Section: General Medicine

Case Report

RECURRENT BILATERAL STROKE PRESENTING WITH UNUSUAL PALATAL MYOCLONUS

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ABSTRACT

Background: Palatal myoclonus is a rare rhythmic movement disorder classified into essential and symptomatic subtypes. Symptomatic palatal myoclonus results from lesions disrupting the dentato-rubro-olivary pathway within the Guillain-Mollaret triangle, leading to hypertrophic degeneration of the inferior olivary nucleus. This condition is typically associated with stroke, demyelinating diseases, or posterior fossa lesions, though bilateral stroke presentations remain exceptionally rare in the literature.

Case Presentation: We report the case of a 68-year-old man with poorly controlled type 2 diabetes mellitus and hypertension who presented with a three-day history of gait ataxia, urinary incontinence, and behavioral changes with emotional lability. He had experienced progressive dysarthria and tinnitus over the preceding two months. Neurological examination revealed cognitive impairment (Mini-Mental State Examination score 20/30), right-sided spastic hemiparesis with hyperreflexia and extensor plantar response, cerebellar signs including nystagmus and truncal ataxia, and characteristic rhythmic palatal myoclonus on cranial nerve examination. Magnetic resonance imaging of the brain demonstrated an acute infarct in the left frontal parasagittal region and a chronic infarct involving the Guillain-Mollaret triangle. Comprehensive cardiovascular and hematological investigations, including carotid Doppler ultrasonography, echocardiography, 24-hour Holter monitoring, and thrombophilia screening, revealed no identifiable cardioembolic source or coagulopathy. The patient was managed with antiplatelet therapy (aspirin 150 mg), statin therapy (atorvastatin 10 mg), sodium valproate for symptomatic control of palatal myoclonus, and comprehensive rehabilitation including physiotherapy, speech therapy, bladder training with pelvic floor exercises, and cognitive-behavioral therapy.

Conclusion: This case highlights the clinical significance of recognizing symptomatic palatal myoclonus as a manifestation of bilateral cerebrovascular events affecting the dentato-rubro-olivary pathway. Early identification of this distinctive clinical sign, coupled with appropriate neuroimaging demonstrating lesions within the Guillain-Mollaret triangle, is essential for accurate diagnosis, risk stratification, and implementation of targeted therapeutic interventions. Clinicians should maintain heightened awareness of this uncommon presentation in patients with recurrent cerebrovascular disease.

Keywords: Palatal myoclonus, Guillain-Mollaret triangle, cerebrovascular accident, hypertrophic olivary degeneration, dentato-rubro-olivary pathway.

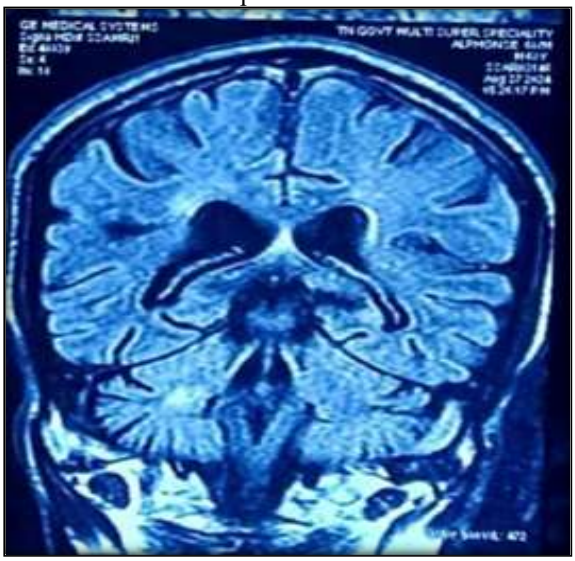
INTRODUCTION

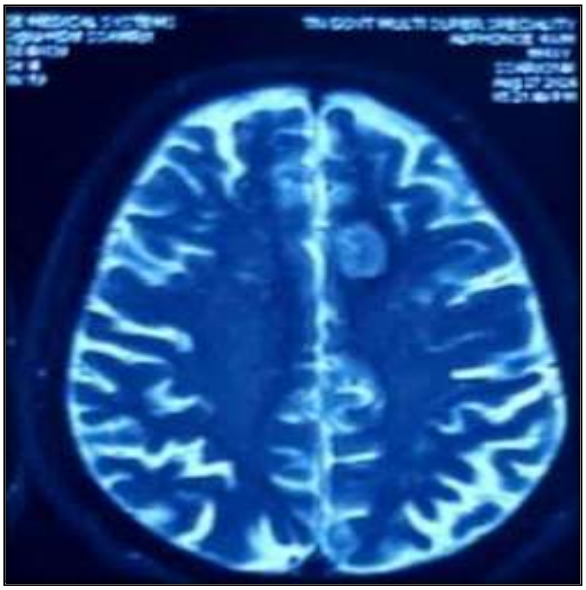
Palatal myoclonus is a rhythmic jerky movement disorder of the soft palate.[1] It has been classified into two subtypes based on etiology: 1) essential palatal myoclonus, which occurs due to contraction of the tensor veli palatini muscle stimulated by the fifth cranial nerve and has no underlying pathology, and 2) symptomatic palatal myoclonus, caused by a

lesion in the dentato-rubral-olivary pathway that leads to contraction of the levator veli palatini muscle stimulated by the ninth and tenth cranial nerves.[2] The symptomatic form is usually associated clinically with tinnitus and progressive ataxia,[1,2] along with hypertrophic degeneration of the inferior olivary and dentate nuclei observed on magnetic resonance imaging (MRI), which is believed to cause the jerky movements.[1-3] We report a rare case of palatal myoclonus following a chronic stroke.

CASE PRESENTATION

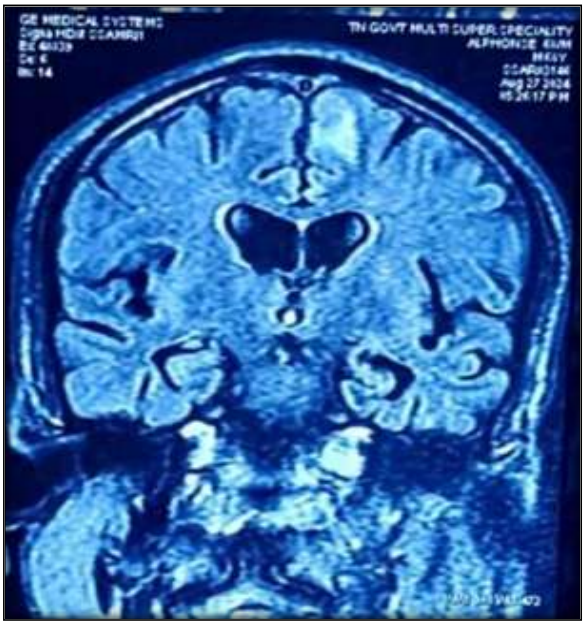
A 68-year-old man with a history of type 2 diabetes mellitus and hypertension presented with a three-day history of unsteady gait, involuntary urination, and behavioral changes with emotional instability. He also reported two months of dysarthria and tinnitus. He denied any sensory disturbances, headache, vomiting, fever, or upper respiratory infections, and no trauma or head injury. His medications included metformin and amlodipine.





On examination, he was afebrile, with a pulse of 86 bpm, respiratory rate of 14 breaths per minute, blood pressure of 144/89 mmHg, and an SpO2 of 98% on room air. Neurological assessment showed impaired higher mental function with mild cognitive impairment (mini-mental status score of 20/30), increased tone (grade 2) in the right upper and lower limbs (modified Ashworth scale), brisk deep tendon reflexes (3+) on the right side, and an extensor plantar response on the right. Muscle strength was 5/5 on the left side and 4/5 on the right lower limb. Cerebellar signs included nystagmus and truncal ataxia. Sensory examination was normal. Cranial nerve exam revealed palatal myoclonus, characterized by rhythmic, involuntary movements of the soft palate. Respiratory, cardiovascular, abdominal, and skin examinations were unremarkable.

The complete hemogram, lipid profile, blood sugar, renal and liver functions, and electrolyte levels all fell within normal ranges. Serum homocysteine, electrophoresis, and antiphospholipid antibody tests were normal. Doppler studies of the carotid and vertebral arteries showed no abnormalities. A 12-lead ECG was performed, with unremarkable results. Echocardiography revealed no abnormalities. Additionally, 24-hour Holter monitoring showed no evidence of paroxysmal arrhythmias. MRI of the brain revealed an acute infarct in the left frontal parasagittal region and an infarct in the Guillain-Mollaret triangle.



The patient was then administered aspirin 150mg, atorvastatin 10mg, and metformin 500mg. Sodium valproate was prescribed to address palatal myoclonus, along with supplementary multi-vitamins. Interventions included limb physiotherapy for paresis, speech therapy for dysarthria, bladder training and pelvic floor (Kegel) exercises for urinary incontinence, and cognitive-behavioral therapy for emotional liability.

DISCUSSION

The Guillain-Mollaret triangle involves the ipsilateral red nucleus in the midbrain, the inferior olivary nucleus (ION) in the medulla, and the contralateral dentate nucleus in the cerebellum, forming the dentato-rubro-olivary pathway.[3,4] Efferent fibers from the dentate nucleus exit through the superior cerebellar peduncle, cross in the brachium conjunctivum, and synapse with the contralateral red nucleus. From there, efferent fibers from the red nucleus travel via the central tegmental tract and stimulate the ipsilateral inferior olivary nucleus. The inferior olivary nucleus completes the triangle by connecting to the contralateral cerebellum via the inferior cerebellar peduncle. The fibers from the inferior olivary nucleus do not directly project to the dentate nucleus but first synapse in the cerebellar cortex through the olivocerebellar tract before projecting to the dentate nucleus.[5,6] This neural circuit plays a role in fine motor control. Any pathology within this triangle, except for olivodentate tract disruption—which causes cerebellar atrophy,[7] and disinhibits (activates) the inferior olivary nucleus—results in hypertrophy of the olivary nucleus. This hypertrophy can produce rhythmical discharges that may clinically appear as oculopalatal tremor.[4]

There is a reciprocal connection between the dentate nucleus and ION mediated via GABAergic pathways; loss of this connection can lead to hypertrophic degeneration of the olivary nucleus.[3,7,8] Symptoms may take weeks or months to appear. Evidence suggests that hypertrophy results from lesions in the dentato-rubral or rubro-olivary pathways, rather than from nearby lesions.[6,9] The muscles of the palate are innervated by the glossopharyngeal (9th cranial nerve) and vagus (10th cranial nerve) nerves. The figure above shows that the vertebrobasilar system nourishes the triangle. Any infarct, ischemia, arteriovenous malformation, or neoplasm that interrupts blood flow to these nuclei can potentially impair the Guillain-Mollaret triangle's function, leading to symptomatic palatal tremor (SPT).[10-12]

These muscles originate from the nucleus ambiguous in the medulla. The levator veli palatini, mainly innervated by the 10th cranial nerve, plays a key role in palatal myoclonus.[13,14] Although the exact cause of palatal myoclonus remains unclear, it is suggested that the close proximity between the inferior olivary nucleus (ION) and the nucleus ambiguus could be a contributing factor. Other parts of the body, including the head, ocular muscles, diaphragm, and extremities, may be affected by olivary degeneration, but they do not necessarily show overt myoclonus.[14]

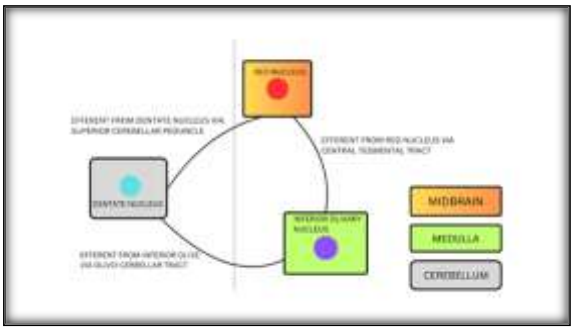


Table-1: Blood Supply of the **Guillain Mollaret triangle**

Nucleus	**Blood Supply**
Dentate nucleus	Superior cerebellar artery Posterior inferior cerebellar artery (PICA).
ION	PICA Anterior spinal artery Basilar artery Vertebral artery
Red nucleus	Posterior cerebral artery Branches from the thalamo-perforating arteries

Our patient exhibited symptoms aligning with a stroke in the left para-sagittal brain region, specifically in the anterior cerebral artery territory. Further detailed history and examination revealed dysarthria, tinnitus, and palatal myoclonus, indicating a previous stroke within the Guillain-Mollaret triangle.

Management of palatal myoclonus remains challenging because clear treatment guidelines are lacking due to the condition's rarity. Patients with the symptomatic form often respond poorly to medical therapy. In fact, only a small proportion, around 20%, experience partial improvement with drugs such as barbiturates, phenytoin, carbamazepine, clonazepam, 5-hydroxytryptophan, other anticonvulsants, anxiolytics, or sedatives. Some patients have reported limited benefit with white noise masking. More advanced approaches like deep brain stimulation are still being studied. In contrast, essential palatal myoclonus, usually affecting the tensor veli palatini muscle, responds well to botulinum toxin injections. Injecting botulinum toxin type A into the palatal muscles under electromyography (EMG) guidance has shown consistent effectiveness. Surgical options, such as tympanic membrane perforation or cutting the levator palatini, tensor veli palatini, and tensor tympani muscles, have been explored, but they tend to offer limited long-term benefits.[15]

CONCLUSION

Palatal myoclonus is an uncommon neurological condition linked to damage in the Guillain-Mollaret triangle, which includes the red nucleus, dentate nucleus, and inferior olivary nucleus. An infarction disrupting the dentato-rubro-olivary pathway can cause hypertrophic olivary degeneration, resulting in symptomatic palatal myoclonus. Identifying this clinical sign and understanding its anatomical basis are vital for correct diagnosis and effective neuroimaging assessment.

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