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CASE REPORT

Isolated Palatal Myoclonus Following SARS-CoV-2 Infection: A Case Report

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ABSTRACT

Palatal myoclonus (PM) is a rare hyperkinetic movement disorder characterized by rhythmic involuntary contractions of the soft palate, sometimes producing audible clicks. We report the case of a 59-year-old woman presenting with persistent isolated PM several months after PCR-confirmed SARS-CoV-2 infection. Extensive neurological, laboratory, neurophysiological, and neuroimaging evaluations revealed no structural, autoimmune, metabolic, or infectious abnormalities. The disorder remained stable without progression over long-term follow-up, and symptomatic management with low-dose clonazepam provided partial relief. The temporal sequence raises the possibility of a post-infectious contribution, although the underlying mechanism remains uncertain. This observation expands the spectrum of focal movement disorders reported following systemic viral illnesses and underscores the importance of recognizing atypical presentations when routine investigations are unrevealing. It also illustrates the generally benign course of isolated PM in the absence of identifiable structural pathology.

Keywords: Palatal Myoclonus; Palatal Tremor; SARS-CoV-2; Post-COVID-19 Syndrome; Cochleovestibular Syndrome.

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1. INTRODUCTION

Palatal myoclonus (PM), also referred to as velopalatal myoclonus or palatal tremor, is an uncommon movement disorder characterized by rhythmic, rapid involuntary contractions (1-3 Hz) of the soft palate musculature [1]. These movements can be unilateral or bilateral and may involve other structures such as the pharynx, larynx, eyes, face, tongue, or diaphragm [1, 2]. A frequent symptom is an audible click, attributed to the rhythmic opening of the Eustachian tube. Unlike most movement disorders, PM often persists during sleep [1, 2].

Although some authors have classified this disorder as a tremor, the original term 'myoclonus' remains more appropriate [2]. Indeed, unlike tremor, which is characterized by continuous sinusoidal movements produced by rhythmic agonist-antagonist contractions, PM is distinguished by bursts of isolated agonist contractions lasting 40-200 ms, separated by periods of silence [2].

The disorder is classified as essential (idiopathic) or symptomatic (secondary). Essential PM (EPM) occurs without detectable structural lesions and is frequently associated with audible clicks attributed to contraction of the tensor veli palatini muscle. Symptomatic PM (SPM) is typically associated with lesions involving the dentato-rubro-olivary pathway—classically the Guillain-Mollaret triangle-and often presents with cerebellar signs or ocular oscillations (oculopalatal tremor) [1, 2, 3]. Such lesions can be caused by stroke, trauma, tumor, or demyelinating disease [2]. The underlying pathophysiology is thought to involve hyperexcitability of the inferior olive, generating persistent oscillations within brainstem circuits [2].

The COVID-19 pandemic has been associated with a broad spectrum of neurological complications, including movement disorders such as myoclonus, ataxia, and tremor [4, 5]. Among these, myoclonus has been reported, though typically in generalized or encephalitic forms [4, 6]. PM following SARS-CoV-2 infection remains exceedingly rare. This report describes a unique case of isolated PM occurring several months after SARS-CoV-2 infection, discusses the diagnostic challenges, the potential causal link, and provides a long-term clinical perspective.

2. CASE REPORT

A 59-year-old woman with a notable family history of cochleovestibular disorders (early-onset deafness in a brother, tinnitus and vertigo in a sister) presented with a complex clinical picture. In early 2020, she gradually developed rotational vertigo, tinnitus, and decreased left-sided hearing acuity. In June 2020, she contracted SARS-CoV-2, confirmed by a positive PCR test, with 25% pulmonary involvement. Five months post-infection (November 2020), she experienced an abrupt worsening of hearing, a sensation of fullness in the ipsilateral ear, tinnitus, and vertigo. By February 2021, persistent, audible bilateral clicking had developed, exacerbated by stress but reducible with external pressure from the palm on the pinna. The patient also reported diffuse headaches and neck pain.

Neurological examination revealed bilateral PM, visible upon mouth opening as rhythmic, involuntary contractions at a frequency of 1.5-2 Hz, producing an objectively audible click. There was no laryngeal, pharyngeal, or respiratory extension; dysphagia; dysarthria; facial weakness; ocular myoclonus; ataxia; or limb involvement. The palatal reflex was present and symmetrical. The remainder of the neurological examination was unremarkable. ENT evaluation confirmed rhythmic elevation of the soft palate without uvular deviation.

Extensive laboratory investigations—including glucose, renal and hepatic function, lipid panel, ionogram, calcium-phosphate, amylase/lipase, complete blood count, thyroid function (TSH), inflammatory markers (CRP, erythrocyte sedimentation rate), vitamins B9 and B12, CA19-9, autoimmunity screening (ANA, ENA, anti-smooth muscle, anti-mitochondrial, anti-LKM/ER antibodies), and infectious serologies (HIV, syphilis, hepatitis B/C) - were unremarkable. The only notable findings were a mildly elevated total cholesterol (2.57 g/L) and vitamin D insufficiency (25OHD at 23 ng/ml). Serial audiograms confirmed stable, bilateral, asymmetric sensorineural hearing loss, more severe on the left, compatible with a diagnosis of endolymphatic hydrops (possibly familial Ménière disease) in addition to presbycusis. Videonystagmography revealed left canal hyporeflexia.

Electroencephalography demonstrated normal background activity without epileptiform discharges. Imaging studies (brain CT, brain MRI with T1, T2, FLAIR, DWI, SWI sequences, and angiography) were normal, showing no brainstem lesion, olivary hypertrophy, or abnormalities of the dentato-rubro-olivary pathway (Figure 1).

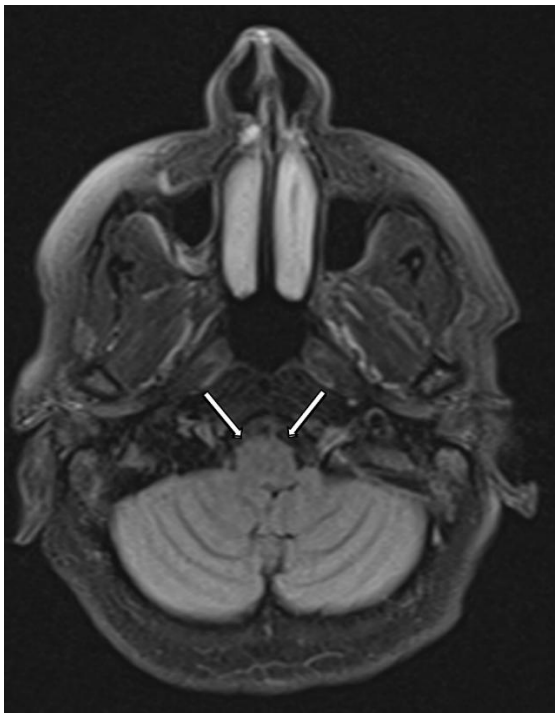

Figure 1. Axial T2-Weighted Fluid Attenuated Inversion Recovery (FLAIR) Magnetic Resonance Imaging (MRI). Absence of olivary hypertrophy or brainstem lesion.

Treatment with clonazepam (0.25 mg, 5 drops nightly, later adjusted to 0.25-0.5 mg nightly) was initiated, resulting in a subjective improvement of approximately 35% in the myoclonus, headaches, and tinnitus. Botulinum toxin therapy was proposed but deferred by the patient.

All clinical data were reported in a strictly anonymized manner in accordance with ethical standards.

3. DISCUSSION

This report describes a case of isolated PM occurring in a post-COVID-19 context, with a five-year follow-up indicating symptom stability. The discussion addresses the potential link with SARS-CoV-2, differential diagnosis, and therapeutic management.

Potential Link Between Palatal Myoclonus and SARS-CoV-2

The five-month interval between infection and symptom onset supports a post-infectious mechanism. Post-infectious neurological syndromes following SARS-CoV-2 infection have been increasingly described, including various forms of myoclonus [4], opsoclonus-myoclonus syndrome [6], and other immune-mediated neurological manifestations [7]. Although isolated PM has rarely been reported after COVID-19, delayed movement disorders emerging weeks to months after infection have been documented [8, 9].

However, causality cannot be firmly established. In this case, no cerebrospinal fluid (CSF) analysis was performed to assess inflammatory markers, antineuronal antibodies, or SARS-CoV-2 RNA/serology. This limitation was common during the pandemic and remains a challenge for establishing definitive causality in post-COVID neurological sequelae. In approximately 99% of published cases during the pandemic, the assertion of a causal link relied on three pillars: temporal association, the exclusion of alternative causes, and pathophysiological plausibility—criteria that align perfectly with the present case [4, 7, 8].

It is crucial to note that specific virological tests in the CSF (PCR or intrathecal serology for SARS-CoV-2) were scarcely available, even in European centers. Furthermore, even in specialised institutions with access to such testing, viral RNA was detected only exceptionally rarely in the CSF. Importantly, a negative CSF PCR does not rule out a post-infectious mechanism, which is, by definition, immune-mediated rather than a direct result of viral persistence [7].

Nevertheless, the elements supporting a potential association in this case are: temporal sequence, consistent with other post-infectious movement disorders [4, 8]; absence of alternative etiologies after exhaustive workup; and biological plausibility, given growing evidence of post-viral immune-mediated processes affecting brainstem and cerebellar circuits, including the Guillain-Mollaret triangle [7, 9]. Proposed mechanisms include a para- or post-infectious immune response leading to localized brainstem inflammation or disinhibition of the inferior olive [7, 10].

The subsequent evolution over five years was marked by symptom stabilization, without recurrence or objective progression. This prolonged follow-up provides an additional element of robustness to the clinical interpretation, although it does not establish causality.

Aetiological Considerations and Differential Diagnosis

Common symptomatic causes of PM were systematically ruled out. Normal neuroimaging excluded structural lesions within the Guillain-Mollaret triangle, and the absence of olivary hypertrophy on MRI made SPM unlikely [3, 10]. There was no evidence for metabolic, toxic, hypoxic, or autoimmune etiologies. The presence of audible clicks is a feature more commonly associated with EPM [2, 3]. However, EPM typically manifests in younger individuals, making this etiology less likely in our 59-year-old patient.

Therefore, the most plausible diagnosis is symptomatic PM of post-infectious origin. The concomitant cochleovestibular pathology, diagnosed as endolymphatic hydrops, may have been triggered or exacerbated by the same infection, representing a multifocal post-COVID syndrome. Regarding vaccination, the patient received her first dose after the onset of PM, making a causal link highly improbable. Published cases of PM post-vaccination are anecdotal [9].

Therapeutic Management

The management of PM is challenging and primarily pharmacological. Clonazepam, due to its antimyoclonic and GABAergic properties, is a first-line treatment [11, 12]. Our patient showed notable improvement with very low doses, consistent with literature reports [11, 13]. Its mechanism likely involves GABAergic modulation of hyperexcitable neuronal oscillators in the inferior olive [10, 14].

Botulinum toxin injections constitute an effective treatment for focal, disabling PM [13]. Its use was appropriately proposed but postponed by the patient. Other therapeutic alternatives have been explored with limited and inconsistent success. While initial

pathophysiological hypotheses centered on serotonin, trials with serotonergic agents such as 5-HTP/carbidopa or sumatriptan have proven disappointing and non-reproducible [1, 2, 14]. Other molecules like baclofen or valproate have only anecdotal efficacy and are considered second-line options [10, 11]. Given the benign long-term evolution and partial symptomatic relief, conservative management with clonazepam was deemed appropriate.

4. CONCLUSION

We report a unique case of isolated palatal myoclonus with a suggestive temporal relationship following SARS-CoV-2 infection. The exhaustive workup excluded other principal causes, while the clinical features point towards a post-infectious etiology. Although a definitive causal link cannot be established—primarily due to the lack of specific CSF analysis, which was a widespread limitation during the pandemic—the temporal sequence, absence of alternative causes, and pathophysiological plausibility strongly support this hypothesis. The concomitant cochleovestibular syndrome may represent another manifestation. This case broadens the spectrum of post-COVID-19 movement disorders and underscores the virus's potential to trigger focal neurological phenomena via likely immune-mediated mechanisms. The patient's five-year stability supports the generally benign course of isolated PM in this context. Optimal management relies on symptomatic pharmacological therapy with clonazepam and the consideration of botulinum toxin for refractory symptoms.

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