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A RARE CASE OF VOCAL CORD PALSY DUE TO VARICELLA ZOSTER VIRUS REACTIVATION

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ABSTRACT
We report a rare case of vocal cord paralysis in a 55-year-old female with aplastic anemia, attributed to delayed sequelae of Varicella Zoster Virus (VZV) reactivation.[1] The patient presented with hoarseness of voice and difficulty speaking, and was diagnosed with left vocal cord paralysis. Despite initial treatment with steroids, the patient showed significant improvement only after initiating voice and speech therapy.[2] This case highlights the importance of considering immune-mediated nerve damage in the differential diagnosis of vocal cord paralysis, particularly in immunocompromised patients with a history of VZV infection.[3]

KEYWORDS: Varicella Zoster Virus, Vocal Cord Paralysis, Aplastic Anemia, Immune-Mediated Neuropathy.

CASE REPORT
A 55-year-old female presented with a three-day history of hoarseness of voice and difficulty in speaking. History of shingles (herpes zoster) six months prior.[4] Diagnosed case of aplastic anemia. No history of diabetes mellitus. On examination, no evidence of active VZV infection or skin lesions. No neck swelling, lymphadenopathy, or tenderness. No facial or limb weakness. Neurological examination was unremarkable except for vocal cord involvement.

Differential diagnosis thought of are
1. Inflammatory/Autoimmune: Sarcoidosis, vasculitis[5]
2. Infectious: Viral nerve damage (e.g., shingles, Epstein-Barr Virus, Lyme disease)[6]
3. Neoplastic: Tumors in the lung, thyroid, esophagus, or mediastinum compressing the recurrent laryngeal nerve[7]
4. Vascular: Thoracic aortic aneurysm causing nerve compression[8]

Diagnosis tests
Laryngoscopy: Revealed left vocal cord paralysis.[9]
Laboratory Tests: Normal full blood count and routine biochemistry. Positive VZV IgG, normal IgM, indicating past infection.[10]
Imaging Studies: CT Neck/Chest: No structural abnormalities. MRI Brain/Neck: No central nervous system pathology.[11]
Nerve Cconuction Studies: Normal nerve conduction velocity (NCV) of bilateral upper and lower limbs. Electromyography indicated left vocal cord denervation, consistent with chronic nerve damage.[12]
A diagnosis of delayed vocal cord paralysis due to immune-mediated nerve damage following VZV reactivation was made after ruling out other causes.[13]

Treatment
Initial Therapy: Low-dose steroids were administered for 15 days but yielded no significant response.[14]
Supportive Measures: Voice and speech therapy were initiated, leading to marked improvement in vocal function within one month[15] Prognosis: Recovery from VZV-induced vocal cord palsy is unpredictable. Some patients achieve spontaneous recovery with supportive care, while others may have permanent deficits.[1]

DISCUSSION
Varicella Zoster Virus (VZV): VZV is the causative agent of chickenpox and shingles, which can affect peripheral nerves and cranial ganglia.[2] Reactivation often occurs in immunocompromised individuals, as in this patient with aplastic anemia.[3] Mechanism of

Paralysis: Reactivated VZV likely affected the recurrent laryngeal nerve, causing delayed onset of vocal cord paralysis through an immune-mediated process.[4] Delayed neurological complications from VZV, such as cranial neuropathies, should be considered in patients with a history of shingles and immunocompromising conditions.[5]

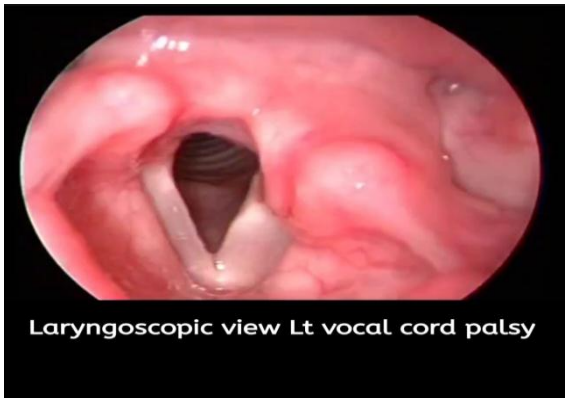

Figure 1

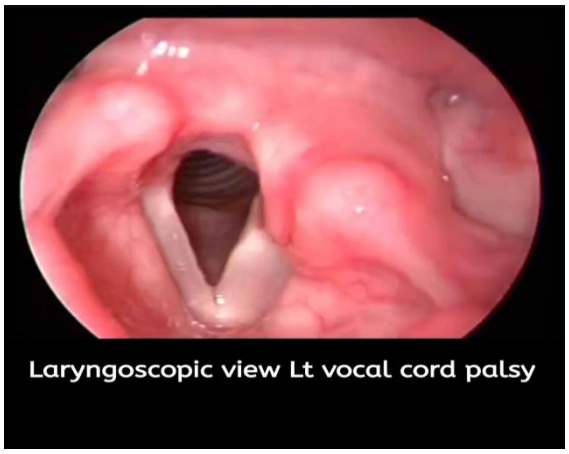

Figure 2

CONCLUSION

VZV vaccination should be considered for immunocompromised patients to prevent reactivation.[6] Monitoring: Regular follow-up for post-shingles patients, especially those with weakened immunity, to identify early neurological complications.[7] More studies are needed to elucidate the pathophysiology of VZV-induced cranial neuropathies and establish optimal management protocols.[8]

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