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Prolonged bilateral vocal cord paralysis as an isolated sequela of fulminant Guillain-Barré syndrome after Campylobacter jejuni enteritis

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Case Report

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Abstract

Background

Guillain-Barré syndrome (GBS) rarely presents with bilateral vocal cord paralysis (BVCP), and persistent BVCP as an isolated long-term sequela is exceptionally uncommon. We describe the clinical course of fulminant axonal GBS complicated by prolonged BVCP and discuss its implications for airway management.

Case presentation

A previously healthy man in his early 50s developed rapidly progressive tetraplegia, multiple cranial neuropathies and respiratory failure several days after an episode of enteritis, ultimately progressing to a near-locked-in state. Anti-GT1a IgG antibodies were strongly positive, and serology was consistent with recent *C. jejuni* infection. Despite immunotherapy and near-complete recovery of limb strength and bulbar function, serial laryngoscopy demonstrated persistent BVCP with vocal cords fixed in the paramedian position, precluding decannulation and necessitating long-term tracheostomy.

Conclusion

BVCP may persist as the sole disabling sequela even after marked systemic motor recovery in severe axonal GBS, potentially reflecting the length-dependent vulnerability and protracted reinnervation of the recurrent laryngeal nerve. Upper airway evaluation should be incorporated into ventilator weaning protocols and tracheostomy management, particularly in severe GBS.

Background

Guillain-Barré syndrome (GBS) is an acute peripheral neuropathy caused by autoimmune cross-reactivity, often triggered by infectious enteritis due to *Campylobacter jejuni* or various respiratory tract infections. The disease typically presents with flaccid limb paralysis; however, facial nerve palsy, bulbar palsy, or respiratory failure can occasionally be the initial manifestations. Although the symptoms of GBS typically reach their peak within four weeks after onset and gradually improve thereafter, severe cases may result in long-term disability. Vocal cord paralysis is a rare manifestation of GBS, and persistent vocal cord paralysis as an isolated sequela has been rarely reported. Its pathogenesis remains uncertain and may easily be overlooked, particularly in patients undergoing mechanical ventilation. Here we report a severe case of the axonal GBS in which BVCP persisted for over fifteen months, long after the complete recovery of limb strength and other bulbar functions.

Case presentation

A previously healthy Japanese man in his early 50s developed watery diarrhea after consuming homemade seasoned chicken breast. Four days after the onset of diarrhea, he noticed mild muscle weakness and abnormal tingling sensations in the tip of his tongue and his extremities. He was referred for neurological evaluation, and on the same day (Day 1), intravenous immunoglobulin (IVIg) therapy (400 mg/kg/day for five consecutive days) was initiated under a strong clinical suspicion of GBS. However, his condition deteriorated almost hourly, and he required endotracheal intubation the following day due to respiratory failure. Tracheostomy was performed on Day 7, but his general condition remained critical, with violent fluctuations in blood pressure and the development of new-onset dissecting aneurysm of the celiac artery (Fig. 1). Although he was awake and alert, he rapidly entered an almost completely locked-in state as his paralysis progressed.

On Day 11, his Medical Research Council (MRC) sum score for all muscle groups was zero, and all tendon reflexes were absent. His cranial nerve functions were severely impaired, involving the trigeminal (V), abducens (VI), facial (VII), glossopharyngeal (IX), vagus (X), accessory (XI) and hypoglossal (XII) nerves. He could communicate only through limited eye movements and faint head rotations, and required tube feeding because of bulbar palsy. The soft palate showed poor elevation on both sides. The vocal cord findings during intubation were not documented. Vibration sense was slightly reduced, while other sensory modalities were preserved. Laboratory findings revealed no abnormalities in complete blood count and blood chemistry. Other possible etiologies, including vasculitis, sarcoidosis, myasthenia gravis, connective tissue diseases and intracranial infections were excluded. Serum IgG antibodies reacting strongly with GT1a and weakly with GQ1b were detected (Koga J Neurol (2012)

259:1366–1374). Although blood culture and fecal tests were unremarkable, positive serology for recent *C. jejuni* infection was proven (Koga Neurology (2005) 64:1605–1611). The key clinical data are summarized in Table 1. The nerve conduction study demonstrated that compound muscle action potential (CMAP) and F waves could be hardly evoked in all limbs tested (Fig. 2), findings consistent with severe axonal neuropathy.

Table 1
Laboratory findings on admission.

Blood sample								
RBC	5.58×10^6	/μL	Alb	4.6	g/dL	CRP	0.27	mg/dL
Hb	17.2	g/dL	ALP	83	U/L	BNP	8.5	pg/mL
Ht	50.8	%	ALT	32	U/L	HBs Ag	-	
WBC	7.93×10^3	/μL	AST	29	U/L	HCV Ab	-	
Neut	56.0	%	LDH	225	U/L	RPR	-	
Ly	30.0	%	CK	65	U/L	TPHA	-	
Mono	7.0	%	BUN	12.6	mg/dL	VCA IgM	-	
Eosino	1.5	%	Cr	0.78	mg/dL	CMV IgM	-	
Baso	0.0	%	Glucose	77	mg/dL	ANA	< 40	
pH	7.404					SS-A/Ro Ab	-	
PaO_2	79.6	mmHg	Na	143	mEq/L	SS-B/La Ab	-	
$PaCO_2$	42.4	mmHg	K	4.0	mEq/L	P-ANCA	-	
HCO_3^-	26.0	mmol/L	Cl	104	mEq/L	C-ANCA	-	
Lactate	8.0	mmol/L	Ca	9.8	mg/dL	AChR Ab	-	
			Mg	2.6	mg/dL	MuSK Ab	-	
			P	3.2	mg/dL	ACE	5.3	IU/L
Anti-glycolipid antibody screening						**CSF sample**		
	IgG	IgM				Appearance	transparent	
GM2	-	-				Cell	1	/μL
GM1	1+	-				Polymorpho-nuclear	0.0	%
GD1a	1+	-				Mononuclear	100.0	%
GD1b	1+	-				Protein	60	mg/dL
GT1b	1+	-				Glucose	52	mg/dL
GQ1b	1+	-				pH	8.4	
GalNAc-GD1a	-	-						
GT1a	3+	-						
Gal-C	1+	1+						

Positive results of anti-glycolipid antibodies are displayed in six levels (from 1 + to 6+); Neut – neutrophil; Ly – lymphocyte; Mono – monocyte; Eosino – eosinophil; Baso – basophil; Ag – antigen; Ab – antibody; Gal — galactocerebroside; AChR — acetylcholine receptor; MuSK — muscle-specific tyrosine kinase

His symptoms peaked around Day 14 and gradually improved thereafter; however, the initial recovery was discouragingly slow, as illustrated in Fig. 3. The dissecting aneurysm of the celiac artery was managed conservatively and followed an uneventful course. Approximately 100 days after onset, a subtle improvement in his facial expressions and a significant return of muscle contraction in the lower extremities were observed. Fortunately, the rate of recovery markedly accelerated about seven months after admission, and the patient regained independent ambulation over the following three months.

Recovery of respiratory function was also delayed. He was finally weaned from mechanical ventilation and fitted with a speaking valve approximately six months after tracheostomy. The movement of the soft palate recovered fully. However, he experienced episodes of dyspnea whenever the tracheostomy cannula was displaced or occluded. Laryngoscopic examination revealed bilateral vocal cords fixed in the paramedian position with no detectable abduction during inspiration, accounting for the recurrent dyspnea. Computed tomography (CT) of the trunk and magnetic resonance imaging (MRI) of the brain showed no structural abnormalities along the vagus nerve or its recurrent laryngeal branches. Because the tracheostomy site could not be closed, he has been closely followed after discharge. Fifteen months after onset, BVCP persists as the only residual deficit (Fig. 4 and the additional movie file show this in more detail), whereas all other muscles have regained nearly full strength, except for impaired hand dexterity due to contracture deformities. Electrophysiological studies of the limbs also demonstrated substantial improvement (Fig. 5).

Discussion

GBS is a well-recognized acute immune-mediated motor polyneuropathy that most commonly presents with ascending limb weakness, often following gastrointestinal or respiratory infections, typically with pathogens such as *C. jejuni*. Cranial nerve involvement and ventilator-dependent severe cases are not uncommon in GBS, and it is reported more frequently in Asia than in Western countries [1]. This regional difference is thought to reflect the higher prevalence of the axonal type of GBS, and our case aligns with this clinical phenotype. In general, bulbar palsy, bilateral facial palsy and ophthalmoplegia are well-documented cranial manifestations of GBS; however, clinically significant laryngeal involvement leading to vocal cord paralysis is exceedingly rare. BVCP associated with GBS or its variants has been described only in a small number of case reports and series and may easily be overlooked in the context of severe generalized weakness or prolonged mechanical ventilation. Our case demonstrates a fulminant axonal form of GBS with prolonged ventilator dependence and an unusually protracted course of BVCP that persisted despite near-complete recovery of limb and bulbar motor function.

Previous reports have indicated that BVCP can occur at various stages of GBS, ranging from an early presenting manifestation to a delayed complication. In one case from South Korea, hoarseness and dysphagia preceded limb weakness, leading initially to otolaryngological rather than neurological evaluation and delayed recognition of the underlying neuropathy [2]. Neuromuscular disorders are identified in approximately 26% of patients who initially present with idiopathic vocal cord paralysis [3]. Because these encompass a broad spectrum of diseases, including cerebrovascular disorders, multiple sclerosis and neurodegenerative conditions such as motor neuron disease and multiple system atrophy, the differential diagnosis of acute vocal cord paralysis should always include neuromuscular etiologies. As Kim et al. emphasized, GBS should not be excluded even in the absence of generalized muscle weakness [4]. In other reported cases, upper airway obstruction due to BVCP developed after the diagnosis of GBS, often during recovery from respiratory failure, frequently necessitating prolonged tracheostomy management [5][6]. Our case falls into this latter category: BVCP became clinically evident when ventilator weaning and transition to a speaking valve were attempted. Laryngoscopy revealed bilateral vocal cord immobility, and despite substantial neurological recovery elsewhere, decannulation remained impossible even fifteen months after the onset — highlighting the potential for BVCP to determine long-term airway and functional outcome.

The pathophysiological basis of BVCP in GBS remains incompletely understood, but several mechanisms have been proposed. Because vocal cord movement is primarily controlled by the recurrent laryngeal nerve (RLN), bilateral dysfunction likely reflects a predominantly peripheral motor cranial neuropathy involving the vagus nerve and its branches. This hypothesis is supported by

reports of concomitant lower cranial nerve involvement [7][8][9] and pathological evidence of axonal or demyelinating lesions affecting motor fibers of bulbar muscles [10]. In Fisher syndrome (FS) and FS/GBS overlap, where ophthalmoplegia and ataxia predominate, BVCP has also been observed, suggesting that immune-mediated injury may selectively target multiple cranial nerves in a characteristic pattern [5]. Anti-ganglioside antibodies — particularly anti-GT1a IgG — are closely associated with axonal variants and with pronounced bulbar or lower cranial nerve involvement [11][12]. They are thought to play a cardinal role by binding to ganglioside-rich nodal or paranodal regions and triggering complement-mediated injury. The severe axonal pattern on nerve conduction studies and the strongly positive anti-GT1a antibodies in our patient support this immunopathogenic mechanism.

Furthermore, the persistent BVCP in our case, despite recovery of other lower cranial nerve functions (including soft palate movements), may relate to the exceptional length of the RLN, particularly on the left. Axonal regeneration across long distances is often incomplete or misdirected, leading to simultaneous activation of antagonistic laryngeal muscles and resulting in functional immobility rather than simple paralysis [13]. Historically, Wagner and Grossman proposed that length-dependent nerve injury could cause earlier recovery of the superior laryngeal nerve (SLN) compared with RLN, which innervates the cricothyroid muscle responsible for closing the vocal cords. As a result, bilateral vocal cords may remain fixed in the paramedian position. This anatomical vulnerability may explain why severe laryngeal dysfunction requiring permanent tracheostomy can persist as a long-term sequela even after full recovery of systemic weakness, particularly in cases associated with anti-GT1a antibodies.

From a clinical standpoint, BVCP in the context of GBS presents several diagnostic and management challenges. First, BVCP may masquerade as a primary laryngeal or structural airway disorder, particularly when limb weakness is mild or absent at initial presentation. This can lead to investigations focused on local causes such as trauma, postsurgical injury, malignancy or idiopathic neuropathy. Several reports have shown that initial imaging and routine laboratory evaluations are often unrevealing, and that only the subsequent development of neurological signs, cerebrospinal fluid changes and electrophysiological abnormalities establishes the diagnosis of GBS [2][4][5]. Second, respiratory failure in GBS is usually attributed to diaphragmatic or intercostal muscle weakness; however, BVCP can independently contribute to upper airway obstruction or hinder decannulation despite adequate respiratory muscle recovery. In our case, the patient was successfully weaned from mechanical ventilation but remained tracheostomy-dependent due to persistent BVCP, exemplifying how laryngeal involvement can represent the principal limiting factor in achieving full respiratory recovery.

As summarized in Table 2, the prognosis of BVCP associated with GBS is variable. Some patients experience relatively rapid resolution of vocal cord immobility, paralleling or shortly following systemic neurological recovery, with successful decannulation and complete resolution of hoarseness or stridor within weeks. Others — including several reported and our own patient — show a markedly protracted course, with BVCP persisting for months to over a year, occasionally necessitating permanent tracheostomy. There is substantial heterogeneity in the reversibility of laryngeal dysfunction. Factors potentially influencing prognosis include the underlying GBS subtype, the extent of axonal damage, the presence of anti-ganglioside antibodies associated with severe cranial neuropathy, and possibly patient age or comorbidities; however, conclusive predictors have not been established. Our case represents the first reported case of BVCP in GBS following *C. jejuni* enteritis.

Table 2
Previous reports of GBS presenting with BVCP and their clinical characteristics.

Author /Year	Age /Gender	Preceding infections/pathogens	First symptoms	Other cranial nerve involvement	Progressive muscle weakness	Ventilator use for respiratory failure	Recovery of vocal cord mobility
Lee DH, et al. (2013) [2]	61F	RTI *M. pneumoniae*, EBV?	Hoarseness, dysphagia	-	+ (3 days after onset)	+	+ (1 month)
Son SA, et al. (2022) [4]	82F	COVID-19 vaccination?	Dyspnea	-	-	+	unknown
Bahk J, et al. (2021) [5]	65M	Chickenpox (1 month prior), VZV	Diplopia, dizziness	III (mild bilateral ptosis)	+ (6 days after onset)	+	-
Rodrigues JF, et al. (1984) [6]	64M	RTI	Lower limb weakness	-	+ (the first symptom)	+	-
Turan MI, et al. (2014) [14]	11M	RTI	Tingling and numbness of lower limbs	-	+ (14 days after onset)	-	+
Yoskovitch A, et al. (2000) [15]	70M	RTI	Dyspnea, dysphonia	-	+ (3 days after onset)	- (only tracheostomy)	-
Panosian MS, et al. (1993) [16]	33F	-	Hoarseness, dysphagia, lower limb weakness	III, IV, VI, VII, IX, X, XI, XII	+ (the first symptom)	+	+ (6 weeks)

The reported cases show variation in age and sex, with respiratory tract infections being the most common preceding events. Some patients initially presented with hoarseness or dyspnea, whereas in others BVCP was recognized only after treatment for GBS had begun. All but one patient required tracheostomy due to respiratory failure secondary to respiratory muscle paralysis. Recovery from BVCP appeared more frequent in younger patients, although considerable interindividual variability was observed. None of the available reports specified whether anti-ganglioside antibodies were detected in patient serum. M – male; F – female; RTI – respiratory tract infection

The management of BVCP in GBS requires close multidisciplinary collaboration among neurologists, intensivists and otolaryngologists. Intravenous immunoglobulin and plasmapheresis — the standard disease-modifying therapies for GBS — remain the cornerstone of treatment and should be initiated promptly once GBS is suspected, as earlier intervention is associated with faster recovery and a lower likelihood of prolonged mechanical ventilation in severe cases. However, no specific therapy has been proven to facilitate recovery of BVCP itself. Most cases are managed with supportive airway strategies tailored to symptom severity and the risk of obstruction. Tracheostomy should be considered when upper airway compromise or prolonged ventilatory support is anticipated, and the decisions regarding decannulation should take into account both respiratory muscle strength and laryngeal mobility, typically guided by serial laryngoscopic assessments. In patients with persistent BVCP, surgical options such as posterior cordotomy, arytenoidectomy or other glottic-widening procedures may be considered, although these involve balancing airway patency against phonatory quality [17][18]. A high body mass index (BMI)

and underlying neurological disorders have been associated with a higher risk of surgical treatment failure [19]; therefore, individualized multidisciplinary discussion of surgical and rehabilitative approaches is warranted.

Conclusion

This case contributes to the limited but expanding body of literature indicating that BVCP, although rare, represents an important and potentially persistent manifestation of GBS and its variants. Clinicians should maintain high vigilance for GBS in patients presenting with new-onset BVCP, particularly when there is a recent history of infection or subtle neurological deficits accompanying laryngeal symptoms. In patients with established GBS — especially those with severe bulbar involvement or axonal subtypes associated with anti-GT1a antibodies — careful assessment of the upper airway during ventilator weaning is essential to identify unrecognized BVCP that may hinder successful decannulation. While gradual systemic motor recovery is typical of GBS, the exceptional length and complex reinnervation process of the RLN may predispose patients to long-lasting laryngeal dysfunction. Early recognition of this complication and long-term, multidisciplinary follow-up are crucial for optimizing airway management, functional recovery and quality of life in this small but clinically important subgroup of patients.

Abbreviations

BVCP
bilateral vocal cord paralysis
C. jejuni
Campylobacter jejuni
GBS
Guillain—Barré syndrome
RLN
recurrent laryngeal nerve
SLN
superior laryngeal nerve

Declarations

- Ethics approval and consent to participate: Not applicable
- Clinical trial number: Not applicable
- Consent for publication: Written informed consent has been obtained from the patient.
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- Competing interests: None
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- Authors' contributions: TS was primarily responsible for drafting the manuscript, while KT and MK provided clinical information including examination findings. MK and TM reviewed and edited the manuscript and provided overall supervision.
- Acknowledgements: None
- Author's information: Not applicable

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Figures

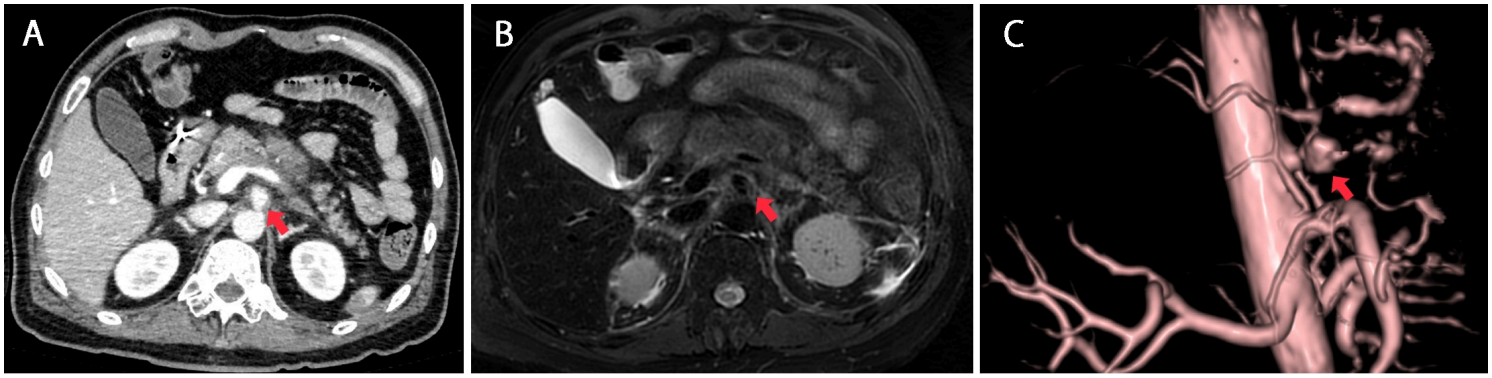


Figure 1

Radiological findings of the celiac artery aneurysm. (A) Contrast-enhanced CT shows narrowing at the origin of the celiac artery, followed by a 13 mm saccular dilatation (arrow). At this stage, the differential diagnosis included a dissecting aneurysm or an infectious aneurysm. (B), (C) Magnetic resonance angiography (MRA) demonstrates an extensive dissection extending from the celiac artery (arrow) to the common hepatic and splenic arteries. The findings are consistent with a thrombosed false-lumen type dissection. The aneurysm was managed conservatively and remained stable.

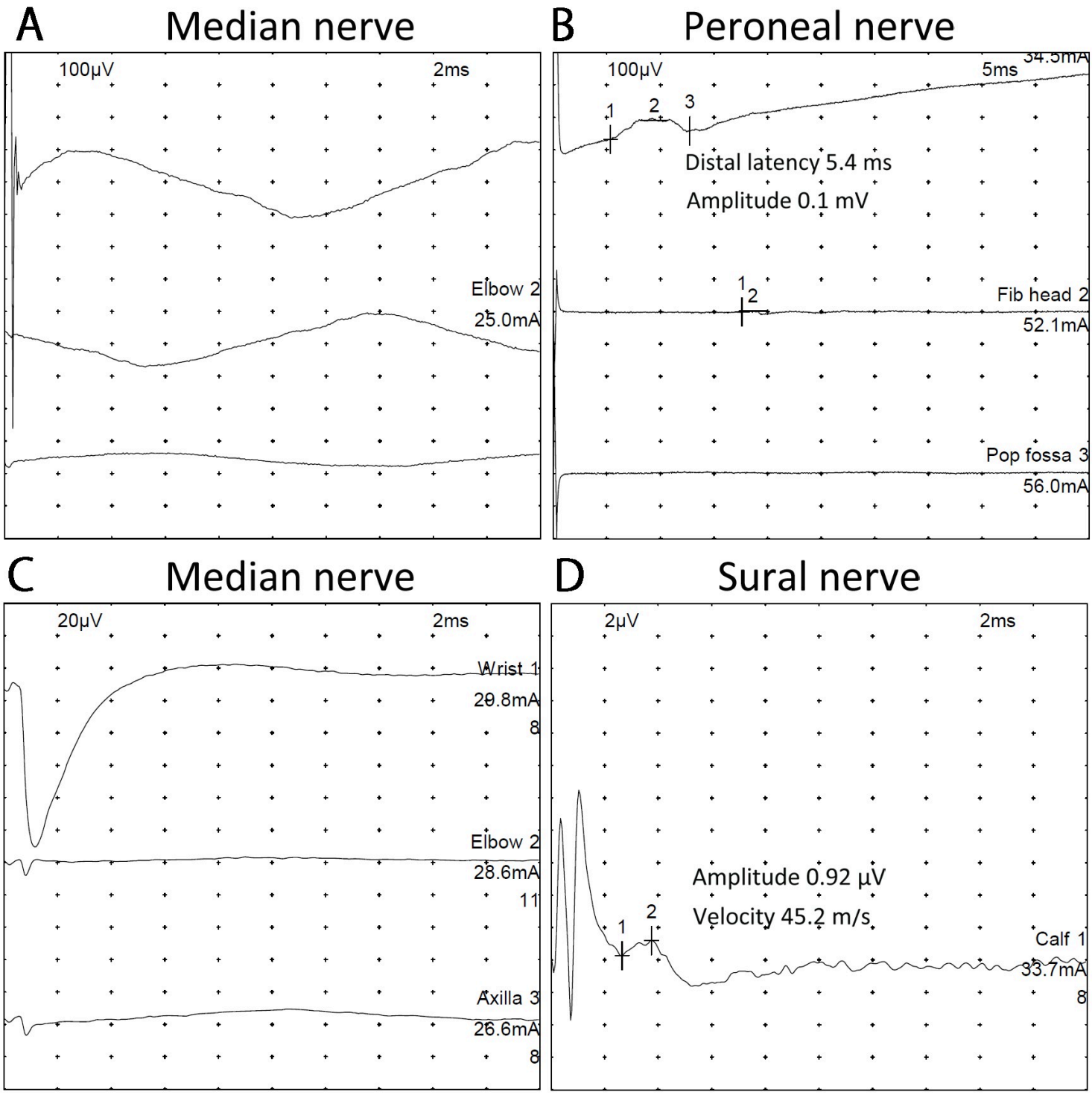


Figure 2

Electrophysiological findings on Day 11. Representative waveforms from motor and sensory nerve conduction studies. (A), (B) Motor nerve conduction studies of the right median and peroneal nerves demonstrate a generalized, profound reduction in compound muscle action potential (CMAP) amplitudes; responses were nearly unelicitable in all limbs tested. F waves were

completely absent. (C), (D) Sensory nerve conduction studies show a marked reduction in sensory nerve action potential (SNAP) amplitudes, with only minimal preservation in the right sural nerve. No definitive demyelinating features, such as prolonged distal latencies or marked slowing of conduction velocities, were observed.

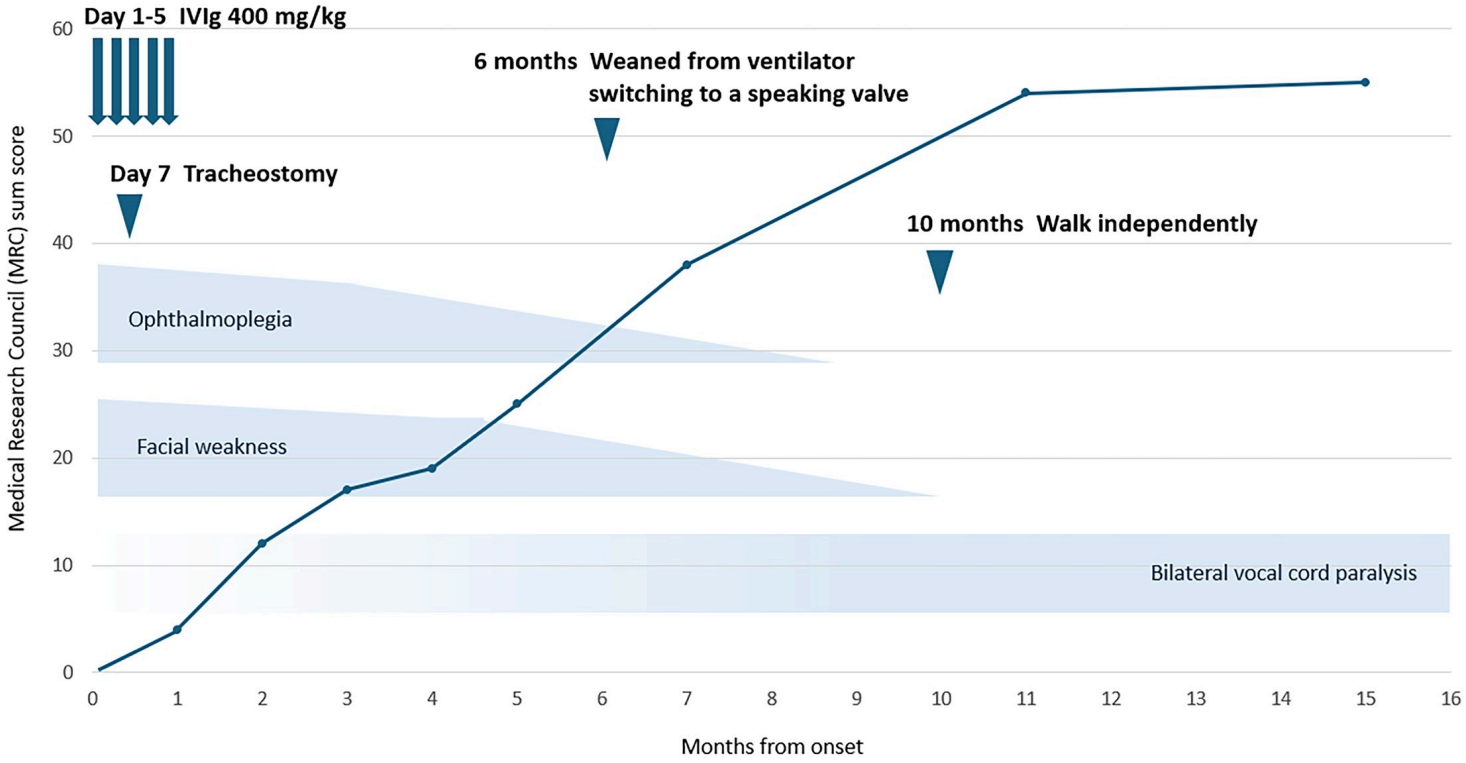


Figure 3

Clinical course and recovery of motor function. The graph illustrates the patient's neurological progression, as measured by the Medical Research Council (MRC) sum score (maximum = 60 across 12 muscle groups). IVIg therapy was initiated immediately after admission, but the patient required intubation shortly thereafter, and tracheostomy was performed on Day 7. By that time, multiple cranial nerve symptoms had developed, including ophthalmoplegia and facial weakness; however, assessment of vocal cord mobility was not possible because mechanical ventilation was required for respiratory muscle paralysis. Over time, limb strength and cranial nerve deficits gradually improved, enabling transition to a speaking valve approximately six months after tracheostomy. Bilateral vocal cord paralysis was subsequently identified during daily rehabilitation and recurrent episodes of dyspnea. The patient ultimately regained independent ambulation, but tracheal decannulation proved difficult.

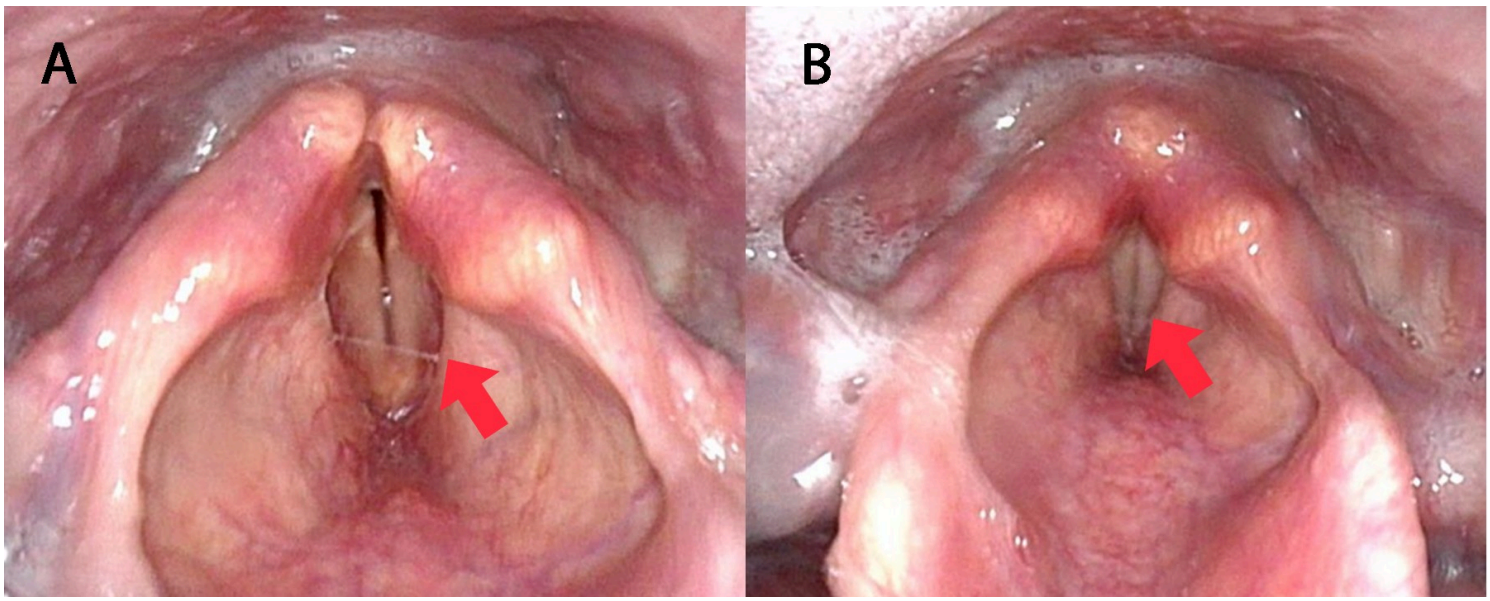


Figure 4

Laryngoscopic findings eleven months after disease onset. Still images from flexible fiberoptic laryngoscopy during (A) inspiration and (B) expiration. The bilateral vocal cords are fixed in the paramedian position (arrows). No abduction of either vocal cord is observed during deep inspiration.

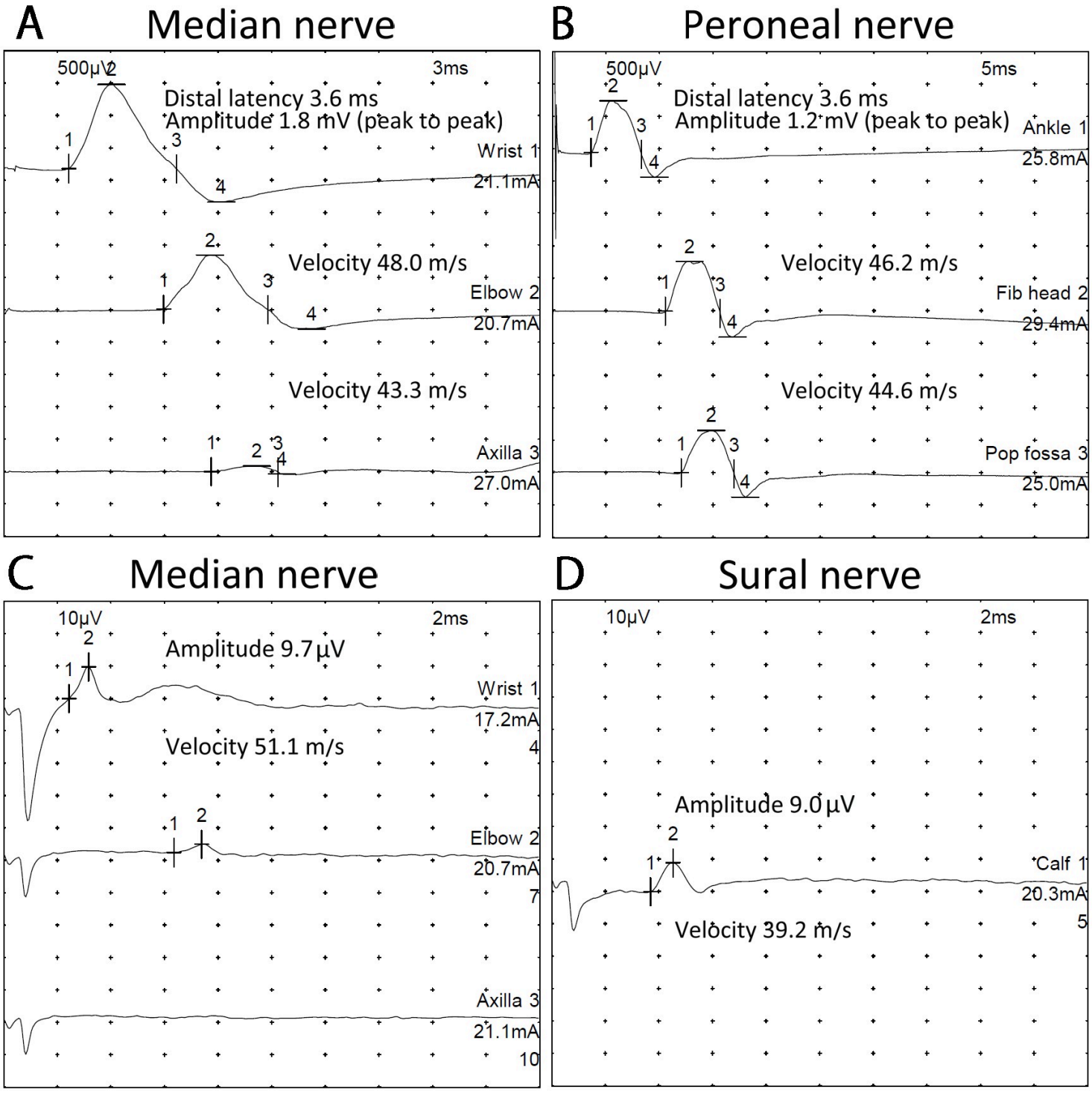


Figure 5

Electrophysiological findings one year after disease onset. Marked improvement is observed in CMAP and SNAP amplitudes in most nerves examined.

Supplementary Files

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