Cureus
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Open Access Case Report

Neuropathic Tremor During Recovery From Guillain–Barré Syndrome: A Case Report and Pathophysiological Insights

Review began 10/09/2026
Review ended 10/18/2026
Published 10/21/2026

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DOI: 10.7759/cureus.118298

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Abstract

Neuropathic tremor is a movement disorder associated with peripheral neuropathy. Mainly described in hereditary and chronic inflammatory neuropathies, it remains rarely reported in Guillain-Barré syndrome (GBS). We report the case of a 15-year-old boy who developed an asymmetric postural tremor of the upper limbs, with a kinetic component during writing and associated dystonic posturing during recovery from a demyelinating form of GBS. Propranolol provided partial improvement. The pathophysiology remains uncertain but may involve disrupted peripheral sensorimotor feedback promoting maladaptive central oscillatory activity, possibly within cerebellar networks. Further neurophysiological studies are required to clarify the mechanisms underlying GBS-associated tremor and dystonia.

Categories: Neurology
Keywords: central oscillator, dystonia, guillain-barré syndrome, neuropathic tremor, upper limbs

Introduction

Tremor is an involuntary, rhythmic, oscillatory movement of a body part, typically resulting from alternating or synchronous contractions of antagonistic muscle groups [1]. It is one of the most common movement disorders and may be physiological or pathological, occurring in various neurological conditions, including Parkinson's disease, essential tremor, and certain peripheral neuropathies [1-4]. Neuropathic tremor is more commonly observed in demyelinating than in axonal neuropathies [3-7]. In Guillain-Barré syndrome (GBS), it has received limited attention and may be underrecognized [8-10]. We report the case of a patient who developed neuropathic tremor during the recovery phase of GBS and discuss the potential underlying pathophysiological mechanisms.

Case Presentation

A 15-year-old patient developed a demyelinating form of GBS with respiratory involvement, requiring a one-month stay in the intensive care unit, endotracheal intubation, and mechanical ventilation. He received a course of intravenous immunoglobulin therapy, followed by progressive motor recovery. Although the GBS was classified as demyelinating at the time of diagnosis, the detailed nerve conduction study and cerebrospinal fluid reports were unavailable for retrospective review. Detailed information regarding clinical or electrophysiological side-to-side asymmetry during the acute phase of GBS was also unavailable.

One month after motor recovery, the patient developed a low-amplitude hand tremor that was asymmetric and more pronounced on the left side. The tremor was predominantly postural and more pronounced in the left upper limb, as demonstrated by spiral drawing with the right hand (Figure *1A*) and left hand (Figure *1B*). It had no resting component and exhibited a mild kinetic component during writing. It was associated with dystonic posturing of both hands (Figure *2*). The dystonic posturing was limited to the hands and was not observed in the lower limbs, trunk, or during walking. Writing did not induce or worsen the dystonic posturing, and no abnormal elevation or posturing of the shoulder or elbow was observed.

How to cite this article
El Hannach H, Ouhabi D, Naciri Darai H, et al. (October 21, 2026) Neuropathic Tremor During Recovery From Guillain–Barré Syndrome: A Case Report and Pathophysiological Insights. Cureus 18(10): e118298. DOI 10.7759/cureus.118298

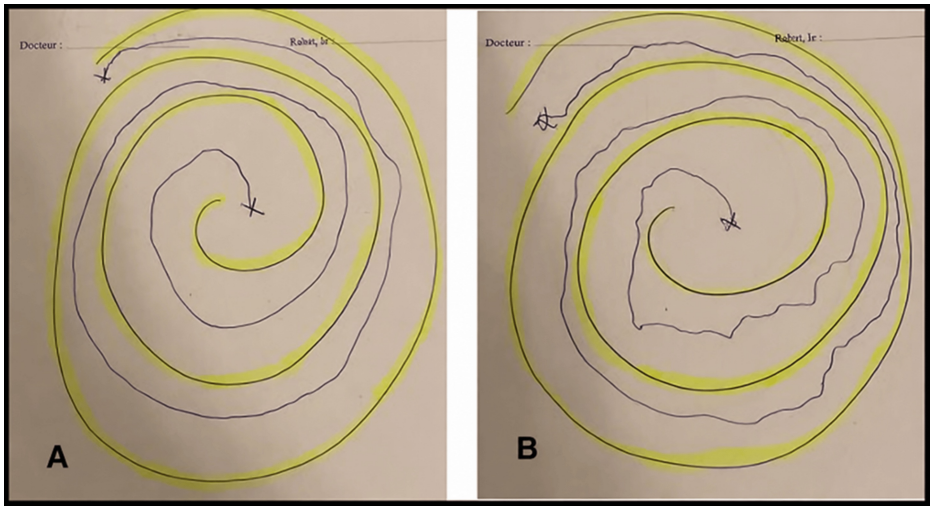


FIGURE 1: Spiral drawing demonstrating bilateral upper-limb tremor.

(A) Spiral drawn with the right hand. (B) Spiral drawn with the left hand, showing a more pronounced tremor.



FIGURE 2: Dystonic posturing of both hands.

Dystonic posturing involving both hands.

There was no family history of hand or head tremor and no exposure to medications or toxic substances that could account for the tremor. The tremor showed neither distractibility nor suggestibility. Neurological examination revealed bilateral thenar muscle atrophy and mild symmetrical paraparesis, with muscle strength graded at 4+/5 in both lower limbs, while strength was preserved at 5/5 in both upper limbs. The sensory examination was normal, including joint position sense, which was specifically tested in the fingers of both hands. Deep tendon reflexes were present in all four limbs.

Liver, kidney, and thyroid function tests, as well as blood glucose, calcium, and phosphate levels, were within normal limits (Table *1*). Wilson disease and genetic causes of dystonia were not specifically investigated. Symptomatic treatment with propranolol 20 mg three times daily resulted in partial improvement of the tremor. The patient was followed clinically for two years.

Parameter	Patient's result	Reference range
Blood glucose	0.8 g/L	0.70–1.10 g/L
Creatinine	6 mg/L	5.7–12.5 mg/L
Aspartate aminotransferase	17 U/L	<35 U/L
Alanine aminotransferase	19 U/L	<35 U/L
Thyroid-stimulating hormone	3.35 mIU/L	0.40–4.00 mIU/L
Calcium	98 mg/L	88–102 mg/L
Phosphate	40 mg/L	25–45 mg/L

TABLE 1: Laboratory test results.

Written informed consent for publication of this case and the accompanying images was obtained directly from the patient after he reached the age of 18 years.

Discussion

Our patient developed an asymmetric postural hand tremor with a kinetic component during writing while recovering from a demyelinating form of GBS. Based on the current two-axis classification, this phenotype corresponds to an acquired tremor syndrome associated with GBS [1]. The tremor was of low amplitude and predominantly affected the left hand, and was absent at rest. It was accompanied by hand dystonia. The temporal relationship with GBS, together with the absence of a family history, relevant drug or toxin exposure, or metabolic abnormalities, supported the diagnosis of an acquired neuropathic tremor.

Neuropathic tremor is a movement disorder caused by, or occurring in association with, a lesion of the peripheral nervous system [2-4]. It is relatively uncommon and typically presents as an action tremor predominantly affecting the distal upper limbs, although a resting component may occasionally occur. Neuropathic tremor is more frequently associated with demyelinating than with axonal neuropathies. It has been particularly described in IgM monoclonal gammopathy-associated neuropathies [5], chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) [6], multifocal motor neuropathy with conduction block [3], and hereditary neuropathies, particularly Charcot-Marie-Tooth disease (CMT) [7].

Neuropathic tremor following GBS was initially documented through isolated cases and small series [8,9]. In the 2023 series by Rajan et al., which included five patients, tremor emerged during the recovery phase, between three months and five years after GBS onset, when muscle strength had almost completely recovered [9]. More recently, Baizabal-Carvallo et al. identified upper-limb tremor in 16 of 18 patients evaluated after GBS, although only 35.5% reached a Fahn-Tolosa-Marin Tremor Rating Scale score of ⩾10 [10]. These findings suggest that mild tremor following GBS may be underrecognized. The phenotype reported by Rajan et al. was relatively homogeneous, consisting of a fine, rapid, mildly jerky postural tremor with a frequency of 8-10 Hz, predominantly affecting the upper limbs. Dystonic posturing was observed in four of the five patients. Our case shared several of these features, including the postural nature and upper-limb distribution of the tremor, as well as its association with dystonia.

From a physiological perspective, tremor may arise from four non-mutually exclusive mechanisms: mechanical oscillations of the affected body part, oscillations generated within peripheral reflex loops, central neuronal oscillators, and disturbances of feedforward or feedback motor control [11]. More recent neurophysiological evidence indicates that pathological tremor frequently emerges from interactions between distributed oscillatory networks rather than from a single anatomically fixed generator [12]. Within this framework, an oscillatory source may initiate or drive rhythmic activity, while its propagation, amplification, and translation into visible movement depend on interactions within a broader sensorimotor network. Importantly, peripheral sensory signals can access and modulate central tremor networks, as supported by the identification of proprioceptive inputs to tremor-related neurons in thalamic regions receiving cerebellar projections [13]. Different tremor syndromes illustrate complementary mechanisms through which an oscillatory source may interact with a broader motor network. In Parkinsonian tremor, Dirkx et al. showed that tremor-related activity may arise within the internal globus pallidus and subsequently propagate through the motor cortex to the cerebello-thalamo-cortical circuit [14]. This model distinguishes the structure involved in initiating tremor from the network responsible for its amplification and clinical expression. Essential tremor provides a different model. The resting-state functional MRI findings of Nicoletti et al. demonstrated altered intrinsic connectivity within the cerebello-thalamo-cortical network, supporting the concept that tremor may emerge from abnormal coupling and synchronization among distributed oscillatory nodes rather than from a single anatomical generator [15]. Cerebellar tremor

in multiple sclerosis illustrates how structural disruption of a motor-control network may generate oscillatory activity. Boonstra et al. found that tremor severity was associated with structural abnormalities involving the thalamus and superior cerebellar peduncle, supporting the involvement of the cerebello-thalamic pathway [16]. Demyelination or degeneration within this pathway may disrupt the temporal organization of cerebellar motor signals and impair the correction of ongoing movement. Finally, inflammatory neuropathies demonstrate how altered peripheral input may interact with central motor networks. Schwingenschuh et al. found impaired cerebellar associative learning specifically in neuropathic patients with tremor, suggesting that an inability of the cerebellum to adapt appropriately to altered peripheral afferent input may contribute to tremor development [17]. Consistently, Silsby et al. found that tremor in CIDP displayed features of central generation while remaining associated with peripheral nerve conduction abnormalities [18]. These complementary pathophysiological hypotheses are summarized in Table *2*. Collectively, they indicate that the anatomical site of the initial dysfunction does not necessarily correspond to the network producing the visible tremor. They also suggest that tremor may emerge when an oscillatory drive is amplified or synchronized within the cerebello-thalamo-cortical network, particularly when the timing of sensory or motor information is disrupted.

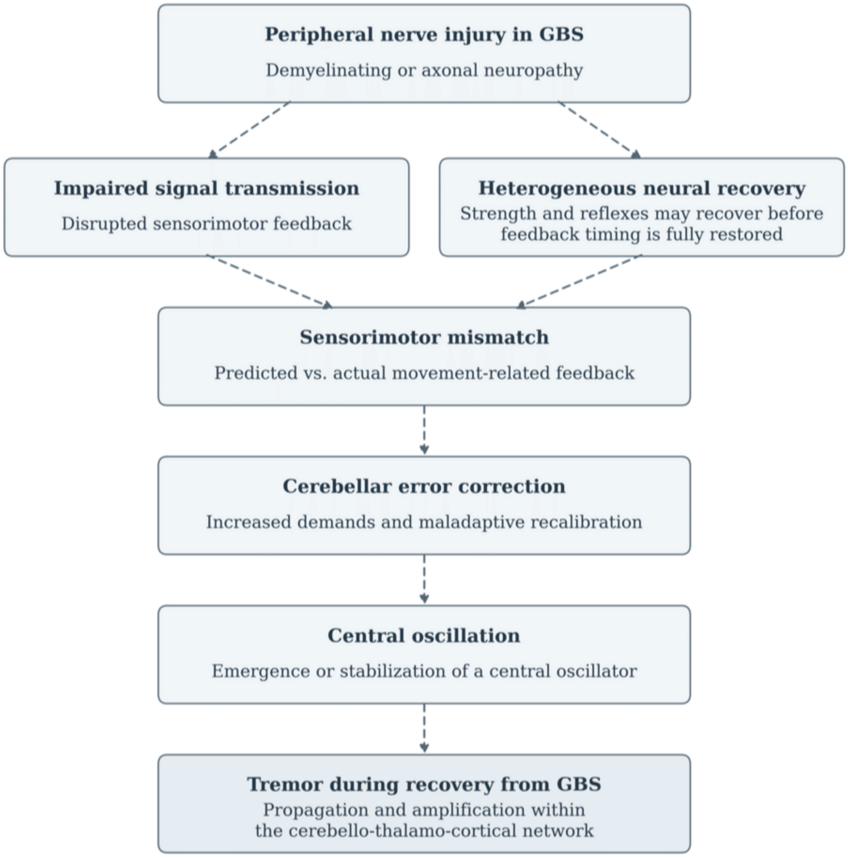
Tremor syndrome and reference	Initial dysfunction	Main network involved	Proposed pathophysiological mechanism	Relevance to neuropathic tremor following GBS
Parkinsonian tremor (Dirkx et al.) [14]	Dopamine depletion and abnormal basal ganglia activity	Basal ganglia and cerebello-thalamo-cortical network	Tremor-related activity originates in the internal globus pallidus and is subsequently propagated through the motor cortex to the cerebello-thalamo-cortical circuit, which contributes to tremor amplification and motor expression	Demonstrates that the structure initiating the tremor may differ from the network responsible for its clinical expression
Essential tremor (Nicoletti et al.) [15]	Abnormal functional organization of the central motor network	Cerebellum, thalamus, and motor cortex	Tremor may result from abnormal functional coupling and mutual entrainment of multiple oscillatory nodes rather than from a single anatomical oscillator	Supports the possibility that altered peripheral input may recruit or synchronize a pre-existing central oscillatory network
Cerebellar tremor in multiple sclerosis (Boonstra et al.) [16]	Structural damage to cerebellar output pathways	Cerebellum, superior cerebellar peduncle, thalamus, and motor cortex	Demyelination and degeneration of the cerebello-thalamic pathway disrupt the timing and integration of cerebellar motor signals, thereby promoting tremor	Provides a model in which demyelination and temporal disorganization within a motor-control network can generate tremor
Neuropathic tremor in inflammatory neuropathies (Schwingenschuh et al.) [17]	Altered peripheral afferent input secondary to demyelinating neuropathy	Peripheral sensorimotor loops interacting with cerebellar circuits	Altered peripheral feedback may induce maladaptive cerebellar plasticity. Impaired cerebellar associative learning in tremulous patients suggests that abnormal cerebellar adaptation contributes to tremor development	Indicates that peripheral nerve damage alone may be insufficient and that abnormal cerebellar adaptation could determine which patients develop tremor
Neuropathic tremor following GBS (Rajan et al.) [9]	Peripheral nerve injury followed by heterogeneous recovery	Presumed interaction between peripheral sensorimotor feedback and a central, possibly cerebellar, oscillator	Electrophysiological findings support a central oscillator modulated by altered peripheral sensorimotor feedback. During recovery, temporally disorganized afferent signals may promote maladaptive cerebellar adaptation and recruit a central oscillatory network*	Provides an integrative hypothesis explaining why tremor may emerge during recovery, despite near-complete restoration of muscle strength

TABLE 2: Pathophysiological mechanisms proposed for different tremor syndromes.

*: The proposed sequence involving temporally disorganized afferent input, maladaptive cerebellar adaptation, and recruitment of a central oscillator represents a pathophysiological hypothesis derived from the available evidence, rather than a mechanism directly demonstrated in Guillain-Barré syndrome (GBS).

These models provide a framework for interpreting the electrophysiological findings of Rajan et al. in GBS-associated neuropathic tremor [9]. In their series, tremor frequency remained relatively stable after mechanical loading, and corresponding spectral peaks were identified on accelerometry and surface electromyography, supporting a centrally generated rather than a purely mechanical or reflex oscillation.

Although these findings did not localize the central oscillator, Rajan et al. hypothesized that it might involve the cerebellum. This interpretation was based on previous evidence from inflammatory neuropathies, particularly the impaired eyeblink classical conditioning demonstrated by Schwingenschuh et al. in patients with neuropathic tremor, suggesting abnormal cerebellar associative learning [17]. Based on these findings and on the preceding models, we propose that peripheral nerve injury in GBS may create the conditions for the emergence of a central oscillation rather than directly generate tremor. Regardless of whether the underlying neuropathy is primarily demyelinating or axonal, impaired peripheral signal transmission and heterogeneous neural recovery may disrupt sensorimotor feedback. Motor strength and tendon reflexes may recover before the temporal and functional organization of sensorimotor feedback has been fully restored. The resulting mismatch between predicted and actual movement-related feedback could place increased demands on cerebellar error-correction mechanisms. Maladaptive cerebellar recalibration may then facilitate the emergence or stabilization of a central oscillator, whose activity could subsequently be propagated and amplified within the cerebello-thalamo-cortical network, producing tremor during recovery from GBS (Figure *3*).



Dashed arrows indicate hypothesized relationships, not established causal mechanisms.

FIGURE 3: Proposed pathophysiological mechanism of neuropathic tremor during recovery from Guillain–Barré syndrome (GBS).

Our patient also exhibited dystonic posturing, a feature reported in four of five patients by Rajan et al. [9]. Although proprioceptive loss may cause pseudodystonia [19], the absence of sensory deficits in their series and in our patient makes this explanation unlikely. Subtle impairment of sensorimotor integration and maladaptive central plasticity induced by altered peripheral input may represent an alternative, although still hypothetical, mechanism [2]. Neuropathic tremor predominantly affects the distal upper limbs, as observed in our patient and in the series of Rajan et al. [9]. This distribution is noteworthy because GBS usually begins in and more severely affects the lower limbs. The reason for this discrepancy remains unknown.

There is no established treatment for GBS-associated neuropathic tremor, and the response to symptomatic agents appears variable. Propranolol produced inconsistent results in the series of Rajan et al. [9], while pregabalin was effective in an isolated case [20]. In our patient, propranolol provided partial improvement.

Conclusions

We report a case of neuropathic tremor emerging during recovery from a demyelinating form of GBS, illustrating a potentially underrecognized manifestation of this neuropathy. Its pathophysiology remains uncertain but may involve disrupted peripheral sensorimotor feedback and maladaptive central oscillatory mechanisms. Further neurophysiological studies are needed to clarify the mechanisms underlying both tremor and associated dystonic posturing.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Hajar El Hannach, Wafa Regragui

Acquisition, analysis, or interpretation of data: Hajar El Hannach, Dahab Ouhabi, Hajar Naciri Darai, Ali Benomar, Houyam Tibar, Wafa Regragui

Drafting of the manuscript: Hajar El Hannach, Dahab Ouhabi, Hajar Naciri Darai, Ali Benomar, Houyam Tibar, Wafa Regragui

Critical review of the manuscript for important intellectual content: Dahab Ouhabi, Hajar Naciri Darai, Ali Benomar, Houyam Tibar, Wafa Regragui

Supervision: Wafa Regragui

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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