsening hoarseness, dysarthria, dysphagia, and right-sided tongue deviation
Day 5	Admitted to hospital, CPAP therapy discontinued, diagnostic workup initiated
Day 6	Initiated on high-dose oral corticosteroids (prednisone 60 mg daily)
Week 2	Marked improvement in dysphonia and dysphagia
Week 5	Complete resolution of tongue deviation
Week 6	Follow-up appointment with near-complete resolution of all symptoms. Patient opted to permanently discontinue CPAP therapy; treatment plan revised to include mandibular advancement device and positional therapy

diagnostic pathway, effectively localizing the pathology to the peripheral course of the nerves.

The diagnosis of Tapia's syndrome was therefore made by exclusion, based on the characteristic clinical phenotype and the comprehensive workup that ruled out alternative diagnoses. The differential diagnosis is summarized in Table 2.

Therapeutic Intervention and Outcomes

Upon admission, the suspected offending agent – the CPAP machine – was immediately discontinued to eliminate the source of potential mechanical nerve stress. The patient was started on a course of high-dose oral corticosteroids (prednisone 60 mg daily, tapered over 2 weeks) with the aim of reducing potential nerve inflammation and edema.

A multidisciplinary approach was initiated, including a prompt referral to speech and swallowing therapy. The therapy team implemented compensatory strategies and exercises to manage dysphagia, mitigate the risk of aspiration, and improve articulatory precision.

The patient's clinical course was favorable. She demonstrated gradual but steady improvement, with a noticeable reduction in hoarseness and swallowing difficulty within 2 weeks. Her tongue deviation had fully resolved by the fifth week of follow-up. At her 6-week outpatient review, she reported a near-complete resolution of all symptoms, with only minor, subjective residual dysphagia. To prioritize her safety and prevent a recurrence of neurological symptoms, the patient opted to permanently discontinue CPAP therapy. Her treatment plan for OSA has been revised to include noninvasive alternatives, such as mandibular advancement devices and positional therapy.

Discussion

This case is, to our knowledge, the first to report an association between Tapia's syndrome and the use of noninvasive CPAP therapy. While CPAP is a cornerstone of OSA management and is generally considered safe, this case highlights a rare but significant potential complication. The mechanism likely involves external nerve compression, a

Table 2. Differential diagnosis and reasoning

Diagnosis	Supporting features	Refuting features	Conclusion
Brainstem (medullary) stroke	Acute onset of cranial nerve palsies, dysphagia, dysarthria	Absence of other brainstem signs (e.g., contralateral sensory loss, ataxia), normal magnetic resonance imaging/magnetic resonance angiography of the brain	Ruled out
Skull base neoplasm/ meningitis	Can cause compression of multiple cranial nerves	Acute onset without a progressive history, absence of systemic signs, normal contrast-enhanced magnetic resonance imaging	Ruled out
Guillain-Barré syndrome (GBS) variant	Can present with bulbar palsy; post-vaccinal association possible	Isolated CN X/XII palsy is atypical; absence of limb weakness, areflexia, or sensory deficits	Unlikely
Infectious/ autoimmune neuropathy	Can cause cranial neuropathies	No systemic signs or symptoms, negative infectious and autoimmune workup, normal inflammatory markers	Unlikely
Tapia's syndrome (peripheral)	Classic clinical phenotype of isolated, ipsilateral CN X and XII palsy, strong temporal link to a new mechanical stressor (CPAP)	None	Diagnosis of exclusion

phenomenon recognized in other contexts, such as external compression headaches caused by overly tight CPAP mask straps that irritate superficial nerves like the trigeminal or occipital nerves [9]. The patient's use of a full-face mask, which often has straps that cross the upper neck and angle of the mandible, combined with a relatively high-pressure setting of 12 cm H_2O, supports a mechanical etiology.

The specific pattern of neurological deficit – involving CN X and XII while sparing CN IX and XI – provides a precise anatomical clue to the location of the injury. Cranial nerves IX, X, and XI exit the skull together through the jugular foramen, whereas CN XII exits separately via the hypoglossal canal [10]. A lesion at the skull base would likely have affected CN IX and/or XI as well. However, in the extracranial parapharyngeal space, the vagus and hypoglossal nerves course in close proximity to one another, anterior to the transverse process of the first cervical vertebra (C1), and near the greater horn of the hyoid bone [11]. In this location, CN IX and XI have already diverged onto different paths. This specific anatomical "choke point" is uniquely vulnerable to external pressure, which could selectively compress CN X and XII against these underlying bony structures, perfectly explaining the isolated nature of the palsy seen in this patient [12]. The pressure exerted by the straps of a full-face CPAP mask can be directly transmitted to this vulnerable region.

The role of the patient's recent vaccinations warrants consideration [13]. The temporal proximity of four vaccinations within 8 days of symptom onset is striking. Cranial neuropathies are recognized, albeit rare, adverse events following various vaccinations, including those for COVID-19, with onsets reported within days of administration [14]. The proposed mechanism is immune-mediated, potentially involving molecular mimicry or the activation of quiescent autoreactive T cells that lead to transient nerve inflammation [15]. It is plausible that the vaccinations induced a subclinical, inflammatory state that rendered the vagus and hypoglossal nerves more susceptible to a mechanical injury that might otherwise have been tolerated. This "two-hit" model, where an immunological event creates a state of heightened vulnerability (the first hit) and a subsequent mechanical stressor provides the

Table 3. Differential diagnosis of lower cranial nerve syndromes

Syndrome	Cranial nerves involved	Key distinguishing features and anatomical location
Tapia's syndrome	X, XII	Peripheral injury sparing CN IX and XI. Presents with vocal cord and tongue paralysis but preserved soft palate function. Location is extracranial, where CN X and XII run in close proximity
Vernet's syndrome (jugular foramen)	IX, X, XI	Lesion confined to the jugular foramen, sparing CN XII. Presents with hoarseness, dysphagia, and trapezius weakness, but no tongue deviation
Collet-Sicard syndrome	IX, X, XI, XII	Lesion affects both the jugular foramen and hypoglossal canal. Involves all four lower cranial nerves, adding tongue paralysis to Vernet's syndrome symptoms
Villaret's syndrome	IX, X, XI, XII, + sympathetic chain	Same nerve involvement as Collet-Sicard, plus Horner's syndrome (ptosis, miosis, anhidrosis), indicating a lesion in the retroparotid space affecting the sympathetic trunk
Schmidt's syndrome	X, XI	Presents with prominent trapezius and sternocleidomastoid weakness alongside vocal cord paralysis. Considered a variant of Vernet's syndrome
Jackson's syndrome	X, XII	A medullary (brainstem) syndrome involving the nuclei of CN X and XII, often associated with contralateral hemiplegia due to proximity to the pyramidal tracts

precipitating insult (the second hit), offers a compelling explanation for the occurrence of this rare syndrome in this specific clinical context. A key part of the diagnostic process for multiple cranial neuropathies is distinguishing between the various eponymous syndromes based on the specific combination of nerves affected, which in turn localizes the lesion anatomically (Table 3).

The primary strength of this report is the documentation of a novel etiology for Tapia's syndrome, supported by a thorough diagnostic evaluation that systematically excluded more common causes. The successful outcome after removing the suspected mechanical trigger and changing the CPAP interface further strengthens the proposed hypothesis. The main limitation is that, as a single case report, a definitive causal relationship cannot be established; the associations remain temporal. The therapeutic effect of corticosteroids cannot be disentangled from the natural history of recovery or the benefit of discontinuing CPAP.

Conclusion

This case of Tapia's syndrome illustrates a novel and clinically important potential complication of noninvasive CPAP therapy. It suggests that clinicians should maintain a high index of suspicion for peripheral nerve compression injuries even with noninvasive medical devices. The case further posits that a patient's recent clinical history, including significant immunological events such as multiple vaccinations, may create a window of vulnerability to otherwise benign mechanical insults. Early diagnosis, prompt removal of the suspected mechanical stressor, and a multidisciplinary rehabilitative approach are paramount for achieving favorable patient outcomes.

Statement of Ethics

Ethics approval was not required for this study, in accordance with local legislation and institutional requirements. Written informed consent for publication of the patient's clinical details and accompanying images was obtained.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Funding Sources

This study was not supported by any sponsor or funder.

Author Contributions

O.P.: main author, case write-up, literature review, and manuscript drafting. D.R.A.V.: literature review and manuscript editing. F.S.: patient care, clinical input, and manuscript revision. M.B.: patient care, supervision, and manuscript revision. All authors reviewed and approved the final manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this published article. Further details are available from the corresponding author upon reasonable request.

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