Facial Dysesthesias with Tube Feeding Intolerance After Cerebellar Stroke: A Case Report

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Abstract

Case description: 55-year-old male with atrial fibrillation presented with dysarthria and balance impairment. Imaging demonstrated acute left posterior inferior cerebellar artery infarction and left cerebellar stroke. He required percutaneous endoscopic gastrostomy (PEG) tube. On day 20, he was admitted to inpatient rehabilitation where he was found to have right facial numbness and weakness, bilateral horizontal gaze nystagmus, and ataxia. He had frequent large volume gastric residuals, with heartburn, increased salivation, and right temporal-maxillary dysesthesias. Scopolamine patch was initiated, and the patient reported symptom relief and improved tube feeding tolerance.

Discussions: Dysphagia is a sequela of stroke, however, facial dysesthesia after cerebellar stroke is not previously reported. Gastric distention from enteral tube feeds produces a parasympathetic response to initiate salivation and digestion. A lesion to the trigeminal tract can alter physiologic salivation to be perceived as painful facial discomfort. Generally, anticholinergic medications are minimized following stroke as they impair neurologic recovery. Furthermore, they slow gastric emptying and reduce lower esophageal sphincter tone, worsening nausea or gastric reflux. In this case, the use of a transdermal anticholinergic medication provided adequate relief of symptoms to permit PEG tube use and ensure adequate nutrition, while reducing deleterious cognitive effects.

Conclusions: In this patient, whose salivation and facial dysesthesias were associated with PEG utilization, trigeminal tract dysfunction is an important clinical consideration. Scopolamine was utilized to reduce salivary output, reduce parasympathetic tone, and alleviate discomfort. Tube feeding intolerance warrants prompt investigation and treatment in order to maintain adequate nutrition, which is essential to neurologic recovery.

Teaching Points

- When compared to MRI, usual CT imaging has reduced sensitivity for detecting acute or subacute ischemia in patients with posterior cranial fossa lesions.
- It is important to have a high index of suspicion for additional impairments that could reveal other sites of neurologic involvement in patients with a cardioembolic history.
- Tube feeding intolerance warrants prompt investigation and treatment in order to maintain adequate nutrition, which is essential to neurologic recovery.

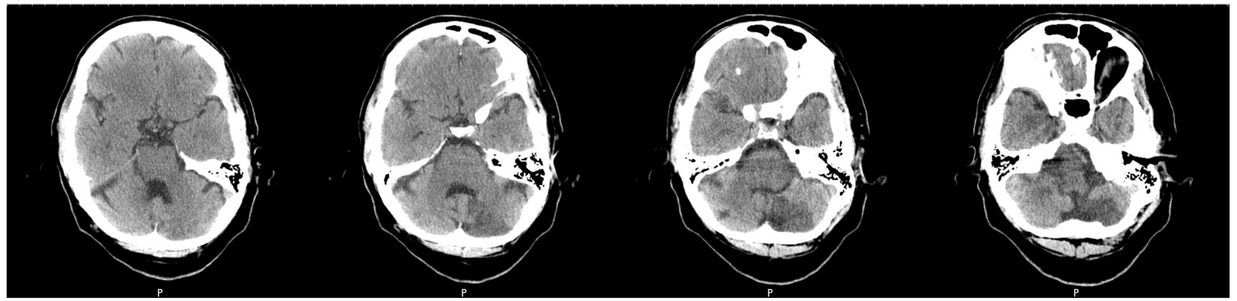

Fig 1. CT series showing hypoattenuation in the left posterior inferior cerebellar artery territory with chronic right inferior cerebellar infarcts

Case Description

A 55-year-old male with past medical history of hypertension presented with new onset dysarthria and balance impairment. Initial work up identified occult atrial fibrillation, with acute left posterior inferior cerebellar artery (PICA) infarction. He was treated with intravenous thrombolysis. Three days later, he had new anisocoria and brain computerized tomography (CT) revealed new left cerebellar stroke. He was started on apixaban for anti-coagulation, and statin therapy for secondary stroke prophylaxis. His acute care course was further complicated by Mallory-Weiss tear and upper gastrointestinal bleeding, managed non-operatively, and he required PEG tube placement for dysphagia. On post-stroke day 20, he was admitted to inpatient rehabilitation.

Upon admission to rehabilitation, physical examination revealed right facial numbness and weakness, bilateral horizontal gaze nystagmus, dysarthria, hypophonia, and ataxia in all limbs. Subsequent CT imaging studies demonstrated evolving bilateral cerebellar encephalomalacia without hemorrhagic transformation or brainstem involvement. With tube feedings, free water flushes, or medication administration, nursing staff reported frequent large volume gastric residuals (over 200mL), and he endorsed heartburn, increased salivation, and facial dysesthesias to a nonspecific, right temporal-maxillary region. Adjustments to bolus volumes and frequency, trial of continuous slow rate night time tube feeding, and trials of other tube feed formulations were not helpful. Treatment with acetaminophen prior to feedings, and potential comorbid gastroesophageal reflux with lansoprazole and calcium carbonate, was also not helpful. A bowel regimen consisting of stimulant laxatives and intermittent suppository use was implemented producing regular bowel movements, but without resolution of symptoms. Given the temporal association of symptoms with PEG tube use, a neurologic mechanism involving the gastrointestinal phase of salivation was hypothesized, and anticholinergic therapy pursued to reduce oral secretions. Off-label use of scopolamine patch was initiated, and within 24 hours, the patient reported a decrease in symptoms and improved tolerance of tube feedings, and medication administration.

Discussion

Following stroke or traumatic brain injury, optimized nutrition is necessary for neurocognitive recovery, particularly in the acute and subacute phases. Malnutrition is found in up to 62% of stroke patients, and any adverse effects from enteral feeding that disrupt adequate nutrition can be detrimental to recovery [1,2,3]. Patients with inadequate nutrition have associated increased complications such as pneumonia, urinary infections, pressure ulcers, falls and increased length of stay in the hospital[1,2,3]. Improvement in nutritional status is associated with greater gain in Functional Independence Measure (FIM) scores during inpatient rehabilitation[4].

Relevant Neuroanatomy

Cranial nerve V, the trigeminal nerve, is responsible for facial sensation and is implicated in trigeminal neuralgia and other painful facial neurologic disorders. It originates in the pons, medial to the middle cerebellar peduncle, and its sensory nucleus extends throughout the brain stem. In particular, the auriculotemporal nerve, a branch of the mandibular division of the trigeminal nerve, carries afferent nerve fibers from the parotid glands as well as post-ganglionic parasympathetic nerve fibers from the otic ganglion.

Cranial nerve X, the vagus nerve, regulates digestive tone in the stomach. As the stomach distends in volume, mechanoreceptors and chemoreceptors stimulate the vagus nerve. Afferent signals travel to the nucleus solitarius in the medulla oblongata, through the sensory thalamus, to the cerebral cortex and limbic system, triggering the parasympathetic response for digestion[8,9], including salivation.

Given this neurophysiology, it is plausible that gastric distention from enteral tube feeds produces a parasympathetic response to initiate salivation and digestion. A lesion to the trigeminal tract or nucleus, with disordered processing of afferent signaling, is a mechanism by which physiologic salivation, in response to gastric distention, can be perceived as painful facial discomfort.

Discussion cont.

Clinical Considerations

This patient's subsequent CT brain studies showed extensive bilateral cerebellar encephalomalacia, and given the cardioembolic etiology of stroke, there is potential for additional brainstem involvement in the pons or medulla, not well visualized on CT. Apart from facial dysesthesias, this patient presented with ataxia, nystagmus, dysphagia, dysarthria, and hypophonia. However, he did not have the classic sensory impairments to pain and temperature sensation on the face and body from injury to the spinothalamic tract, nor did he have ptosis, miosis, and anhidrosis (Horner's syndrome)[7], that are also characteristic of LMS. The spectrum of deficits correlates with his described cerebellar stroke history, but also the lateral medulla and pons, most resembling Wallenberg's syndrome. His presentation is confounded by the history of bilateral cerebellar involvement, lack of brainstem findings on subsequent CT studies, and lack of follow-up MRI studies to confirm ischemia in the brainstem.

Treatment Considerations

Generally, anticholinergic medications are minimized following stroke or traumatic brain injury, as they impair neurologic recovery and cognitive function. Furthermore, they slow gastric emptying and reduce lower esophageal sphincter tone, often with worsening nausea or gastric reflux[9]. For those with symptomatic gastroparesis or tube feeding intolerance, metoclopramide or other prokinetic agents are employed off-label, but their antidopaminergic effects also impair arousal, memory, and motor function. During inpatient rehabilitation, agents that facilitate cholinergic transmission (e.g., donepezil) and dopaminergic transmission (e.g., amantadine, methylphenidate) are commonly used off-label to enhance neurologic recovery. Utilizing anticholinergic and antidopaminergic medications, while giving cognitive enhancing medications, may negate their potential benefits and contribute to polypharmacy. In this case, the use of a transdermal anticholinergic medication provided adequate relief of symptoms to permit PEG tube use and ensure adequate nutrition, while reducing systemic absorption and deleterious cognitive effects.

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