Postinfectious Cranial Neuropathy in a Pediatric Patient:

A Case of Acute Palatal Palsy

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Sudden-onset nasal regurgitation and changes in speech are unusual symptoms in the pediatric population. These symptoms often suggest neurological or infectious etiology, such as cranial nerve dysfunction responsible for palatal movement and speech articulation. Postinfectious cranial nerve involvement, particularly following upper respiratory tract infection, is an important differential diagnosis in such cases.[1–4] Here, we present the case of a 14-year-old boy who developed acute nasal regurgitation and dysphonia following a febrile illness and discuss the diagnostic approach, therapeutic response, and underlying immune-mediated mechanisms.

Case Report

A 14-year-old previously healthy boy presented with a 2-day history of painless, progressive nasal regurgitation of liquids and nasal quality to his speech. The symptoms followed a febrile illness with sore throat and cough 2 weeks earlier. The febrile illness resolved spontaneously within 5 days without specific treatment. No throat culture or viral studies were performed.

The patient denied anosmia, visual disturbances, difficulty in eye movement, facial weakness, taste disturbances, limb weakness, incoordination, or bowel/bladder dysfunction. There was no history of facial trauma or envenomation. His immunizations were up to date, and he had no significant medical history. The only family history was essential tremors in his mother.

On physical examination, the patient exhibited bilateral absence of palatal movement and a diminished gag reflex, with a normal uvula and tonsils. General and systemic examinations were otherwise unremarkable. Routine laboratory investigations revealed the following values: complete blood count: hemoglobin of 13.8 g/dL, white blood cell count of 6,800/µL, and platelets of 250,000/µL; metabolic panel: sodium of 138 mmol/L, potassium of 4.1 mmol/L, urea of 28 mg/dL, creatinine of 0.8 mg/dL, and blood glucose of 98 mg/dL; cerebrospinal fluid (CSF) analysis: white blood cell count of 0/µL, protein of 28 mg/dL, and glucose of 68 mg/dL (serum glucose: 98 mg/dL); CSF viral polymerase chain reaction for herpes simplex virus, varicella-zoster virus, enteroviruses, and Epstein-Barr virus was negative; inflammatory markers, including erythrocyte sedimentation rate (14 mm/h) and C-reactive protein (<1 mg/L), were normal.

Repetitive nerve stimulation testing showed no decremental response, excluding neuromuscular disorders like myasthenia gravis. Diagnostic nasal endoscopy and video laryngeal stroboscopy were normal. Magnetic resonance imaging of the brain showed no central nervous system pathology.

The patient was initiated on intravenous dexamethasone 4 mg twice daily for 5 days. Dexamethasone was chosen for its potent anti-inflammatory properties and favorable side effect profile. No tapering was needed, and no adverse effects were observed. Speech quality improved within 48 hours, and nasal regurgitation resolved completely by day 6.

Follow-up for a month revealed no residual symptoms or recurrence. Neurological examination at the last follow-up was within normal limits.

Discussion

Isolated palatal palsy is an uncommon clinical presentation, typically manifesting as nasal speech, nasal regurgitation, and dysphagia.[5] Various etiologies include viral infections (eg, herpes simplex virus, Coxsackievirus, measles, and Epstein-Barr virus), craniofacial trauma, autoimmune conditions (eg, Guillain-Barré syndrome and multiple sclerosis), neuromuscular disorders, and malignancies (eg, lymphoma and leukemia).[3,6]

In this case, the acute onset following a febrile illness strongly suggested a postinfectious inflammatory process affecting the vagus nerve (cranial nerve X), which innervates the palatal and laryngeal muscles.[7] Rapid symptom resolution with corticosteroid therapy supports an immune-mediated mechanism. Postviral cranial neuropathies, particularly following respiratory infections, have been documented. The proposed mechanism involves immune-mediated inflammation or damage to cranial nerve nuclei or peripheral nerves, causing transient dysfunction.[8,9]

Prognostic factors for favorable outcomes include the absence of systemic or structural pathology, early anti-inflammatory treatment, and a self-limiting course. For primary care

physicians, early recognition and referral for neurological evaluation, coupled with imaging and laboratory investigations, are essential to rule out serious conditions.

This case emphasizes the importance of considering postviral immune-mediated neuropathies in isolated cranial nerve dysfunction. Early recognition and corticosteroid therapy can result in complete resolution, as demonstrated in this case.

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