

Vagal Mononeuritis Associated with Zoster Sine Herpete: A Case Report and Review of the Literature

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Abstract

We report a case of varicella zoster–induced vagal mononeuritis presenting with right-sided laryngeal paralysis. The patient reported a localized burning sensation in the left anterior thoracic T4–T5 dermatomes without cutaneous eruption two weeks prior to the development of dysphonia, consistent with zoster sine herpete. Cerebrospinal fluid analysis obtained through lumbar puncture detected varicella zoster viral deoxyribonucleic acid, confirming the diagnosis of herpes zoster infection. Treatment with intravenous acyclovir and glucocorticoids resulted in rapid clinical improvement and eventually complete neurological recovery. This case highlights the importance of considering varicella zoster virus

reactivation in the differential diagnosis of recurrent laryngeal nerve paralysis.

Introduction

Varicella Zoster Virus (VZV)-associated laryngeal paralysis results from viral reactivation within the sensory ganglia of the vagus nerve [1]. While shingles is caused by reactivation of VZV, which remains dormant in the dorsal root ganglion and is characterized by cutaneous eruptions, Cranial Nerve (CN) infections arise from reactivation within sensory ganglia such as the trigeminal ganglion (CN V), the geniculate ganglion (CN VII), or the superior and inferior vagal (nodose) ganglia (CN X) [2-4]. Reactivation confined to cranial nerve ganglia

without involvement of the dorsal root ganglia results in the absence of dermatomal cutaneous manifestations and is termed Zoster Sine Herpete (ZSH) [5]. This atypical presentation poses significant diagnostic challenges and often leads to misdiagnosis and delayed treatment. Varicella zoster induced-vagal mononeuritis without mucocutaneous findings is rare but most likely underreported. Laryngopharyngeal VZV presents with vesicular or ulcerative lesions involving the laryngopharyngeal mucosa and, in some instances, with concurrent cutaneous or otologic manifestations [1,6].

Case Presentation

A 67-year-old male was hospitalized for rapidly progressive sore throat, dysphagia, dysphonia, and right-sided neck pain. Physical examination revealed no stridor, oropharyngeal lesions, or abnormalities of the external ears or tympanic membranes. The patient also reported flu-like symptoms and a localized burning sensation in the left anterior thoracic T4–T5 dermatomes without cutaneous eruption two weeks prior. Laboratory evaluation showed no leukocytosis and normal renal and liver function. Computed Tomography (CT) of the neck with intravenous contrast showed no abscesses or lymphadenopathy. Magnetic Resonance Imaging (MRI) of the brain with intravenous gadolinium showed no evidence of ischemic or hemorrhagic infarcts, brain abscesses, or midline shift. Blood and urine cultures yielded no pathogens. Treatment with IV dexamethasone corticosteroids alone was attempted but resulted in no clinical improvement. Further evaluation with flexible laryngoscopy and Esophagogastroduodenoscopy (EGD) revealed right-sided vocal cord paralysis with incomplete glottic closure and no pharyngeal or esophageal mucosal

lesions. Cerebrospinal Fluid (CSF) analysis demonstrated mild pleocytosis with a positive VZV Polymerase Chain Reaction (PCR). Intravenous acyclovir (10 mg/kg every 8 hours) was initiated, resulting in marked improvement in hoarseness and dysphagia. Antiviral therapy was continued for two weeks with sustained clinical improvement. Sustained clinical response was observed thirteen months after therapy had concluded.

Discussion

Varicella-zoster virus is an alpha-herpesvirus of the genus Varicellovirus that causes chickenpox. After primary infection, VZV remains dormant and can reactivate years later to cause shingles [7,8]. The virus initially infects epithelial cells in the upper respiratory tract and subsequently gains access to the tonsils and local lymph nodes, where it infects T-cells [9]. These activated T-cells transport the virus through the bloodstream, leading to systemic dermal manifestations and the characteristic vesicular rash that appears after a 10 to 21-day incubation period [9]. The virus then establishes latency in sensory ganglia via retrograde axonal transport [9]. When virus-specific immunity declines, VZV reactivates from the dorsal root, cranial nerve, or autonomic ganglia and presents as a painful dermatomal rash [10]. Once the diagnosis is established, treatment includes antiviral medications such as acyclovir, valacyclovir, and famciclovir. These medications do not eliminate latent infection but reduce disease severity [11].

Isolated VZV vagal mononeuritis presents with hoarseness and dysphagia and may progress to aphonia and acute weakness of the soft palate [12]. Zoster sine herpete is a rare presentation, particularly when isolated to the vagus nerve. In our patient, laryngeal paralysis without dermal manifestations

delayed diagnosis, which was ultimately confirmed by positive CSF VZV PCR. The absence of additional findings beyond laryngopharyngeal involvement makes this case unique, and early antiviral treatment led to recovery of vocal cord

function and swallowing. We reviewed 19 reported cases of VZV-associated laryngeal paralysis and laryngopharyngeal cranial neuropathies, summarized in **Table 1**.

Table 1: Summary of the characteristics of patients with VZV-associated laryngeal paralysis and laryngopharyngeal cranial neuropathies [1,12-27].

Ref.	Demographics / Risk Factors	Presenting Symptoms	Findings	Treatment	Outcome	Diagnostics
1	65F; poorly controlled DM	Dysphagia, odynophagia, rightotalgia, and otorrhea	Vesicular lesions of lingual epiglottis and right aryepiglottic fold, right arytenoid edema, right true vocal fold hypomobility, insensate right hemilarynx, and right conchal bowl erythema	IV ACV+ steroids + injection medialization	Complete resolution of dysphagia and dysphonia after 2.5 years of therapy	Intraoperative cultures were positive for VZV
1	57F; MM	Dysphagia, odynophagia, diplopia, vertigo, and hearing loss	Left pharyngeal weakness, insensate hemilarynx, left vocal fold immobility, vesicular rash involving left conchal bowl, cheek, nasal ala, soft palate, and oropharynx	IV ACV+ high-dose corticosteroids ; repeated vocal fold medialization	Deceased due to hematologic malignancy	Cutaneous cultures demonstrated VZV

1	25F; SLE	Fever, rash, blurred vision, odynophagia, and dyspnea	Palatal hypomobility, bilateral vocal fold paralysis, vesicular rash in trigeminal V1 distribution, laryngeal and supraglottic vesicles	IV ACV + corticosteroids + tracheostomy + lifelong VCV	Persistent dysphonia, chronic cough, and dysphagia at 1.5 years	MRI showed scattered lesions in subcortical white matter, pons, and medulla
						CSF VZV PCR was positive
12	50F; no significant PMH	Hoarseness, dysphagia, andotalgia	Left vocal cord paralysis, soft palate weakness, and mild facial palsy	IV ACV + prednisolone	Full recovery	MRI showed no lesions of brain stem
						Serum VZV IgG showed significant increase from 10.5 AI to >128 AI
13	37M; no significant PMH	Pharyngalgia, hoarseness, and dysphagia	Left soft palate and vocal cord paralysis, reduced gag reflex, and enanthem on left epiglottis and aryepiglottic fold	ACV + steroid	Full recovery within 1 year	Serum VZV IgM and IgG antibodies were increased

14	76M; history of poliomyelitis	Dysphonia, right shoulder weakness, and headache	Right vocal cord palsy, vesicular lesions in right concha	ACV + prednisolone	N/A	MRI showed enhancement of right lower cranial nerves and jugular foramen lesion
						Serum VZV IgG and IgM were positive
15	64M; no significant PMH	Progressive dysphagia, dysphonia, shoulder weakness, and scalp dysesthesia	Right vocal cord paresis, sequential CN IX–XII, and C2 involvement	IV ACV + vocal cord medialization	Significant improvement in 6 months	MRI showed nonspecific lesion near petrous apex; PET scan revealed increased uptake in jugular foramen
						Serum serologies for VZV IgM and IgG were positive
16	36F; no significant PMH	Sore throat, lymphadenopathy, dysphagia, and hoarseness	Left soft palate paresis and blisters on left aryepiglottic fold	PO VCV (3 g/day x 7 days)	Full resolution	CT showed enhancement of left CN IX–X complex
						Serum VZV IgG and IgM were significantly elevated

17	48F; no significant PMH	Hoarseness, dysphagia, left neck pain	Left vocal cord paralysis, and impaired laryngeal elevation	IV ACV followed PO VCV + prednisone taper	Full recovery	Elevated serum anti-VZV IgM titers
18	76F; no significant PMH	Dysphagia, and mild dysphonia	Paralysis of the pharynx on the right and incomplete closure of the vocal cords	IV ACV, 750 mg Q8h x 32 days	Significant improvement in 8 months	CSF VZV PCR positive with lymphocytic pleocytosis
19	74M; no significant PMH	Left facial weakness, dysphagia, and hoarseness	Left peripheral facial nerve paralysis, prolapsed left soft palate, inflamed granulation tissue in the left external ear canal, left supraglottic lesion, and left sensorineural hearing loss	IV ACV	Pharyngeal paralysis and dysphagia resolved over a 4-week period. The unilateral vocal cord paralysis and sensorineural hearing loss did not recover	Serological VZV testing positive
20	62F; history of Ramsay Hunt Syndrome	Hoarseness and dysphagia	Paresis of the soft palate on the right side with left deviated uvula and mixed type hearing loss	ACV+ prednisolone	Partial clinical recovery	Serum anti-VZV IgM elevated
21	57M; no significant PMH	Dysphagia and right dysphonia	Right posterior auricular lesions appeared 5 days after dysphagia	IV FCV + steroids	Complete resolution with no sequelae	Skin biopsy demonstrated VZV PCR positive

22	74F; history of Barrett's Esophagus, right medial temporal lobe meningioma, and corpus callosum infarct	Hoarseness and dysphagia	Vesicles on right antihelix, mild swelling of right auricle, and vocal cord paralysis	IV VCV + steroids + artificial tears + right eye lubricant patch	Vocal cord paralysis persisted 4 months after diagnosis	EGD, head CT, head CT arteriogram, head MRI, otolaryngology exam, electromyography No PCR was performed
23	41M; well-controlled DM	Fever and right otalgia	Vesicles on auricle and larynx	IV FCV + steroids	Complete resolution of symptoms and vesicular lesions	CSF VZV PCR positive
24	48M; no significant PMH	Right otalgia, odynophagia, pharyngeal swelling, dizziness, hiccups, and facial palsy	Vesicles on right auricle, external auditory canal, ipsilateral jaw, and perioral region and cheek	IV ACV + steroids + wet dressings + ACV cream + mupirocin ointment	Swallowing improved by discharge; no improvement in other symptoms; MRI 2 weeks later revealed damage to vestibulocochlear nerve	CSF VZV PCR positive

25	64F; PMH significant for hypertension, hyperlipidemia, prediabetes, peripheral artery disease, and migraines	Dysphagia, dysphonia, cough, sore throat, and rhinorrhea	Complete absence of mucosal or skin lesions	IV ACV + steroids	Improvement in headache, sore throat, rhinorrhea but dysphagia and dysphonia remained for 4 weeks	CSF VZV PCR positive
26	80M; no significant PMH	Sore throat, dysphagia, and hoarseness	Unilateral vocal cord paralysis with pooling of secretions, mucosal vesicles on the hemilarynx	IV VCV + steroids	Complete resolution of symptoms with no sequelae	CSF VZV PCR positive
27	70M; history of Invasive anal squamous cell carcinoma treated with excision and chemo-radiation	Right-sided neck pain, sub-mandibular lymphadenopathy	Dysphonia, dysphagia, and trapezius muscle weakness	IV ACV + IV VCV + steroids	Dysphagia and dysphonia improved but patient continued to have neuropathic pain, and scalene and trapezius muscle atrophy	VZV antibody titers positive

Abbreviations: ACV, acyclovir; AI, antibody index; CN, cranial nerve; CSF, cerebrospinal fluid; CT, computed tomography; DM, diabetes mellitus; EGD, esophagogastroduodenoscopy; FCV, famciclovir; F, female; IgG, immunoglobulin G; IgM, immunoglobulin M; IV, intravenous; M, male; MM, multiple myeloma; MRI, magnetic resonance imaging; PCR, polymerase chain reaction; PET, positron emission tomography; PMH, past medical history; PO, per os; SLE, Systemic Lupus Erythematosus; VCV, valacyclovir; VZV, varicella zoster virus.

Conclusion

Our case emphasizes the need for clinicians to maintain a high index of suspicion for VZV reactivation in patients presenting with unexplained cranial nerve palsies and laryngopharyngeal dysfunction, even when dermatologic findings and imaging studies are unremarkable. Cerebral spinal fluid analysis can facilitate early detection and prompt initiation of antiviral therapy, which may lead to complete neurological recovery and prevent long-term morbidity.

Conflict of Interests

The authors of this case report no conflict of interest in the publication of this paper.

Informed consent for publication was obtained from the patient.

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