

Post Polio Syndrome with Sensorineural Hearing Loss: A Case Report

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Abstract:

An enterovirus causes the neurological disorder known as poliomyelitis. Many survivors have Post-polio syndrome, a poorly understood condition. It has been estimated that 20% to 85% of those who have experienced poliomyelitis will develop PPS. The following factors are necessary for a PPS diagnosis: 1) motor neuron loss from a previous episode of poliomyelitis; 2) neurologic and functional stability for at least 15 years following the initial onset of polio; and 3) a gradual start of new weakness and atypical muscular fatigability that has lasted for at least a year. We present the case of an elderly patient who complained of headache and giddiness, as well as a history of poliomyelitis and other coexisting conditions.

Keywords: Poliomyelitis, motor neuron disease, Lifestyle Modification, post polio syndrome

INTRODUCTION:

A polio survivor may develop post-polio syndrome (PPS) years after the initial polio infection(1). Even though motor neurons are the primary target of polio, some research points to a potential link between the poliovirus and auditory nerve involvement, especially when the brainstem is affected. In these situations, sensorineural hearing loss could be seen. Sensorineural hearing loss, a type of hearing impairment, can be caused by damage to the auditory nerve circuits that connect the brain and the inner ear. The objective of this page is to increase awareness of Post-Polio Syndrome (PPS), a suspected poliovirus sequela that is becoming more common as people live longer lives after catching the virus(2). It takes muscular strength, joint mobility, and central nervous system coordination to perform a physiological walking exercise. PPS compromises the typical motor pattern by causing a significant impairment in one or more of these skills. PPS is characterized by significant motor system

weakness and exceptional difficulty with physiological walking activities(3).

CASE PRESENTATION:

A 63-year-old male participant with a previous medical history of primary hypertension and on medication T. Cilnidipine 10MG 1-0-1 and a polio which is then cured, patient was brought to casualty by wheel chair along with attender came with the complaints of giddiness since 1 month acute in onset, progressive in nature, not associated with postural variation, also complaints of headache since 1 month, holocranial, burning type mild-moderate in intensity. On 17/7/25 Patient complaints of throat pain associated with difficulty and pain during swallowing more to solids than liquids, blood in sputum (minimal) since yesterday (3 episodes- past 1 month) and one episode of hemoptysis hence referred to ENT in which associated with ringing sensation in both the ears on and off for few seconds, also associated with nausea, no relieving factor. PTA has done on 16/7/25 and shows bilateral moderate sensorineural

hearing loss right- 45dB left- 41.5dB, VLS done with 70° scope shows congestion: over arytenoids, false vocal cords, minimal edema present, bilateral vocal cords mobile. Eye examination showed both eye senile immature cataract NS 1 with refractory error and presbyopia. Based on the investigations the patient was diagnosed with post polio syndrome, acute gastritis and systemic hypertension Laboratory investigations were done in which haemoglobin, RBC, triglycerides, total cholesterol, HDL, VDL, and VLDL. The patient was treated with Inj. Eldervit 1 amp in 100ML NS IV 1-0-0, Inj. Emeset 4MG IV 1-0-1, T. Cilnidipine 10MG 1-0-1, T. Pan 40MG 1-0-0, T. Aspirin 75MG 0-0-1, T. Lipikind F 0-0-1, T. Vertin 16MG 1-0-1 and Syp. Sucrafil 10ML 1-1-1.

DISCUSSION:

Our study's primary goal was to characterize a patient sample's demographic, clinical, and neurophysiological features(4). Trojan et al.'s study on electro diagnostic studies—conventional EMG, SFEMG, and macro-EMG—provided details on the post-polio motor unit and the potential causes of certain PPS symptoms. Some people who have had polio may have neuromuscular junction transmission abnormalities, which can cause muscle weariness and, indirectly, overall fatigue. Post-polio syndrome's precise pathophysiology is unknown, however a number of possibilities have been put up(5). Collateral motor neurons are thought to arise as a result of excessive motor neuron destruction during an acute infection. The capillarization and innervations of these compensatory motor neuron sprouts cannot be sustained over time. Muscle weakness, discomfort, and easy fatigability are the outcomes. The persistence of polio infection is another explanation. Several investigations have discovered poliovirus genome fragments in the cerebrospinal fluid (CSF) of post-polio syndrome patients(6).

CONCLUSION:

An intriguing condition known as post-polio syndrome is defined by fresh neuromuscular abnormalities that appear years after a polio infection has cleared up. Among the many diagnostic criteria, ruling out alternative diagnosis is one of the most crucial. Multiple concomitant illnesses make diagnosing post-polio syndrome in the older population even more challenging. In this population, a proper diagnosis may require extensive testing. The public should, however, pay attention to both the acute condition and the possibility of long-term consequences. This example should therefore be used as a signal for further research on the expanding number of PPS patients as well as the need for vaccines as both immediate and long-term protection.

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