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Guillain-Barré Syndrome (AIDP Variant) Temporally Associated With Purified Vero Cell Rabies Vaccination (Rabivax-S) in a 23-Year-Old Male: A Case Report

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Case Report

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Abstract

Background: Guillain-Barré Syndrome (GBS), particularly the Acute Inflammatory Demyelinating Polyneuropathy (AIDP) variant, is a rare but serious immune-mediated polyradiculoneuropathy. While most cases follow infections, vaccination is a recognized, albeit uncommon, trigger. This report describes a case of AIDP following administration of purified Vero cell rabies vaccine (PVRV, Rabivax-S) and tetanus toxoid as post-exposure prophylaxis, highlighting the need for continued pharmacovigilance with modern cell-culture rabies vaccines in rabies-endemic regions.

Case Presentation: A previously healthy 23-year-old male developed acute ascending flaccid quadriparesis 11 days after receiving the first three doses of purified Vero cell rabies vaccine (Rabivax-S) along with tetanus toxoid following a monkey bite. He presented with severe symmetric weakness (Medical Research Council grade 2/5 in upper limbs, 1/5 in lower limbs), generalized hypotonia, and areflexia, without autonomic instability or respiratory compromise (diaphragmatic excursion ~4 cm). Nerve conduction studies performed on day 6 of weakness revealed markedly reduced compound muscle action potential amplitudes, prolonged distal motor latencies, severely slowed conduction velocities, and conduction block — most strikingly a 99% CMAP amplitude reduction in the right peroneal nerve — with preserved sensory responses, fulfilling electrodiagnostic criteria for AIDP. Cerebrospinal fluid analysis revealed albuminocytologic dissociation (protein 231 mg/dL, 4 cells/μL). The patient received intravenous immunoglobulin (0.4 g/kg/day for 5 days), supportive care, and physiotherapy. After three weeks of hospitalization and six months of rehabilitation, he achieved partial recovery with residual moderate weakness.

Conclusion: This case illustrates a temporal association between purified Vero cell rabies vaccination (administered concurrently with tetanus toxoid) and AIDP. While causality cannot be proven, clinicians should maintain a high index of suspicion for GBS in patients developing acute flaccid paralysis after rabies post-exposure prophylaxis. Early nerve conduction studies and prompt immunomodulatory therapy are essential. The overwhelming life-saving benefits of rabies vaccination continue to far outweigh this rare potential risk.

Introduction

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy with an estimated annual incidence of 1–2 cases per 100,000 population [1]. The acute inflammatory demyelinating polyneuropathy (AIDP) variant is the predominant subtype in sub-Saharan Africa, characterized by progressive symmetric weakness, areflexia, electrodiagnostic evidence of motor demyelination, and CSF albuminocytologic dissociation [1, 2]. Diagnosis is established using Brighton Collaboration Level 1 criteria, which require bilateral flaccid limb weakness, absent or reduced deep tendon reflexes, and either CSF albuminocytologic dissociation or electrodiagnostic evidence of a peripheral neuropathy consistent with GBS [3].

Vaccination is a recognized, albeit rare, precipitant of GBS. The proposed mechanism is molecular mimicry, in which structural similarities between vaccine antigens — particularly glycoproteins — and peripheral nerve gangliosides (GM1, GD1a, GD1b) generate cross-reactive antibodies that target the myelin sheath [4, 5]. Rabies post-exposure prophylaxis (PEP) is a life-saving intervention in endemic regions. Older neural tissue-derived rabies vaccines carried substantial neurological risk due to myelin basic protein contamination [9]. Modern purified Vero cell rabies vaccine (PVRV, Rabivax-S) has eliminated this risk and carries an extremely low incidence of GBS [8, 10]; however, reports of GBS following purified chick embryo cell (PCEC) vaccine — a distinct platform — have been described [11, 12].

We report AIDP fulfilling Brighton Level 1 criteria occurring 11 days after PVRV (Rabivax-S) administered concurrently with tetanus toxoid as rabies PEP following a monkey bite in Ethiopia. This is among the few cases specifically linking Rabivax-S to AIDP, and from sub-Saharan Africa documented with full quantitative electrodiagnostic data, underscoring the need for clinical awareness and pharmacovigilance in resource-limited endemic settings.

Case presentation

A previously healthy 23-year-old Ethiopian male presented to our hospital with a two-day history of rapidly progressive generalized body weakness. The weakness began upon waking, initially affecting the right upper limb proximally and progressing distally within hours. It rapidly spread to the contralateral upper limb and then to both lower limbs in an ascending pattern, reaching peak severity within 24 hours. Eleven days prior to symptom onset, the patient had sustained a deep, bleeding monkey bite to his right knee. He received immediate wound care, tetanus toxoid, and purified Vero cell rabies vaccine (PVRV, Rabivax-S; 0.5 mL intramuscularly) on days 0, 3, and 7 of the Essen regimen. The day-14 dose was not administered due to symptom onset. There was no history of preceding fever, gastrointestinal illness, upper respiratory infection, or any other prodromal illness.

On admission, the patient was hemodynamically stable with no signs of autonomic instability; heart rate variability and blood pressure were within normal limits and there were no urinary retention or fecal incontinence/constipation throughout admission. Single breath count was 20–24, and diaphragmatic excursion measured approximately 4 cm, indicating preserved respiratory function not requiring ventilatory support. The healed bite wound on the right knee showed no vesicular lesions, erythema, or signs of lymphangitis. Systemic examination was otherwise unremarkable.

Neurological examination revealed a fully conscious and oriented patient (Glasgow Coma Scale 15/15). Motor examination demonstrated flaccid quadriparesis with symmetric severe weakness: Medical Research Council (MRC) grade 2/5 in upper extremities and 1/5 in lower extremities. Generalized hypotonia was present. Deep tendon reflexes were absent throughout all four limbs, with mute plantar responses bilaterally. Sensation to light touch, pinprick, vibration, and joint position sense was fully preserved in all four limbs. No cranial nerve deficits, cerebellar signs, or meningeal signs were detected.

Nerve conduction studies (NCS) were performed on the sixth day of weakness and interpreted using established electrodiagnostic criteria for GBS subtype classification [13, 14, 15]. Raw NCS values are summarized in Table 1. Motor NCS (Fig. 1) demonstrated markedly reduced compound muscle action potential (CMAP) amplitudes, prolonged distal motor latencies (DML), and severely slowed motor conduction velocities (CV) in all tested nerves, with greater severity in the lower limbs. Conduction block was documented in the right ulnar nerve (54% CMAP amplitude reduction from wrist to elbow) and was most pronounced in the right peroneal nerve, where a 99% CMAP amplitude drop from ankle (3.7 mV) to knee (0.05 mV) was recorded — one of the most striking indicators of demyelinating conduction block in this case. Temporal dispersion was also observed. F-wave latencies were prolonged in the lower limbs, supporting proximal root involvement. Sensory NCS (Fig. 2) showed fully preserved sensory nerve action potential (SNAP) amplitudes and sensory CV across all tested nerves — bilateral median, right ulnar, and right sural — consistent with a classic sural-sparing pattern. This combination of motor demyelinating features with preserved sensory responses fulfils electrodiagnostic criteria for AIDP [13, 14].

Table 1

A. Motor nerve conduction studies Normal reference values: DML < 4.4 ms (median/ulnar), < 6.5 ms (peroneal), < 6.1 ms (tibial); CV > 49 m/s (upper limb), > 40 m/s (lower limb); CMAP amplitude: median > 4 mV, ulnar > 3 mV, peroneal > 2 mV, tibial > 4 mV.

Source: Preston & Shapiro, 2021.

Nerve	Side	Site	DML (ms)	CMAP (mV)	CV (m/s)	Conduction block	F-wave mean (ms)
Median	Right	Wrist	2.92	1.4	43	No	24.0
		Elbow	—	0.9			
Ulnar	Right	Wrist	1.98	1.3	32	Yes (54% drop wrist→elbow)	—
		Elbow	—	0.6			
		Above elbow	—	0.23			
Median	Left	Wrist	3.44	2.9	43	No	24.0
		Elbow	—	0.9			
Peroneal	Right	Ankle	5.52	3.7	22	Yes (99% drop ankle →knee)	31.0
		knee	—	0.05			
		Above knee	—	0.004			
Tibial	Right	Ankle	9.17	0.7	25	No	31.0
		Knee	—	0.8			
Tibial	Left	Ankle	8.02	1.1	29	No	31.0
		Knee	—	0.4			

DML = distal motor latency; CMAP = compound muscle action potential; CV = conduction velocity. DML measured at distal site only. Abnormal values in red; conduction block defined as > 50% CMAP amplitude drop between adjacent sites.

Table 1
B. Sensory nerve conduction studies

Nerve	Side	Site	SNAP (μV)	Sensory CV (m/s)	Interpretation
Median	Right	Wrist	35.3	66	Normal
Ulnar	Right	Wrist	31.3	57	Normal
Sural	Right	Mid-calf	145.1	55	Normal (sural sparing)
Median	Left	Wrist	31.6	64	Normal

SNAP = sensory nerve action potential; CV = conduction velocity. Normal values in green. All sensory responses preserved, confirming sural-sparing pattern characteristic of AIDP.

Cerebrospinal fluid (CSF) analysis performed on day 11 of weakness demonstrated classic albuminocytologic dissociation: protein 231 mg/dL with a white blood cell count of 4 cells/μL. Routine laboratory investigations, including complete blood count, renal and hepatic function tests, serum electrolytes, and blood glucose, were all within normal limits. Serology for common antecedent pathogens was not performed due to logistical constraints in our resource-limited setting.

Differential diagnoses were carefully considered given the monkey bite exposure. Herpes B virus (Cercopithecine herpesvirus 1) infection typically presents with local vesicular lesions, severe ascending myelitis, or encephalitic features; the absence of vesicular changes at the healed wound site, intact sensation, and the absence of encephalopathy made this diagnosis unlikely. Paralytic rabies was considered but excluded on the basis of preserved sensation, the absence of progressive encephalopathy or hydrophobia, and eventual partial recovery with immunotherapy — all features inconsistent with fatal rabies. Acute disseminated encephalomyelitis (ADEM) was not supported clinically given the intact level of consciousness, absence of seizures, and absent upper motor neuron signs; neuroimaging was therefore not pursued. These exclusions, together with the characteristic NCS and CSF findings, supported a diagnosis of AIDP temporally associated with rabies PEP.

The diagnosis of Guillain-Barré syndrome (AIDP variant) fulfilling Brighton Collaboration Level 1 criteria [3] was established. The patient was treated with a five-day course of intravenous immunoglobulin (IVIG; 0.4 g/kg/day), subcutaneous heparin for venous thromboprophylaxis, pregabalin for neuropathic pain, and intensive inpatient physiotherapy. He was hospitalized for three weeks and discharged with continuation of outpatient rehabilitation.

At six-month follow-up, he showed gradual but incomplete recovery with persistent moderate weakness (MRC grade 3/5 upper limbs, 2/5 lower limbs) and reduced deep tendon reflexes (+ 1/4).

Discussion

We report Guillain-Barré syndrome (AIDP variant) in a 23-year-old male occurring 11 days after PVRV (Rabivax-S) and tetanus toxoid administered as rabies PEP. The diagnosis fulfilled Brighton Collaboration

Level 1 criteria [3, 22].

The 11-day post-vaccination interval is consistent with the recognized 1–6 week window for vaccine-associated GBS [7], reflecting the time required for adaptive immune activation and effector antibody generation. The absence of any prodromal infection, a normal peripheral blood count, and the temporal proximity to vaccination strengthen the association, while coincidence cannot be excluded given the background incidence of GBS. Both PVRV and tetanus toxoid have been independently implicated as rare GBS triggers. The most plausible mechanism is molecular mimicry: the inactivated rabies glycoprotein and tetanus toxoid fragments share structural motifs with peripheral nerve gangliosides (GM1, GD1a, GD1b), inducing cross-reactive antibodies that target the myelin sheath [4, 5]. Residual Vero cell proteins in the vaccine preparation may further contribute through bystander immune activation [4]. Attribution to one vaccine over the other is not possible from this single case; however, the PVRV-specific literature — particularly Kulkarni et al. [10] — supports rabies vaccine as a plausible trigger.

The unique clinical context of monkey bite exposure required active exclusion of Herpes B virus (Cercopithecine herpesvirus 1) infection and paralytic rabies. Herpes B typically produces ascending myeloencephalitis with local vesicular lesions [18]; neither was present. Paralytic rabies can mimic GBS with ascending flaccid weakness, but invariably progresses to fatal encephalopathy without response to immunotherapy [19]. The preserved sensation, intact consciousness, characteristic NCS pattern, and partial recovery with IVIG collectively exclude rabies. ADEM was excluded by the absence of upper motor neuron signs, intact consciousness, and the purely peripheral demyelination pattern on NCS [20]. These exclusions are particularly relevant in resource-limited settings where diagnostic tests for these conditions are unavailable.

Treatment with IVIG (0.4 g/kg/day for 5 days) is consistent with established first-line therapy [16]. Partial recovery at six months — MRC grade 3/5 upper limbs, 2/5 lower limbs — is within the range of known GBS outcomes; approximately 20% of patients remain significantly disabled at six months, particularly when severe conduction block causes secondary axonal degeneration [1, 16]. Serial NCS would have been valuable to track axonal injury progression, but was not feasible in our setting [21].

This report adds to the sparse literature on GBS following Rabivax-S/PVRV, particularly in sub-Saharan Africa. It highlights the need for clinicians in rabies-endemic regions to consider GBS when acute flaccid paralysis develops after PEP, to perform early NCS for electrodiagnostic subtype classification, and to initiate IVIG promptly. The life-saving benefit of rabies PEP overwhelmingly outweighs this rare risk, but systematic pharmacovigilance reporting of neurological adverse events following rabies vaccination in endemic regions is essential to better characterize this association.

Limitations

This case report has several inherent limitations. As a single case, it can only demonstrate a temporal association between the administration of purified Vero cell rabies vaccine (PVRV, Rabivax-S) concurrently with tetanus toxoid and the development of AIDP, rather than proving causality for either

vaccine. Given the background incidence of GBS (1–2 per 100,000 population annually), coincidence cannot be entirely excluded. Both rabies vaccines and tetanus toxoid have been rarely associated with GBS in the literature, making it challenging to attribute the syndrome to one agent over the other or to their combined administration.

The diagnostic evaluation, while appropriate in a resource-limited setting, did not include advanced immunological testing such as anti-ganglioside antibody profiling (e.g., anti-GM1, anti-GD1a) or specific assays to support molecular mimicry. Such tests could have further strengthened the pathophysiological link. Additionally, we could not definitively rule out subclinical infection related to the monkey bite (e.g., other simian viruses) as a potential trigger, since serological testing for common antecedent pathogens (Campylobacter jejuni, cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae) was similarly not available, and a subclinical antecedent infection cannot be definitively excluded.

Only a single nerve conduction study was conducted; serial electrophysiological assessments would have been valuable to evaluate for secondary axonal degeneration and provide more robust prognostic information. Neuroimaging was not pursued in the absence of clinical features suggestive of central nervous system involvement. The six-month follow-up period, although useful, is relatively short to fully capture long-term functional outcomes, residual disability, or quality-of-life impact commonly seen in GBS survivors. Finally, as this report describes a young, previously healthy male, the findings may not be generalizable to other age groups, comorbidities, or populations with different genetic backgrounds.

Despite these limitations, the comprehensive clinical, electrophysiological, and cerebrospinal fluid documentation, along with careful exclusion of key differential diagnoses, provides meaningful insight into this rare association in a rabies-endemic setting.

Conclusion

In conclusion, we report a case of acute inflammatory demyelinating polyradiculoneuropathy (AIDP) temporally associated with purified Vero cell rabies vaccine (PVRV, Rabivax-S) administered concurrently with tetanus toxoid as post-exposure prophylaxis following a monkey bite. While a definitive causal relationship cannot be established from a single case, the close temporal proximity, absence of other identifiable infectious triggers, and characteristic clinical, electrophysiological, and cerebrospinal fluid findings support a possible immune-mediated link.

Clinicians practicing in rabies-endemic regions should maintain a high index of suspicion for Guillain-Barré syndrome in patients who develop acute flaccid paralysis following rabies post-exposure prophylaxis. Early performance of nerve conduction studies and cerebrospinal fluid analysis is essential for timely diagnosis, while prompt initiation of intravenous immunoglobulin therapy can improve outcomes. This rare potential adverse event must be carefully weighed against the well-established, life-saving benefits of rabies vaccination and post-exposure prophylaxis. Continued pharmacovigilance with modern cell-culture rabies vaccines remains important.

Declarations

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal. Not applicable. This is a case report describing standard clinical care.

Acknowledgement

None.

Ethics approval and consent to participate

Not applicable. No experimental intervention or additional procedures beyond routine management were performed.

Human Ethics and Consent to Participate declarations: not applicable

Competing interests

The authors have no conflicts of interest to declare. No financial or personal relationships influenced this work.

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Reshid Yimam Nega: Formal analysis, Supervision, Validation, Writing – review & editing.

All authors approved the final manuscript.

Data Availability Statement

The data that support the findings of this case report are not publicly available due to privacy and ethical

restrictions but are available from the corresponding author upon reasonable request.

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Figures

Motor Nerve Studies

Nerve: Median-Rt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	2.92	14.06	1.4 mV	43.48
2:Elbow	7.29	11.98	0.9 mV	

Nerve: Ulnar-Rt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	1.98	13.44	1.3 mV	34.90
2:Elbow	7.71	17.29	0.6 mV	32.05
3:Above Elbow	10.83	10.00	231.2 µV	

Nerve: Median-Lt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Wrist	3.44	15.00	2.9 mV	43.27
2:Elbow	7.60	19.90	0.9 mV	

Nerve: Peroneal-Rt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Ankle	5.52	17.60	3.7 mV	22.16
2:Knee	19.06	7.60	50.5 µV	7.74
3:Above Knee	31.98	8.02	3.5 µV	

Nerve: Tibial-Rt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Ankle	9.17	13.65	0.7 mV	25.11
2:Knee	25.10	14.38	0.8 mV	

Nerve: Tibial-Lt

Site	Lat1 (mS)	Dur (mS)	Amp	NCV (m/S)
1:Ankle	8.02	12.81	1.1 mV	29.09
2:Knee	21.77	11.46	0.4 mV	

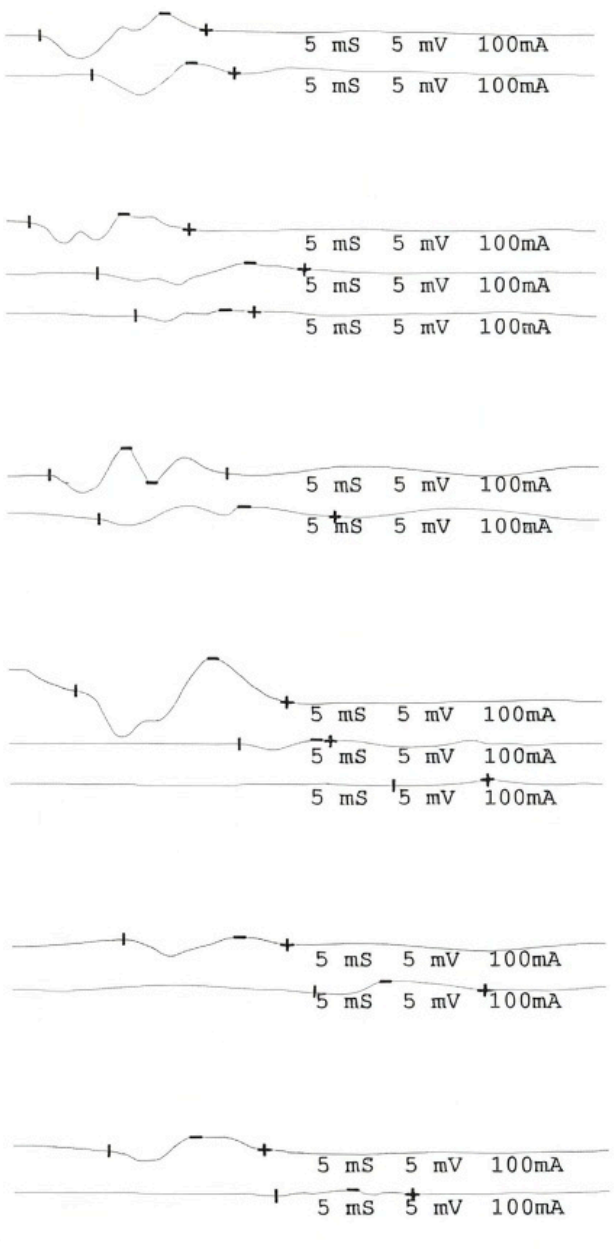


Figure 1

Motor nerve conduction studies of the bilateral median and ulnar nerves (upper extremities) and right peroneal and bilateral tibial nerves (lower extremities), demonstrating reduced compound muscle action potential (CMAP) amplitudes, markedly prolonged distal motor latencies, severely slowed conduction velocities, conduction block (including a 99% CMAP amplitude reduction in the right peroneal nerve between ankle and knee), temporal dispersion, and prolonged F-wave latencies — findings consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP).

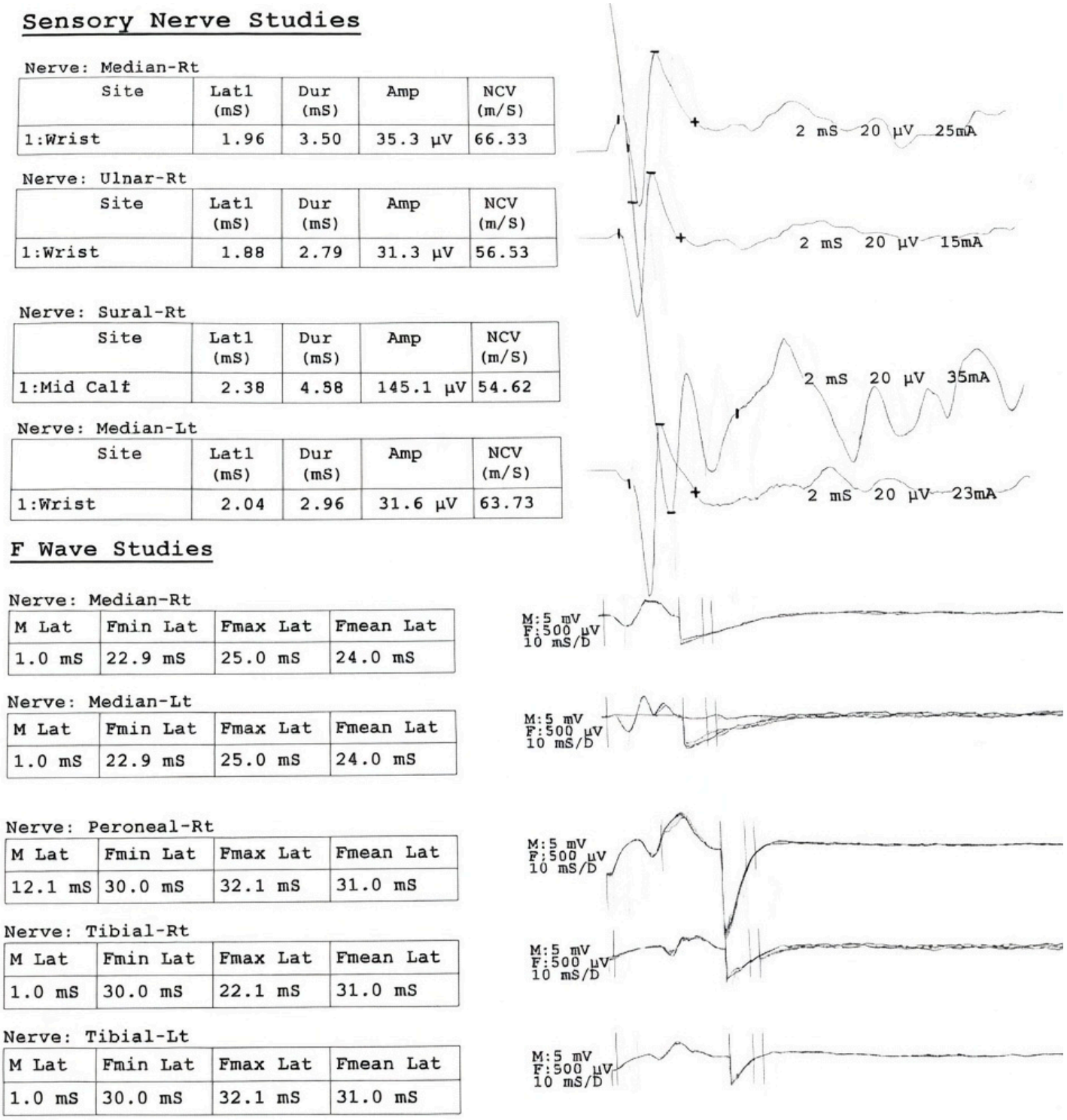


Figure 2

Sensory nerve conduction studies of the bilateral median and ulnar nerves (upper extremities) and right sural nerve (lower extremity), demonstrating preserved sensory nerve action potential (SNAP) amplitudes and conduction velocities across all tested nerves, consistent with a sural-sparing pattern characteristic of AIDP. F-wave studies are also shown for bilateral median, right peroneal, and bilateral tibial nerves.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- CAREChecklist.pdf