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Research paper

Reversible conduction failure on the deep tendon reflex response recording in early Guillain-Barré syndrome

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ABSTRACT

Objective: To describe the case of a patient with Guillain-Barré syndrome (GBS) showing early reversible conduction failure (RCF) detected by means of serial deep tendon reflex response (T-reflex) study.
Methods: A 36-year-old woman had a 5-day history of foot and hand paresthesias ascending to thighs and arms, throbbing interscapular and neck pain, mild to moderate tetraparesis, and areflexia. Nerve conduction studies (NCS) were performed on days 7 and 33 after onset.
Results: NCS showed an equivocal electrophysiologic pattern, just an isolated distal RCF being detected on the right radial nerve at initial examination. Motor latency on deltoid muscle after Erb's point stimulation was preserved. Sensory conduction velocities were normal or slightly slowed. Somatosensory evoked potentials from median and tibial nerves were normal. Initially, F-wave study demonstrated reversible abnormalities, consisting of multiple A waves and low F-wave persistence, minimal F-wave latencies being preserved. Biceps brachii T-reflex was normal, whereas Achilles T-reflex was absent bilaterally, appearing on the second study with normal T-wave morphology and latency, thus conforming to the requirements for RCF diagnosis. Soleus H-reflex was also initially absent.
Conclusions: Serial T-reflex study is a useful technique for detecting early RCF of proximal nerve trunks in early GBS.
Significance: T-reflex is useful tool for GBS in association with NCS.

1. Introduction

Guillain-Barré syndrome (GBS) is an acute-onset, immune-mediated disorder of the peripheral nervous system, which is currently divided into several subtypes based on electrodiagnostic, pathologic and immunological criteria (Hughes and Cornblath, 2005; Van den Berg et al., 2014; Wakerley et al., 2014). GBS includes at least three disease patterns: acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal and acute motor-sensory axonal neuropathy (AMAN and AMSAN), and Fisher syndrome (Griffin et al., 1996).

Electrophysiology plays a determinant role in Guillain-Barré syndrome (GBS) diagnosis, classification of the subtypes and in establishing prognosis (Uncini et al., 2012, 2017; Rajabally et al., 2015). The recent recognition of reversible conduction failure (RCF) in GBS has been of enormous importance to avoid fallaciously classifying AMAN with RCF as AIDP or AMAN/AMSAN with axonal degeneration (Uncini et al., 2013, 2018). RCF implies transient conduction block/slowing in proximal, intermediate or distal nerve segments mimicking demyelination, but without the development of abnormal temporal dispersion. RCF is an "a posteriori"

Abbreviations: AIDP, acute inflammatory demyelinating polyneuropathy; AH, abductor hallucis; ADM, abductor digiti minimi; AMAN, acute motor axonal neuropathy; AMSAN, acute motor sensory axonal neuropathy; APB, abductor pollicis brevis; CIDP, chronic idiopathic demyelinating polyneuropathy; CMAP, compound muscle action potential; CMT1A, Charcot-Marie-Tooth disease type 1A; DML, distal motor latency; EDB, extensor digitorum brevis; EDC, extensor digitorum communis; EMG, electromyography; GBS, Guillain-Barré syndrome; LLN, lower limit of normal; MCV, motor conduction velocity; MRC, Medical Research Council; NCS, nerve conduction study; RCF, reversible conduction failure; SCV, sensory conduction velocity; SEP, somatosensory evoked potentials; SNAP, sensory nerve action potential; TA, tibialis anterior; ULN, upper limit of normal.

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diagnosis as can be recognized only by serial conduction studies of motor or sensory nerves (Uncini and Vallat, 2018).

Measurement of electromyographic deep tendon reflex responses (T-reflex) is a simple, painless, and accurate tool for the assessment of proximal conduction (Péréon et al., 2004), which has been applied primarily to the investigation of radicular syndromes (Schott and Koenig, 1991; Miller et al., 1999). As stated by Kuruoglu and Oh (1994), though the H-reflex and T reflex have the same afferent and efferent pathways, there is a difference in the activation site of stimulation: the H-reflex stimulates directly the Ia fibers, bypassing the muscle spindle organs, while the T-reflex activates the muscle spindle organs. In comparison with H-reflex, T-reflex is more rapidly elicited, more accurate, and allows the examiner to explore more root different segmental levels. Therefore, we have added it to our electrophysiologic protocol (García et al., 2009, 2015).

T-reflex latency testing is a useful indicator of the presence of a demyelinating peripheral neuropathy both in chronic idiopathic demyelinating polyneuropathy (CIDP) and Charcot-Marie-Tooth disease type 1A (CMT1A) (Kuruoglu and Oh, 1994; García et al., 2015). Intriguingly, T-reflex latencies were prolonged in the majority of CIDP patients, even in those showing well-preserved clinical reflexes. Furthermore, biceps T-reflex latencies were markedly prolonged in CMT1A patients, who exhibited either abolition or preservation of clinical reflexes.

Here we describe the first GBS study showing early RCF detected by means of serial T-reflex study.

2. Methods

2.1. Patient

This study is based upon serial clinical and electrophysiological evaluation of a GBS patient. Informed consent was obtained from the patient according to the declaration of Helsinki. The study was approved by the Ethical Committee of the University Hospital "Marqués de Valdecilla".

2.2. Electrophysiological techniques

Electrophysiological studies were carried out using a Synergy EMG system (Oxford Instruments, UK) as reported elsewhere (García et al., 2009, 2015; Gallardo et al., 2015). Recordings were performed by standard methods using surface stimulating and recording electrodes. Skin temperature was maintained at 32–34 °C using infrared heating.

We studied motor conduction velocities (MCV) and distal motor latencies (DML) of the radial, median, ulnar, tibial, and peroneal nerves, bilaterally. MCV measurement of the median and ulnar nerves was carried out by stimulation at the elbow and at the wrist while recording the compound muscle action potentials (CMAPs) over abductor pollicis brevis (APB) and abductor digiti minimi (ADM), respectively. Radial nerve was stimulated at elbow and axilla while recording the CMAP over extensor digitorum communis muscle (EDC). In the same way, MCV of the peroneal and tibial nerves was assessed by stimulation at the knee and ankle while recording the CMAP over extensor digitorum brevis (EDB) and abductor hallucis (AH) muscles, respectively. Minimal F-wave latencies were recorded from APB, ADM, EDB and AH muscles after stimulation at the wrist and ankle; for the identification of A-waves we followed the criteria reported by Kornhuber et al. (1999). H-reflex was recorded over soleus muscle after tibial nerve stimulation at the popliteal fossa. The brachial plexus was stimulated at the Erb's point with recording from deltoid muscle.

Sensory conduction velocity (SCV) of the median nerve was determined from digit III to wrist. Antidromic SCV of the radial and sural nerves was obtained from wrist to forearm and from ankle to midcalf, respectively.

Electromyogram (EMG) was recorded with concentric needle electrodes from right deltoid, EDC and tibialis anterior (TA) muscles. We analyzed the duration and morphology of the motor units, the presence of spontaneous activity, and the EMG pattern at maximum voluntary effort.

Bilateral T-reflex responses from biceps brachii and soleus muscles were evoked with a manually operated trigger hammer using a piezoelectric element to allow fast, accurate triggering of the sweep. T-waves were recorded with surface electrodes. Latencies were measured from the start of the sweep to the onset of the first deflection from the baseline caused by the reflex action potential. Subject positions, joints, and electrode positions were standardized following previously published norms (Stam and van Crevel, 1989). In brief, the biceps brachii muscle (biceps T-reflex) was stretched with the patient lying supine, elbow joint at 90°, the active electrode placed on the muscle belly, and the reference electrode fixed 3–5 cm proximally. The triceps surae muscle (soleus or Achilles T-reflex) was stretched with the patient lying prone, ankle at 90°, the active electrode was placed in line with the Achilles tendon at half the distance between the popliteal fossa crease and the ankle, and the reference electrodes were fixed 3–5 cm distally. The ground electrode was placed in the same extremity. To avoid reflex fatigue and conditioning of subjects, 5–10 reflexes were examined at different time intervals (>5 s) between taps in each subject. The sweep display was 10 ms/division. Sensitivity was adjusted according to the amplitude of the response. Filter settings were 2 Hz–5 kHz. T-reflex latencies were compared with the results of our healthy adult control group (García et al., 2015) or those reported by Kuruoglu and Oh (1994).

Only reproducible T-waves over 0.1 mV amplitude from the baseline to the negative peak were considered. We specifically analyzed the morphology of T-waves, which are biphasic for the biceps T-reflex and bi- or triphasic for the Achilles T-reflex (Péréon et al., 2004).

Somatosensory evoked potentials (SEP) from median and tibial nerves were performed according to standard methods (Chiappa, 1997).

3. Results

3.1. Case report

This 46-year-old woman was admitted with a 5-day history of foot and hand paresthesias ascending to thighs and arms, throbbing interscapular and neck pain, and upper- and lower-limb weakness. There was no antecedent of recent infection. On initial examination, there was mild tetraparesis (MRC grade 4/5), which was slightly more prominent in right hand extensors. She was able to walk without assistance. Triceps, knee and ankle jerks were absent. Sensory examination was normal. Two days later, there was progression of her tetraparesis (MRC 3–4/5) with generalized areflexia. There was no cranial nerve weakness, and her vital signs were normal. Treatment with intravenous immunoglobulin (2 g/kg) was administered with rapid improvement. Ten days after admission examination revealed just slight weakness of right wrist/finger extensors and shoulder girdle abductors, muscle stretch reflexes being present except for right ankle jerk. Re-examination a year later was entirely normal.

Routine laboratory investigations including tests for porphyrin, Lyme disease, HIV, autoantibodies and tumoral markers did not reveal any abnormalities. No serum IgG anti-ganglioside antibodies

were detected. On day 6 after onset, cerebrospinal fluid showed normal results. Stool culture revealed no pathogenic microorganisms, particularly *Campylobacter jejuni*.

3.2. Electrophysiological features

The results of two serial nerve conduction studies (NCS) are summarized in Table 1. On the first NCS (day 7 after onset), right radial nerve recordings showed mild amplitude reduction of CMAP on EDC muscle (1.9 mV *vs* 4.6 mV on the left side), with no variation on stimulation from elbow and axilla. MCV, DML and CMAP amplitudes of median (bilaterally), ulnar (bilaterally), peroneal (bilaterally), right tibial and left radial nerves were normal. SCVs and SNAPs of sural, median, ulnar and right radial nerves were normal. Temporal dispersion of CMAP/SNAP was not observed. Motor latency and CMAP amplitude on right deltoid muscle after Erb's point stimulation were preserved (3.5 ms; 6.0 mV). Median and ulnar nerve F-waves showed low persistence (see Table 1), preservation of minimal latency, and multiple A-waves in tibial nerve (Fig. 1A). Latency and morphology of biceps brachii T-reflex were preserved on both sides, whereas Achilles T-reflex was absent bilaterally (Table 1 and Fig. 1C, E). Right soleus H-reflex was absent. SEPs from median and tibial nerves were bilaterally normal. EMG of EDC revealed a decrease of recruitment pattern with no spontaneous activity; EMG of TA and deltoid was normal.

On the second electrophysiological evaluation (day 33), there was complete normalization of previous abnormalities (Table 1; Fig. 1B, D, F), the only new and positive features being minimal SCV decrease of right median and ulnar nerves with preserved SNAP morphology and amplitude (Table 1). Note that Achilles T-reflex now appeared with normal T-wave morphology and latency (Fig. 1D, F).

Table 1
Results of nerve conduction studies.[a]

	Day 7	Day 33	Normal
R Median nerve			
DML (ms)	4.1	4.1	≤4.4
MCV (m/s)	55.4	53.1	≥49.0
CMAP (mV)	5.7	6.9	≥4.0
F wave (ms)	30.7[b]	28.0	≤31.0
SCV (m/s)	45.2	40.5	≥45.0
SNAP (μV)	6.6	4.8	≥4.0
R Ulnar nerve			
DML (ms)	2.6	3.1	≤3.3
MCV (m/s)	64.6	56.8	≥49.0
CMAP (mV)	7.8	10.1	≥6.0
F wave (ms)	25.8[b]	28.9	≤32.0
SCV (m/s)	57.8	43.9	≥45.0
SNAP (μV)	6.4	4.6	≥3.0
R Radial nerve			
DML (ms)	3.5	3.0	≤4.0
CMAP (mV)	1.9	6.2	≥4.0
MCV (m/s)	60.0	57.1	≥49.0
SCV (m/s)	58.0	50.3	≥56.3
SNAP (μV)	16.4	12.0	≥10.0
R Peroneal nerve			
DML (ms)	4.2	3.4	≤5.5
MCV (m/s)	50.4	51.6	≥44.0
CMAP (mV)	8.8	7.2	≥2.0
F wave (ms)	45.7	43.3	≤56.0
R Tibial nerve			
DML (ms)	4.5	4.3	≤5.8
MCV (m/s)	42.5	45.6	≥41.0
CMAP (mV)	10.1	10.2	≥4.0
F wave (ms)	48.3[c]	46.1	≤56.0
R Sural nerve			
SCV (m/s)	56.1	54.2	≥40.0
SNAP (μV)	31.6	28.8	≥6.0
R Biceps brachii T-reflex			
Latency (m/s)	13.2	13.6	≤14.6[d]
L Biceps brachii T-reflex			
Latency (m/s)	13.9	13.5	≤14.6[d]
R Achilles T-reflex			
Latency (m/s)	A	34.6	≤38.0[e]
L Achilles T-reflex			
Latency (m/s)	A	32.9	≤38.0[e]
R Soleus H-reflex			
Latency (m/s)	A	31.0	≤32.0

Underlined indicates abnormal values.
A = absent; L = left; R = right; NV = not valuable; for other abbreviations, see text.
[a] To shorten the table content, right nerve conduction parameters are only indicated.
[b] Low F-wave persistence (to around 40%) with further normalization in the next electrophysiology.
[c] Multiple A-waves (see Fig. 1A).
[d] In accordance with García et al. (2015).
[e] In accordance with Kuruoglu and Oh (1994).

4. Discussion

Acute sensorimotor clinical features of the current patient strongly support the diagnosis of GBS; weakness may be mildly asymmetrical, as in the present case (Asbury and Cornblath, 1990). Furthermore, this case fulfils the Brighton criteria for GBS with a level 2 of diagnostic certainty (Sejvar et al., 2011; Fokke et al., 2014). In any case, given the mild degree of the deficit and rapid response to IVIG therapy, it is not possible to be definite about the pathological background of the disease.

At first electrophysiological evaluation (day 7), the only valuable positive finding was reduced distal CMAP amplitude of the right radial nerve with preserved MCV, DML and CMAP morphology. Under such premises and using optimized electrophysiologic GBS criteria (Uncini et al., 2017), this patient could be classified within the equivocal group. At second electrophysiology (day 33), there was normalization of right radial nerve CMAP attenuation (see Table 1), thus indicating that retrospectively we were confronted with a focal RCF (Uncini et al., 2013, 2018). It is worth noting that this second electrophysiology showed minimal abnormal signs consisting of borderline SCV reduction of right ulnar and median nerves, which are of doubtful pathogenic value given that by then the patient had exhibited nearly full recovery with normal neurologic examination one year after symptom onset. Be that as it may, upon serial studies the electrodiagnostic classification does not change.

We observed low F-persistence (up to 40%) in median and ulnar nerves with preserved minimal F-wave and in the absence of F-chronodispersion. F-wave abnormalities have been used in GBS diagnosis as follows: i/as a criterion of AIDP, if F-response latency >120% of upper limit of normal (ULN) (Hadden et al., 1998; Uncini et al., 2017); ii/idem, if there is F-wave latency >120% of ULN, or >150% of ULN when distal CMAP <50% of lower limit of normal (LLN) (Rajabally et al., 2015); iii/as a criterion of axonal GBS, if F-wave was absent in one nerve with distal CMAP ≥20% of LLN (Rajabally et al., 2015); and iv/idem, if isolated F-wave absence (or 20% persistence) (Uncini et al., 2017). Normal F-wave persistence varies between subjects, muscles and nerves (Panayiotopoulos and Croni, 1996; Mesrati and Vecchierini, 2004). In upper-limb nerves (median and ulnar) F-wave persistence ranges between 60% and 100%, whereas in lower limb nerves a low persistence of 30% is found for peroneal nerve, but a nearly 100% persistence for tibial nerve. Although low F-wave persistence observed here did not reach 20% required in the proposed GBS criteria (Uncini et al., 2017), upon serial electrophysiological

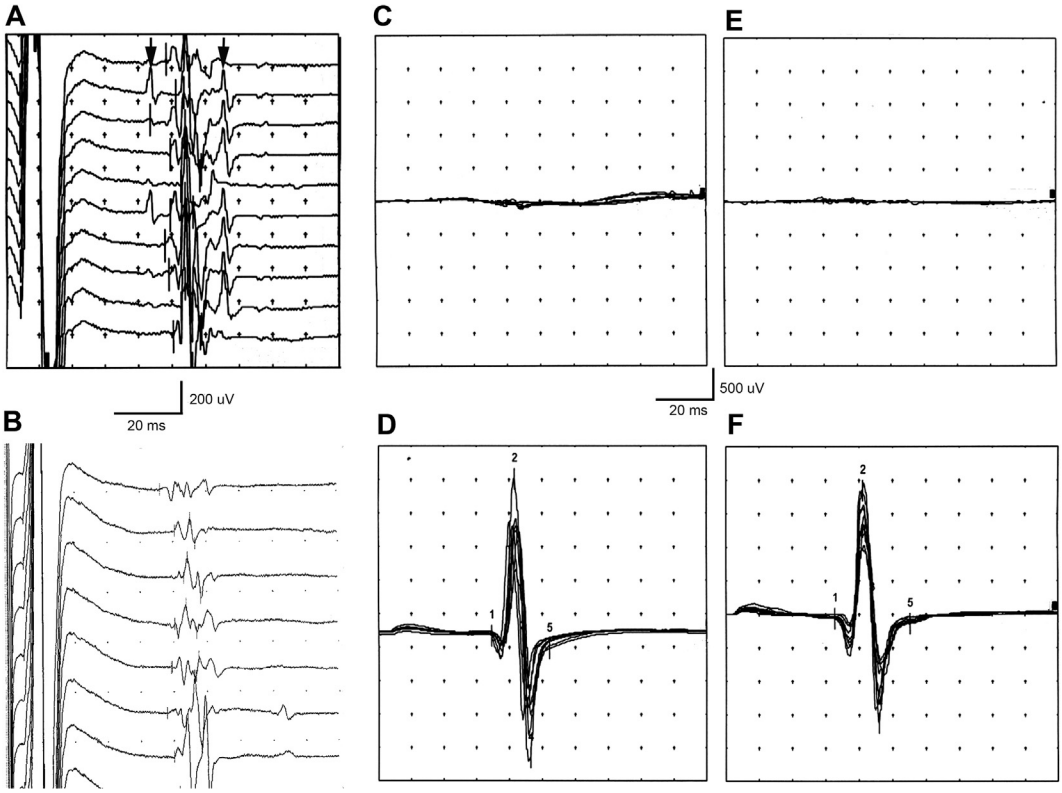

Fig. 1. Two serial recordings of F-waves from right tibial nerve (A, B), and bilateral Achilles T-reflex (C-F). In the first study (day 7 after onset) note normal F-waves with presence of multiple A-waves (supramaximal stimuli), which are observed within and after the range of F-wave latencies (A; arrows); simultaneously, Achilles T-reflex was absent bilaterally (C, E). One month later (lower traces) there were no A-waves (B), and the Achilles T-reflexes were normal (D, F).

recording median and ulnar nerves exhibited almost 100% F-wave persistence. Despite further serial electrophysiological evaluation being needed, we interpret that such low early F-wave persistence is pathogenic pointing to proximal nerve trunk pathology, between the root emergence and the Erb's point, given that motor conduction parameters distal to this point were normal.

The presence of multiple A waves in a patient with neuropathy and no previous history of peripheral nerve disease, as reported here, strongly supports the clinical suspicion of GBS (Kornhuber et al., 1999). We observed that A waves occurred at first evaluation with later disappearance (see Fig. 1A, B). Within weeks 1 and 2 of the clinical course multiple A waves may occur in any GBS subtype (AIDP, non-AIDP, unclassified or AMAN), whereas within 3 and 6 weeks multiple A waves are characteristic of AIDP (Kawakami et al., 2012). So, our observation does not help to clarify the issue.

In the current patient, Achilles T-reflex was bilaterally absent at initial electrophysiological evaluation, four weeks later appearing with normal T-wave morphology and latency. This electrophysiological evolution fits in well with RCF (Uncini et al., 2013, 2018). To the best of our knowledge, there are no previous serial T-reflex studies in early GBS. One might wonder which could be the topography of lesions accounting for these peculiar electrophysiological features. In early GBS, pathological changes usually predominate in proximal nerve trunks, in some studies being more prominent where the spinal roots unite to form the spinal nerves; on very early GBS endoneural inflammatory oedema is the outstanding finding (Haymaker and Kernohan, 1949; Krücke, 1955; Gallardo et al., 2015; Berciano et al., 2017). Accepting that inflammatory oedema in proximal nerve trunks lesions is the pathogenic mechanism altering nerve conduction in very early GBS, this could account for proximal RCF detected by means of isolated F-wave absence (Rajabally et al., 2015; Uncini et al., 2017) or, as describe here, low F-wave persistence and absence of T-reflex. As observed here, absence of Achilles T-reflex combined with preservation of motor and sensory conduction parameters of lower-limb nerves and SEP from tibial nerve could suggest that RCF particularly involves afferent Ia fibers. Selective vulnerability of Ia fibers has been reported in a few neurologic syndromes (Infante et al., 2018). To establish if this is the case in early GBS further serial clinico-electrophysiological studies are needed. Additionally, detection of proximal motor nerve conduction block may require special electrophysiological studies such as root stimulation or triple stimulation technique (Kurt Incesu et al., 2013; Sevy et al., 2018).

5. Conclusion

Together with other NCS, serial T-reflex study may be a useful technique for detecting RCF of proximal nerve trunks in early GBS.

Conflict of interest

None of the authors have potential conflicts of interest to be disclosed.

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