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Speech and Swallowing Impairments in Herpes Zoster: A Case Report

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Abstract

Herpes zoster (HZ), caused by reactivation of varicella-zoster virus, can extend beyond dermatological manifestations to involve cranial nerves, resulting in complex speech and swallowing deficits. This case report presents a 54-year-old male with herpes zoster–related cranial neuropathy, who developed unilateral vocal fold paralysis, dysphonia, and dysphagia. Comprehensive assessment included perceptual, acoustic, endoscopic, and patient-reported outcome measures. Perceptual voice evaluation revealed severe dysphonia with breathiness, asthenia, strain, and reduced loudness, confirmed by high Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) scores and GRBAS ratings. Acoustic analysis showed elevated jitter (1.54%) and shimmer (8.64%), reduced harmonic-to-noise ratio (14.12), and shortened maximum phonation durations (2–3 seconds), indicating glottic insufficiency. The Voice Handicap Index (VHI) reflected moderate psychosocial burden. Swallowing assessments revealed severely reduced quality of life (SWAL-QOL = 109) and moderate dysphagia handicap (DHI = 49), highlighting nutritional and social challenges. Rigid laryngoscopy confirmed left vocal fold paralysis, pharyngeal wall ulceration, and herpes zoster lesions on the arytenoid. Fiberoptic Endoscopic Evaluation of Swallowing (FEES) revealed pooling in the pyriform fossa, consistent with vagal nerve dysfunction and impaired airway protection. Findings align with earlier reports of herpes zoster affecting multiple cranial nerves and emphasize its potential to disrupt phonatory, resonatory, and swallowing subsystems. This case underscores the importance of early, multidimensional assessment to document the functional consequences of HZ and to guide speech-language pathology interventions. Recognition of HZ as an etiology for cranial neuropathy is crucial for timely rehabilitation and improved quality of life.

Keywords: Herpes zoster, cranial neuropathy, vocal fold paralysis, dysphonia, dysphagia, speech-language pathology

INTRODUCTION

Herpes zoster (HZ), commonly known as shingles, is an acute neurocutaneous disorder caused by reactivation of the varicella-zoster virus (VZV), the same virus that produces chickenpox. Following the primary infection, VZV establishes latency in the dorsal root, autonomic, or cranial nerve ganglia and may remain dormant for decades. When immunity declines, particularly in the context of immunosenescence or immunosuppressive states, the virus may reactivate, resulting in unilateral, painful, vesicular eruptions along the distribution of the affected dermatome [1, 2]. Pain often precedes the rash, and the condition is well known for its neuropathic sequelae.

Epidemiological studies indicate that herpes zoster is a common condition with substantial global burden. The incidence of herpes zoster is influenced by age, occurring at a rate of 1.2–3.4

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cases per 1,000 persons annually in younger adults, and increasing to 3.9–11.8 cases per 1,000 persons annually in individuals over 65 years of age [3, 4]. In the United States, about one in three individuals will develop the disorder in their lifetime, with incidence rates ranging from 3–5 per 1,000 person-years and rising to 8–12 per 1,000 among individuals over 60 years [2]. Data from Asia reveal similar patterns, with hospital-based reports in India suggesting an incidence of around 0.7 per 1,000 person-years, though regional variations exist [5]. Systematic reviews further highlight that increasing life expectancy and the growing prevalence of chronic illnesses are expected to amplify the disease burden in coming decades [6].

The reactivation of VZV has been linked to several predisposing factors. Age-related immune decline is the most important risk factor, but immunosuppressive conditions such as HIV/AIDS, malignancies, and organ transplantation also play a significant role. Chronic systemic diseases like diabetes and renal failure, along with stress, malnutrition, trauma, or surgical insult to nerves, have additionally been reported as contributory triggers [1, 7]. Clinically, herpes zoster manifests in several forms depending on the site of viral reactivation. Herpes zoster ophthalmicus involves the ophthalmic branch of the trigeminal nerve and can lead to vision loss [8]. Ramsay Hunt syndrome, or herpes zoster oticus, is characterized by facial paralysis and hearing impairment due to involvement of the facial and vestibulocochlear nerves [9]. Other variants include zoster sine herpete, where the virus reactivates without a visible rash, disseminated zoster in immunocompromised individuals, and multidermatomal or zoster duplex affecting multiple or non-contiguous dermatomes [10].

Symptoms typically include prodromal pain described as burning, stabbing, or throbbing, followed by the appearance of erythematous vesicular rashes. Systemic complaints such as malaise and fever may occur, but one of the most distressing complications is postherpetic neuralgia (PHN), where neuropathic pain persists for months after lesion healing, disproportionately affecting the elderly population [11].

Although herpes zoster is primarily described as a dermatoneurological condition, cranial nerve involvement can have important implications for speech and swallowing. When cranial nerves VII, IX, and X are affected, patients may present with dysarthria, hypernasality, hoarseness, or vocal fatigue, which can significantly impair speech intelligibility. Dysphagia is also common, characterized by poor lip seal, nasal regurgitation, odynophagia, and aspiration risk, posing both nutritional and respiratory challenges [9]. Language functions are usually preserved; however, in rare cases of central nervous system involvement, such as vasculopathy or encephalitis, broader linguistic and cognitive impairments may arise [12].

Several reports highlight the impact of HZ on voice, resonance, and swallowing. Reference [13] described pharyngeal dysphagia due to VZV meningoradiculitis with complete recovery following treatment. Reference [14] reported pharyngolaryngeal involvement in HZ causing hoarseness, hypernasality, and aspiration risk. Reference [15] documented unilateral vocal fold paralysis associated with Ramsay Hunt syndrome, presenting with severe dysphonia. Reference [16] described dysphagia with unilateral vocal fold paralysis due to herpes zoster, confirming impaired airway protection. Cases of zoster sine herpete have also been reported with significant weakness of the left side of the soft palate causing hypernasality and nasal regurgitation, paralysis of the left vocal cord, and left facial nerve palsy [17]. Dysarthria and prosodic disturbances have been observed when multiple lower cranial nerves are affected, producing mixed bulbar deficits [18, 19]. Collectively, these studies emphasize that HZ can compromise multiple speech subsystems, but systematic and comprehensive documentation remains limited.

Despite evidence from such reports, the literature remains sparse, fragmented, and often focused on neurological or otolaryngological findings rather than multidimensional profiles of communication and swallowing. Very few studies employ standardized perceptual, acoustic, and quality-of-life measures alongside instrumental swallowing evaluations to provide a holistic understanding of the impact of herpes zoster [20].

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The present case addresses this gap by profiling the speech and swallowing characteristics of a patient with herpes zoster infection, using structured perceptual, acoustic, endoscopic, and patient-reported outcome measures. Given the functional consequences of these deficits on nutrition, communication, and quality of life, there is a strong need for detailed documentation of such manifestations. This case study aims to strengthen clinical recognition of HZ as an etiology for cranial neuropathy and to highlight the critical role of speech-language pathology in assessment and rehabilitation.

Aim

To comprehensively evaluate and document the speech and swallowing characteristics in a patient with herpes zoster infection presenting with cranial nerve involvement.

Objectives

- To assess perceptual, acoustic, and quality-of-life dimensions of voice using standardized tools (CAPE-V, GRBAS, VHI, and acoustic analysis with Dr. Speech).
- To examine swallowing function through patient-reported measures (SWAL-QOL, DHI) and Fiberoptic Endoscopic Evaluation of Swallowing (FEES).

METHODS

Case History

A 54-year-old male presented to the Department of Speech-Language Pathology with primary complaints of reduced vocal loudness, dysphagia, and decreased hearing sensitivity. The medical history revealed that he was undergoing pharmacological management for herpes zoster. A comprehensive evaluation was undertaken, including detailed speech and swallowing assessments and an Oral Peripheral Mechanism Examination (OPME).

Ethical Considerations

All ethical guidelines outlined by [20] were followed. Written informed consent was obtained from the patient prior to evaluation, data documentation, and use of clinical findings for academic reporting. Confidentiality and privacy were strictly maintained throughout the assessment and reporting process.

Assessment Protocol

A structured evaluation protocol was followed, incorporating the following tools and procedures:

- *Oral Peripheral Mechanism Examination (OPME):* To evaluate structural and functional integrity of the speech articulators.
- *Perceptual Voice Assessment:* Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) was used to rate overall severity, roughness, breathiness, strain, pitch, and loudness. The GRBAS scale (Grade, Roughness, Breathiness, Asthenia, Strain) was administered using a four-point rating system.
- *Voice Handicap Index (VHI):* To measure the psychosocial impact of the voice disorder on the patient's daily life.
- *Objective Acoustic Analysis:* Dr. Speech Software Version 4 (Tiger DRS, Inc., Seattle) was used to obtain acoustic parameters including fundamental frequency (F_0), vocal intensity (dB), jitter (%), shimmer (%), harmonic-to-noise ratio (HNR), and normalized noise energy (NNE).
- *Swallowing Assessments:* The Swallowing Quality of Life Questionnaire (SWAL-QOL) and the Dysphagia Handicap Index (DHI) were administered to determine functional and emotional impacts of dysphagia.
- *Rigid Laryngoscopy:* Conducted to visualize laryngeal structures, assess vocal fold mobility, and identify mucosal lesions.
- *Fiberoptic Endoscopic Evaluation of Swallowing (FEES):* Direct visualization of the swallowing process was carried out to assess safety and efficiency of swallowing.

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RESULTS

The OPME revealed deviation of the lips towards the left side, with no associated respiratory difficulties. Maximum phonation durations were markedly reduced: 3 seconds for /a/, 2 seconds for /i/, and 2 seconds for /u/.

Perceptual voice assessment revealed severe breathiness and reduced loudness. GRBAS ratings were: Grade (G) – 3, Roughness (R) – 1, Breathiness (B) – 3, Asthenia (A) – 3, and Strain (S) – 3. CAPE-V scores indicated overall severity of 75/100, roughness 35/100, breathiness 75/100, strain 75/100, pitch 50/100, and loudness 75/100.

Objective acoustic analysis with Dr. Speech Software showed: fundamental frequency (F_0) – 135.58 Hz, jitter – 1.54%, shimmer – 8.64%, harmonic-to-noise ratio (HNR) – 14.12, and normalized noise energy (NNE) – −1.37.

The Voice Handicap Index (VHI) yielded a total score of 31, indicating a moderate perceived voice handicap with notable impact on daily communication. Swallowing assessments revealed a SWAL-QOL score of 109, reflecting severe reduction in swallowing-related quality of life, particularly in domains of eating duration, swallowing-related fear, and social participation. The Dysphagia Handicap Index (DHI) score was 49, indicating a moderate swallowing handicap.

A rigid laryngoscopic examination demonstrated left vocal fold paralysis, pooling of saliva within the pyriform fossa, and an ulcerative lesion on the posterior pharyngeal wall. Additionally, herpes zoster lesions were observed over the left arytenoid region, as reported from an ENT specialist (Figure 1).



Figure 1. Rigid laryngoscopic view demonstrating left vocal fold paralysis.

Fiberoptic Endoscopic Evaluation of Swallowing (FEES) findings included successful completion of the cervical auscultation test, along with pooling of saliva in the pyriform fossa, suggesting impaired pharyngeal clearance (Figure 2).

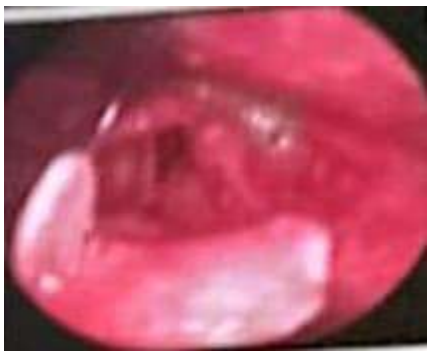


Figure 2. Fiberoptic Endoscopic Evaluation of Swallowing (FEES) showing pooling of saliva in the left pyriform fossa.

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DISCUSSION

The present case illustrates herpes zoster–related cranial neuropathy manifesting as unilateral vocal fold paralysis, dysphonia, and dysphagia, confirmed through perceptual, acoustic, endoscopic, and patient-reported outcome measures. These findings highlight the multisystemic impact of varicella–zoster virus (VZV) reactivation beyond dermatological involvement, with significant implications for speech, voice, and swallowing functions.

The oral peripheral mechanism examination revealed deviation of the lips and markedly reduced maximum phonation durations (MPTs). In healthy adult males, normative MPT values range from 17–26 seconds [21]. The patient's values of 2–3 seconds reflect profound glottic insufficiency due to incomplete closure of the paralyzed vocal fold. Similar reductions in MPT have been reported in patients with unilateral vocal fold paralysis (UVFP), where decreased respiratory–phonatory efficiency is a hallmark [22, 23].

Perceptual assessments (GRBAS and CAPE-V) revealed severe dysphonia characterized by breathiness, strain, asthenia, and reduced loudness. These features are physiologically consistent with glottic insufficiency and compensatory supraglottic activity. The Consensus Auditory-Perceptual Evaluation of Voice (CAPE-V) and GRBAS scale are validated tools for voice assessment [24], and their application in this case confirmed severe voice dysfunction. Earlier studies on UVFP also report similar perceptual voice patterns, with hoarseness, breathiness, and strained phonation being common [25, 26].

Objective acoustic analysis revealed elevated jitter (1.54%) and shimmer (8.64%), and a reduced harmonic-to-noise ratio (14.12), reflecting irregular and asymmetric vocal fold vibration. These findings align with previous reports of acoustic disturbances in UVFP, where increased perturbation measures and reduced harmonic energy are typical [26, 27].

The Voice Handicap Index (VHI) score of 31 indicated a moderate self-perceived handicap. The VHI is widely validated for capturing the psychosocial consequences of dysphonia. Patients with UVFP often report reduced communication confidence and social withdrawal despite retaining some functional vocal ability, consistent with this case [25].

Swallowing assessments demonstrated severe impairment in swallowing-related quality of life (SWAL-QOL = 109) and moderate dysphagia burden (DHI = 49). These results suggest substantial limitations in nutrition and social participation. Both SWAL-QOL and DHI are validated instruments for evaluating the impact of dysphagia [28, 29]. Prior studies of VZV-induced cranial neuropathy have reported similar dysphagia profiles, with pharyngeal residue, aspiration risk, and reduced quality of life [14].

The rigid laryngoscopic examination revealed left vocal fold paralysis, saliva pooling in the pyriform fossa, and an ulcerative lesion on the posterior pharyngeal wall, with herpes zoster lesions over the arytenoid. These findings suggest vagal nerve involvement with concurrent mucosal pathology. Vocal fold immobility likely reflects recurrent laryngeal nerve neuritis, accounting for the patient's dysphonia, while pyriform pooling indicates impaired pharyngeal clearance contributing to dysphagia [22, 30]. Fiberoptic Endoscopic Evaluation of Swallowing (FEES) further confirmed pyriform pooling, reinforcing evidence of vagal dysfunction. Comparable laryngoscopic and FEES findings have been documented in pharyngolaryngeal zoster and Ramsay Hunt syndrome [13, 14, 31], underscoring herpes zoster's potential to cause combined neural and mucosal compromise.

The coexistence of dysphonia, dysphagia, and auditory complaints suggests polycranial neuropathy secondary to herpes zoster. While facial and vestibulocochlear nerve involvement are common in Ramsay Hunt syndrome [9], vagal neuropathy resulting in vocal fold paralysis is less frequent but well-documented [16]. Early reports of multiple cranial nerve involvement in herpes zoster also support the current presentation [32].

CONCLUSIONS

Herpes zoster can cause significant speech and swallowing impairments through vagal nerve involvement and mucosal pathology. Comprehensive assessment combining perceptual, acoustic, endoscopic, and quality-of-life measures is essential. Early recognition and multidisciplinary management, including speech-language pathology intervention, are crucial for improving communication, swallowing safety, and overall quality of life.

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Conflict of Interest

The author(s) declared no conflict of interest.

Data Availability Statement

Data supporting these findings is available within the article or upon request.

Institutional Review Board Statement

All ethical guidelines outlined by [20] were followed. Written informed consent has been obtained from the patient to publish this paper.

Supplementary Materials

Nil

REFERENCES

1. Arvin AM. Varicella–zoster virus. Clin Microbiol Rev. 1996 Jul;9(3):361–81.
2. Centers for Disease Control and Prevention. Shingles (herpes zoster). Atlanta (GA): CDC; 2025. Available from: https://www.cdc.gov/shingles/about/index.html.
3. Nair PA, Patel BC. Herpes zoster. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025. Available from: http://www.ncbi.nlm.nih.gov/books/NBK441824/.
4. Sinha R, Kumari P, Pallavi UK, Sarkar S. Clinical and epidemiological profile of herpes zoster and its complications in a tertiary care center of Bihar: a prospective study. Cureus. 2023 Aug;15(8):e43560.
5. Gummadi P, Srirama S, Lakshmisarojini V. A clinico-epidemiological study of herpes zoster in a tertiary care hospital in South India. Int J Acad Med Pharm. 2024;6(3):366–9.
6. Kawai K, Gebremeskel BG, Acosta CJ. Systematic review of incidence and complications of herpes zoster: towards a global perspective. BMJ Open. 2014 Jun;4(6):e004833.
7. Gnann JW Jr, Whitley RJ. Herpes zoster. N Engl J Med. 2002 Aug 1;347(5):340–6.
8. Pavan-Langston D. Herpes zoster ophthalmicus. Neurology. 1995 Dec;45(12 Suppl 8):S50–1.
9. Sweeney CJ, Gilden DH. Ramsay Hunt syndrome. J Neurol Neurosurg Psychiatry. 2001 Aug;71(2):149–54.
10. Dworkin RH, Johnson RW, Breuer J, Gnann JW Jr, Levin MJ, Backonja M, et al. Recommendations for the management of herpes zoster. Clin Infect Dis. 2007 Jan 1;44(Suppl 1):S1–26.
11. Johnson RW, Rice ASC. Postherpetic neuralgia. N Engl J Med. 2014 Oct 16;371(16):1526–33.
12. Gilden DH, Kleinschmidt-DeMasters BK, LaGuardia JJ, Mahalingam R, Cohrs RJ. Neurologic complications of the reactivation of varicella–zoster virus. N Engl J Med. 2000 Mar 2;342(9):635–45.
13. Muhle P, Suntrup-Krueger S, Dziewas R, Warnecke T. Pharyngeal dysphagia due to varicella-zoster virus meningoradiculitis and full recovery: case report and endoscopic findings. SAGE Open Med Case Rep. 2018 Jan;6:2050313X18756560.

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14. Nisa L, Landis BN, Giger R, Leuchter I. Pharyngolaryngeal involvement by varicella–zoster virus. J Voice. 2013 Sep;27(5):636–41.
15. Rasmussen ER, Mey K. Vocal cord paralysis associated with Ramsay Hunt syndrome: looking back 50 years. BMJ Case Rep. 2014 Feb 6;2014:bcr2013201038.
16. Jung JW, Jang YJ, Hong EH, Kim KH, Kim KJ, Park EJ. Dysphagia with unilateral vocal cord paralysis in herpes zoster: a case report. Ann Dermatol. 2022;34(6):475–7.
17. Fujiwara K, Furuta Y, Fukuda S. Associated laryngeal paralysis caused by varicella–zoster virus without eruption. Case Rep Med. 2014;2014:1–3.
18. Choi JH. Two cases of pharyngolaryngeal zoster advanced to multiple cranial neuropathy. Am J Otolaryngol. 2013 Jul;34(4):369–72.
19. Yokote A, Yamashita KI, Sakoda A, Iwanaga Y, Nakamura Y, Haro K, et al. Zoster sine herpete affecting multiple lower cranial nerves presenting severe dysphagia in a patient with Parkinson's disease. Intern Med. 2025 Apr 15;64(8):1245–8.
20. Basavaraj V, Venkatesan S. Ethical guidelines for biobehavioral research involving human subjects. Mysuru: All India Institute of Speech and Hearing; 2009.
21. Maslan J, Leng X, Rees C, Blalock D, Butler SG. Maximum phonation time in healthy older adults. J Voice. 2011 Nov;25(6):709–13.
22. Ryu CH, Kwon TK, Kim H, Kim HS, Park IS, Woo JH, et al. Guidelines for the management of unilateral vocal fold paralysis from the Korean Society of Laryngology, Phoniatrics and Logopedics. Clin Exp Otorhinolaryngol. 2020 Nov;13(4):340–60.
23. Walton C, Conway E, Blackshaw H, Carding P. Unilateral vocal fold paralysis: a systematic review of speech-language pathology management. J Voice. 2017 Jul;31(4):509.e7–509.e22.
24. Kempster GB, Gerratt BR, Verdolini Abbott K, Barkmeier-Kraemer J, Hillman RE. Consensus auditory-perceptual evaluation of voice: development of a standardized clinical protocol. Am J Speech Lang Pathol. 2009 May;18(2):124–32.
25. Chang WD, Chen SH, Tsai MH, Tsou YA. Autologous fat injection laryngoplasty for unilateral vocal fold paralysis. J Clin Med. 2021 Oct 28;10(21):5034.
26. Hartl DM, Hans S, Vaissière J, Riquet M, Brasnu DF. Objective voice quality analysis before and after onset of unilateral vocal fold paralysis. J Voice. 2001 Sep;15(3):351–61.
27. Uloza V, Padervinskis E, Vegiene A, Pribuisiene R, Saferis V, Vaiciukynas E, et al. Exploring the feasibility of smartphone microphone for measurement of acoustic voice parameters and voice pathology screening. Eur Arch Otorhinolaryngol. 2015 Nov;272(11):3391–9.
28. McHorney CA, Robbins J, Lomax K, Rosenbek JC, Chignell K, Kramer AE, et al. The SWAL-QOL and SWAL-CARE outcomes tool for oropharyngeal dysphagia in adults: III. Documentation of reliability and validity. Dysphagia. 2002 Apr;17(2):97–114.
29. Silbergleit AK, Schultz L, Jacobson BH, Beardsley T, Johnson AF. The dysphagia handicap index: development and validation. Dysphagia. 2012 Mar;27(1):46–52.
30. Langmore SE, Schatz K, Olsen N. Fiberoptic endoscopic examination of swallowing safety: a new procedure. Dysphagia. 1988 Dec;2(4):216–9.
31. Shim JH, Park JW, Kwon BS, Ryu KH, Lee HJ, Lim WH, et al. Dysphagia in Ramsay Hunt syndrome: a case report. Ann Rehabil Med. 2011;35(5):738–41.
32. Murakami S, Honda N, Mizobuchi M, Nakashiro Y, Hato N, Gyo K. Rapid diagnosis of varicella-zoster virus infection in acute facial palsy. Neurology. 1998 Oct;51(4):1202–5.

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