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Case Report

AIDP-SGB in Our Hospital. About a Case. Clinical-Neurophysiological Description of the Process

Angel Molina León*, C Asuncion Sancho and C Tudor Franco

Clinical Neurophysiology Service, University Hospital of Santa Lucia, Cartagena, Murcia, Spain

***Corresponding Author:** Angel Molina Leon, Clinical Neurophysiology, Virgen De la Caridad Medical Center, Cartagena, Murcia, Spain.

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Abstract

AIDP, also known as Guillain-Barre syndrome, is an acute inflammatory polyradiculoneuritis of a demyelinating nature, which may or may not affect axonal fibers, but the etiopathogenesis is demyelinating in nature. It is immune-mediated, rapidly progressive, predominantly motor, often with bulbar and respiratory involvement and occasionally requires intubation and ICU care. It is one of the most common of all neuromuscular emergencies [1], however the prognosis in most cases is favorable, in more than 80% of patients affected by this condition. Nerve conduction studies (ENG) and EMG play an important role in the diagnosis because they are capable of early elucidating the state of the peripheral nervous system and if the condition is axonal, or demyelinating, if it is acute, subacute or chronic. and in this way promptly put the appropriate treatment and minimize the complications that this condition has with it. It is most common in young adults, although it can occur at any age. It is frequent in AIDP, a history between several weeks before respiratory or gastrointestinal infection in approximately 60% of patients and is related to germs of the type: Campylobacter, cytomegalovirus, Epstein-Barr virus, HIV, vaccination, surgery, trauma, and paraneoplastic syndromes (especially Lymphoma) [1,2].

Keywords: *AIDP; Demyelinating Lesion; Motor Conduction Block; Autoimmune Attack; Ataxia; Areflexia*

Introduction to the Clinical Case

The incidence of GBS has been estimated between 0.81 and 1.89 per 100,000 inhabitants in the adult population and between 0.34 and 1.34/100,000 inhabitants in children. With important geographic variations, in relation to the SGB variants (AIDP, AMAM, AMNSA, SMF) [3-5].

The progression and speed of progression of symptoms is variable, 30% of patients require intubation and transfer to intensive care units for monitoring. 14% are left with sequelae [3,5,20]. Among the clinical factors linked to an unfavorable evolution include: Age, older than 50 years, previous diarrhea, short time between the onset of symptoms and hospitalization, the presence or not of associated vegetative nervous system dysfunction, the need or not of early mechanical ventilation, weakness of the neck flexor muscles, marked muscle weakness and a Hughes criteria greater than 3 [6].

Electrophysiological factors associated with poor functional prognosis AND or late recovery include: Low amplitude of the CMAP and inexitability of the motor nerves [7,8]. Neurography and electromyography (EMG) is one of the most important diagnostic tools for confirming the diagnosis, determining GBS variants, and establishing a functional prognosis [1,9,20].

Our clinical case is a 43-year-old male patient who consulted for limb weakness. He is admitted on 01.18.21, the clinic begins 3 days before admission, January 15, 2021. Two nights prior to admission, the patient gets up at dawn and notices clumsiness and claudication of lower limbs, maintaining ambulation. Later he lies down and wakes up with motor clumsiness and left upper limb weakness. and later right upper limb, with falling objects and difficulty grasping. Later he also begins with weakness of the lower right limb. and dysphagia for solids. He refers three days previously to acute neck pain in the context of cervical flexion when trying to get up from the head. No sensory alteration or sphincter control. No fever. No infections in previous days/weeks. No other symptoms. Hospital discharge date: 02/11/202. He spends 3 weeks and 2 days in hospital. 23 days.

Background

- No AMC.
- No known HTA, DM or DLP. No toxic habits. Morbid obesity.
- No medical-surgical history.
- Chronic treatment: does not refer.
- Baseline situation: independent for ABVD. Adequate cognitive. mRS 0.

Physical exploration

Neurological examination on admission: Preserved superior functions. PICNR. MOE's full. Exhaust horizontal extreme gaze nystagmus. Normal vp. Normal VIIp. PPBB preserved. BM: claudication to the MMII plane instantly. Strength (R/L) - MMSS: deltoids 4 +/4-, adductors 5/5, triceps 4-/4-, biceps 4 +/4-, wrist extension 5/5, wrist flexion 3/3, finger flexion 3/3, finger extension 4 +/4-, clamp 3/3, interossei 2/2, little finger 2/2. MMII psoas 3-/3-, quadriceps 3/3, abd/add 5/5, hamstrings 2/2, plantar flexion 5/5, dorsiflexion 5/5. No alterations tactile-algesic sensory, no sensitive level, no proprioceptive or vibratory alterations. ROT's abolished widespread. RCPFB. No dysmetria in D-N.

Neurological examination at discharge: Strength (R/I): MMSS: deltoids 5/5, adductors 5/5, triceps 4 +/4 +, biceps 4/4, wrist extension 5/5, wrist flexion 4 +/4 +, interosseous 4 +/4 +; MMII: psoas 4 +/4 +, quadriceps 5/5, abd-add 5/5, hamstrings 4/4, plantar and dorsal plexions 5/5. Generalized abolished ROTs except stylorradial and right bicipital +/+++ Sensitivity and normal NRL remainder.

Complementary explorations

- Analytical in the emergency room (01/19/21): Glu 191, Cr 0.68, Urea 24, Na 139, K 4, GPT 50, CK 213, LDH 216, PCR 0.72. Hemogram: leukocytosis 13.31 (N 66%, L 22.8%), Platelets 285,000. Normal clotting. Normal serum IgA.
- PCR Covid19 19.1.2021: Negative.
- AyS Urine: proteinuria +. Toxic: positive to BZD. BQ: proteins 28, albumin 69.4, albumin/creatinine ratio 33.8.
- Analytics: general BQ and ionogram within normal parameters. PCR 2.56-1.56, ESR 44. CT 196-216, with HDL 35-36 and LDL 133-155, normal TG. HbA1c 8.7. Hepatic profile and enzymes (LDH, CK) without alterations. TSH, free T4 and PTH without alterations. FG preserved. Negative tumor markers, slope S100. Ceruloplasmin and homocysteine normal. VitB12 and folate normal, vitamin D 4.5, vitamin E pending. Hemogram and coagulation without alterations. Proteinogram: albumin 44.3-47.7, calculated 3.6; gamma

32'2-27'7, calculated 2.6-2'1; compatible with polyclonal hypergammaglobulinemia. Thrombophilia: Weakly positive lupus anticoagulant. Immune: IgG 2222, pending subclasses, normal IgM, IgA and IgE; Kappa 22'38-21'9 free chains, normal Lambda, normal C3 and C4.

- Autoimmunity: Positive for anti-peroxidase (41'6-25'4) and anti-thyroglobulin (129), positive anti-GM1, positive anti-GAD (29.3).
- Serologies: Immune patient hepatitis B, hepatitis C, HIV negative. EBV IgG EBNA and VCA positive, VVA IgG positive, HSV IgG positive. Negative LUEs, borrelia, Coxiella.
- Urine 24 hours: Proteins 0.25, Kappa 3.01 chains, Lambda 0.8. Normal porphyrins. Heavy metals: Zinc and normal lead and copper.
- Cerebrospinal fluid: CSF. Cytobiochemistry: <5 leukocytes, 113 glucose, 22 proteins, ADA and ECA 0, ECA 0'0. Normal CSF. No cytological albumin dissociation. Clear and transparent.
- Negative aerobic culture. Gram negative. Neurotropic virus PCR: negative HSV, VZV, enterovirus and parechovirus. Negative CSF IgGNMO. Acellular cytology, not neoplastic or inflammatory cells.
- Chest Rx: Signs of vascular redistribution, cardiomegaly (although magnified Rx), elevation right hemidiaphragmatic.
- Urgent cervical MRI (19.1.2021): No evidence of collections in extramedullary or extradural spinal space showing stenosis or compressive myelopathy. Movement artifactual study, which limits the spinal assessment in which no obvious intramedullary alterations are detected.
- Skull MRI (01.22.2021): Cerebral MRI was performed with sagittal SET1, transverse FLAIR T1 and T2, diffusion, SWI-MIP and TSE DP/T2 and coronal-oblique TSET2 sequences. It is compared with TAC of 01/19/2021. No alterations are observed in the signal intensity of the cerebral or cerebellar parenchyma. Normal base ganglia, thalamus, and internal and external capsules. Optic nerves unaltered. Posterior fossa without significant alterations. In the diffusion study, no foci of diffusion alteration were seen. No deposits are observed hemosiderin. Ventricular system, cerebral sulci, and cisternal spaces of normal size. Paranasal sinuses within normal. Conclusion: Exploration in the limits of normality.
- Cervico-dorso-lumbar MRI (01.22.2021): MRI of the FULL SPINE was performed with sagittal sequences SET1, TSET2 and STIR and axial T2. It is compared with MRI of the cervical spine of 01/19/2021. Cranio-vertebral junction with normal characteristics. Vertebral bodies of normal morphology, alienation and signal intensity. Medullary canal and foramina of normal caliber, without appreciating significant medullary or root compression. Marrow morphology and normal signal intensity. Normal size hair tail roots. Conclusion: Full spine exploration without significant alterations.
- EMG 01/26/20: Admission was on 01.18.21, 8 days have passed since admission when the EMG was performed. 11 days with the clinic. The clinic starts on 01.16.21.

Electrophysiological data compatible with acute demyelinating inflammatory polyradiculoneuritis (AIDP-Guillain Barre Syndrome), predominantly motor, with incipient involvement of the motor trunks of both MMII, of acute course (10 - 11 days of evolution) without evidence of acute or subacute denervation at the proximal or distal muscle level.

Moderate distal chronic polyradiculopathic pattern of right and left L5 roots. + at the MMII level. In line with possible old lumbosacral radiculopathies that are not related to the current acute picture.

EMG control 02/02/2020: Admission was on 01.18.21 15 days have passed since admission. 18 days with the clinic when the EMG is repeated. The clinic starts on 01.16.21.

Electrophysiological data compatible with the persistence of an acute demyelinating inflammatory polyradiculoneuritis (AIDP-Guillain Barre Syndrome), predominantly motor, with involvement of the motor trunks of both MMII, CPE + bilateral ICC, of subacute course (18 days of evolution) where There is still no evidence of axonal degeneration, no evidence of acute or subacute denervation at the proximal or distal musculature level of both MMII, neurographic changes are evidenced towards clinical and neurographic improvement of the patient. The conduction block and delays are less marked, the answers F and H show data of improvement with respect to the last EMG. There is still no evidence of acute or subacute denervation.

Moderate distal chronic polyradiculopathic pattern selective to right L5 roots and left + at the MMII level. In line with possible old lumbosacral radiculopathies that are not related to the current acute picture.

Endocrine consultation: Unknown type 2 diabetes mellitus (HbA1c 8.7) + obesity. GFR > 90. Thyroid normal function with positive antiTPO and antiTG. Plan: No need for thyroid treatment. We advise TSH controls every 6-12 months in primary care. At discharge for diabetes we advise losing weight (we reinforce habits) and start treatment with Xigduo 5/1000 mg half a tablet with breakfast and a half with dinner for 10 days and if good tolerance go to 1 tablet at breakfast and another.

Principal diagnostic

- Acute demyelinating inflammatory polyradiculoneuritis (SD of Guillain Barre) AIDP.
- Registration date: 02/11/2021. 23 days of entry.

EMG description

Findings

- ENG sensitive and MMSS motor.
- N. ulnar sensitive right: Normal.
- Median sensitive right: Normal.
- Median right motor: Normal.
- Wave F of the median right motor: Normal.
- Right motor ulnar: Normal.

Sensitive ENG and MMII motor:

- Peroneo sensitive right and left: Normal.
- N. Sural right and left: Normal.

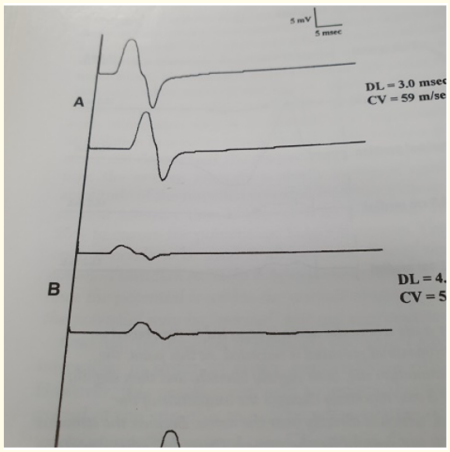

Figure 1: *Difference between NORMAL CMAP and another CMAP with axonal loss.*

Peroneus right motor: At the foot muscle level. The presence of motor conduction block, temporary potential dispersion, and increased distal latency of more than 125% of ULN was confirmed.

Wave F of the right motor peroneum. With less than 50% persistence and latency lag.

Peroneus motor left: At the foot muscle level. The presence of motor conduction block, temporary potential dispersion, and increased distal latency of more than 125% of ULN was confirmed.

Wave F of the right motor peroneum. With less than 50% persistence and latency lag.

Rear tibial right engine: The presence of motor conduction block, temporary potential dispersion, increased distal latency of more than 125% of ULN.

Wave F of the right posterior tibial. With less than 50% persistence and latency lag.

Reflex H of the right and left soleus. Tibialis posterior: Not obtained.

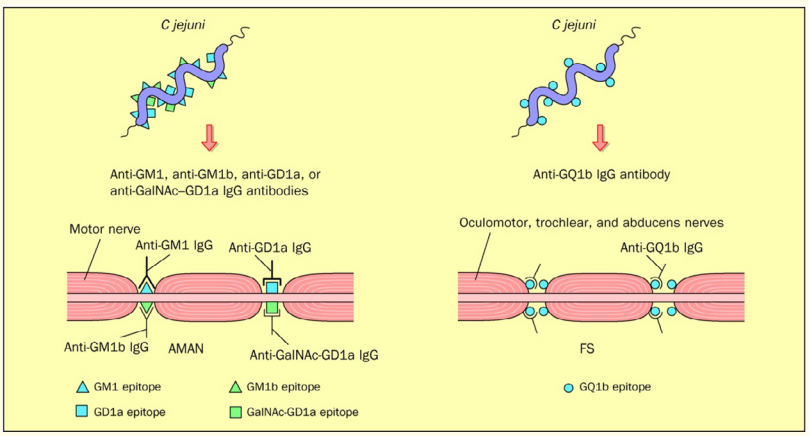

Figure 2: *Unequivocal conduction blocking, latencies, lags, and temporary potential dispersion.*

In this patient the following are observed: Delayed motor latencies, conduction speeds also show delays, the F responses of motor nerves of both MMII have latency increases greater than 120% of the ULN, there are conduction blocks that translate tomocular demyelization at the level of the:

- Right motor peroneal nerve. and left with altered F,
- Right and left tibial motor nerve. with altered F,
- Bilateral pathological H reflex.

Conventional EMG

During the study with the needle electrode in MMII, at rest, no spontaneous activity was detected in the scanned muscles. In the qualitative study of motor units, signs of chronic denervation have been found in muscles innervated by different nerves but sharing right L5 root territory. and left at the level that it speaks in favor of probable old, chronic radiculopathies, L5 right. and left of moderate degree, without data of active denervation (anterior tibialis, pedio, left and right lateral peroneal), with simplified maximal effort tracings. Normality in the rest of the muscles explored. Moderate distal chronic polyradiculopathic pattern, selective to the roots l5 right and left. at the level of MMII without data of active denervation. The presence of these potentials speaks in favor of a lesion of more than 6 months of evolution, showing polyphasic, unstable, irregular PUM, with a notable increase in duration, and degree of polyphasia, and amplitude without signs of denervation. (0/++++), which gives the lesion a chronic nature (6-12 months). No myopathic PUM (polyphasic, short-term and low amplitude PUM) nor signs of primary destruction of muscles, the Turns/amplitude analysis of the PUM (muscle motor unit potentials) studied through EMG presents a normal distribution. No signs of acute or subacute denervation. MUAPs had normal characteristics but a reduced recruitment pattern was observed.

Evolution and complications

This is a 43-year-old patient with no known VRF who is admitted due to progressive limb weakness with no other symptoms of 48 hours of evolution. He highlights tetraparesis on examination, predominantly distal in upper limbs and proximal in lower limbs, with areflexia. Normal urgent cervical MRI. Given the suspicion of acute polyradiculoneuropathy, LP was performed, which is normal, and immunoglobulins were started and he was admitted for study.

During admission, he maintains high blood pressure and blood glucose levels in relation to unknown hypertension and DM, treatment is added, also for vitamin D deficiency, and is assessed by the endocrinologist who gives recommendations at discharge. In analytical polyclonal hypergammaglobulinemia in relation to the administration of immunoglobulins, normal neuroaxis MRI, EMG confirming the diagnosis of suspected Guillain-Barre syndrome (GBS) and control EMG with improvement.

He completes the 5 days of immunoglobulins (35g per day) with good tolerance and RHB is started with progressive improvement in EE strength and disappearance of dysphagia. At discharge, weakness persists in the pelvic girdle that prevents him from standing but that according to the physio and RHB will benefit more from ambulatory RHB.

Discussion

GBS is a post-infectious autoimmune disorder, with *Campylobacter jejuni* being the microorganism that has the highest incidence in the etiopathogenesis of the syndrome [4,10].

Studies have shown that bacteria isolated from patients with Guillain-Barre syndrome possess a lipopolysaccharide that closely mimics ganglioside GQ1b [11,20]. and have shown a molecular mimicry with the terminal structures of the lipo-oligosaccharides of *Campylobacter jejuni* with the GM1 and GD1a gangliosides present in the axons of peripheral motor nerves, hence the preference for autoimmune attack on motor trunks, more that to the sensitive ones, the AIDP is preferentially motor, this does not exclude the affectation of sensory nerves, the anti GM1 and GD1a antibodies bind to the target antigens, located inside and outside the nodule of Ramvier, leading to the destruction of the sodium channels dependent voltage. This damage can lead to detachment of the paranodal myelin, which leads to a decrease in conduction speed, loss of electrical charges, decreasing and altering the conductivity of the nerve and generating the so-called motor conduction block [12,18].

Both SMF and trunk encephalitis (Bickerstaff), isolated ophthalmoplegia and GBS have a clinical spectrum with overlap between them. Although the criteria that define these disorders are very precise in clinical practice, patients frequently express signs of both entities [3,16]. All of them are frequently preceded by respiratory or gastrointestinal infection and share the same autoimmune mechanism. The same germ, for example *C. jejuni*, can induce the formation of Ab anti-GM1, GD1a, GQ1b, among others, since it carries epitopes common to all of them. In the event that the Ab are exclusively anti-GQ1b, the expressiveness of the disease will be in the form of isolated acute ophthalmoplegia or SMF. If there is also Ab against other gangliosides, the expressiveness tends to be mixed, Miller Fisher syndrome (MF) and GBS (Guillain-Barre syndrome) or trunk encephalitis with or without GBS Guillain-Barre syndrome [13-19].

During the first days of the disease (latency period 1 - 7 days from the onset of symptoms) nerve conduction studies may be normal, it is necessary to let the latency period pass (7 - 10 days), but given the possibility of normality in early electrophysiological studies the presence of a characteristic clinical picture accompanied by alterations of the cerebrospinal fluid around the first week after the onset of compatible symptoms should be considered diagnostic and the start of treatment should not be conditioned to the confirmation of compatible electrophysiological alterations [1]. As some electrophysiological studies in the latency period (first and/or second week) may be normal, in our case this was not the case since we saw the patient 11 days after the start of the clinic, admission was 18.01,21 had passed 8 days from admission when the EMG is done. 11 days with the clinic. The clinic begins on 01/16/21 and was already showing electrophysiological data to be able to diagnose the syndrome. One of the purposes of this work is to improve the diagnostic performance of EMG-ENG in GBS, to improve the diagnostic performance, some authors recommend conducting studies in at least 3 sensory nerves and 4 motor nerves, in addition to the importance of the F waves and H reflex of the soleus, in our case 6 sensory nerves and 6 motor nerves (2 upper limbs and 4 lower limbs) were studied, the most frequently found changes in the first days: the absence or delay of the F waves and the H reflex of the soleus [1,19], in mild cases the pathological changes may consist of edema of the nerves or of the roots with a minor inflammatory component, in the most severe cases, the nerves become inexcitable and there is usually axonal degeneration concomitant with the intense demyelination and inflammatory response [1,19].

In our laboratory we usually see the order of 5 - 10 GBS per year and in our experience we have seen patients with GBS of 5 - 7 days of evolution and it was already affected in the right median nerve, the right motor peroneum, mild sensory involvement, and paresis bilateral facial, latencies and conduction velocities still normal, 11 days later the blocks were clear in motor nerves of both MMII,, F altered,, median severely affected, hence the importance of serial studies, to see the degree of destruction that can occur in the peripheral nervous system.

In another serious case of 1 month of evolution: the affectation was plausible and clear:

- Complete absence of F and H responses, absence of CMAP of the median and lower limb nerves, and there was bilateral l5, s1 denervation.
- In another case seen in our laboratory with 7 - 10 days of evolution, the F and the H reflex of the soleus were affected, and there were already conduction blocks of the MMII motor nerves, the peroneal motor nerves more affected.

In cases where the studies are normal because they are very early, it is advisable to put mediation and do a sequential study from the 5 - 7 day of the onset of symptoms.

The most common findings in early studies (less than 2 weeks from the onset of symptoms) are:

- Delay of F.
- H delay
- Motor conduction blocks (upper and lower limbs)
- Blink reflex - altered blink reflex.
- The presence of multiple A waves in the H reflection of the soleus.
- Distal temporal dispersion.

Considering as more reliable findings:

- The abnormalities in the latencies F and the reflex H.
- In addition to the increase in distal motor latencies.
- Sural preserved.

In AIDP, from the electrophysiological point of view, the first findings to be found are the delay, absence or impersistence of the H and F responses (median, ulnar, peroneal motor, bilateral posterior tibial) that indicate a process of proximal demyelination of the lesions. roots at that level studied. Afterwards, routine conduction studies will show processes of focal segmental demyelination (tomocular demyelination) that we can see on motor neurography as motor conduction blocks and temporary potential dispersion.

These changes are present in 50% of patients with AIDP at 2 weeks and in 85% at 3 weeks [1,2,19,20]. Some patients have early nerve inexitability and occasionally Wallerian degeneration or proximal demyelination. We must consider the variants of GBS, (AMAN) acute axonal polyneuropathy (pure motor syndrome) and (AMSAN) acute axonal sensitive-motor polyneuropathy [1,2,19].

In AIDP, the classic demyelinating form, which is our specific case, an acute inflammatory demyelinating polyneuropathy was evidenced without acute or subacute axonal degeneration. It was a purely demyelinating process in our patient. And it is necessary in ENG-EMG to demonstrate the presence of segmental demyelination of motor nerves, motor conduction blocks, temporal dispersion of potential, marked delays in distal latencies, delayed conduction velocities, and the delay, absence or impersistence of the answers F and H [1,2,20].

Electrophysiological criteria to define an AIDP [2,19]:

1. DL prolonged distal latencies 8 2 or more nerves, not in common entrapment sites):
    - Distal DL-latencies greater than 115% (if CMAP amplitude is normal).
    - Distal DL-latencies greater than 125% (for amplitudes less than the lower limit of normal-LLN).

2. VC conduction velocity delays (2 or more nerves, not in common entrapment sites):

    - VC (conduction speeds) less than 90% lower limit of normal-LLN-lower limit of normal) (For CMAP amplitudes greater than 50%).

    - VC (conduction speeds) less than 80% lower limit of normal-LLN-lower limit of normal) (For CMAP amplitudes less than 50%).

3. Latency of the F response and H reflex of the soleus (1 or more nerves) delay more than 125% ULN - ULN-high limit of normality (upper limit of normal.) [1,2,19]. Note if the distal amplitude of the CMAP is very low, the absence of F may not be considered abnormal.

4. Conduction blocks/temporary potential dispersion (1 or more nerves):

    - Unequivocal conduction block: Proximal/distal CMAP area ratio, less than 0.5.

    - Possible conduction block: Proximal/distal CMAP area ratio, less than 0.7.

Temporal dispersion: Proximal/distal CMAP area ratio, greater than 1.5 [1,2,19,20]:

- DL-Distal latencies
- VC-Driving speeds
- LLN-Low limit of normality (lower limit of normal)
- ULN-High limit of normality (upper limit of normal) [1,2,18,19]
- CMAP-Compound muscle action potential - compound muscle action potential - motor nerve neurography [1,2,18-20].

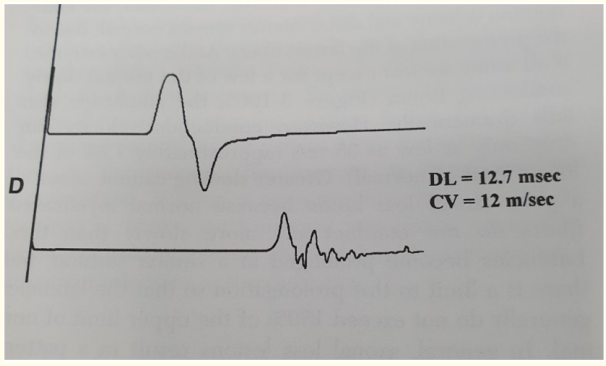

Figure 3: *Unambiguous conduction blocking, latencies, lags, and temporary potential dispersion. Very characteristic of the AIDP.*

The figure above shows the characteristic alterations of the AIDP, delayed latencies and conduction block, as well as temporal dispersion of the potential.

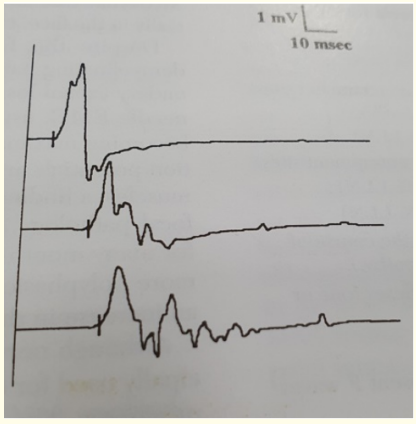

Figure 4: *Another example of conduction block.*

About 90% of patients with AIDP may have abnormalities in conduction studies in the first few weeks after the onset of symptoms, it is common the presence of sensory nerve conduction studies (SNAP) show normal values at the beginning. After the first week or 2 weeks, it is relatively common to find an undamaged sural, a sural with normal values, (normal sural as opposed to a median or an affected ulnar, with absence of SNAP or falling amplitudes). This is what Called in Anglo-Saxon terms ("sural sparing"), this sural sparing is very typical in AIDPs. For this reason, the presence of a normal sural should not make us desist from the diagnosis of AIDP. It is not really known why the sural is especially preserved [1], it is thought that the reason is because the sural nerve has more myelin density in its more proximal place of stimulation (near the soleus muscle), than the median and ulnar nerves Sensitive that have a more distal registration in the fingers of the hand. The sural nerve has a longer diameter of myelin at the site of stimulation and recording and therefore has greater resistance to injury of a demyelinating nature [1,18,19].

The needle EMG, as we were able to verify in our patient, did not show signs of acute or subacute denervation, the MUAP had normal characteristics but a reduced recruitment pattern was observed. In AIDP, the finer myelinated fibers are affected first.

We have seen our patient through 2 EMG, one, the first at 10 days of evolution of the clinical picture and another EMG at 18 days after the onset of symptoms in view of the clinical improvement of the patient.

In conclusion for future nerve conduction studies:

1. Consider whether the patient is in a latency period or not facing the results of the neurography.

2. Extend the ENG study if more motor and sensitive nerves are needed.

3. It is interesting in this type of patient ENG-EMG sequential studies to verify and see the evolution of the process.

4. Carry out a complete ENG-EMG protocol with H, F responses, Sensitive and Motor Neurography of the upper limb and both lower limbs.

In our patient, despite the fact that the 2 EMGs were pathological, the evolution was favorable after 21 days of hospital admission. In analytical polyclonal hypergammaglobulinemia in relation to the administration of immunoglobulins, normal neuroaxis MRI, EMG confirming the diagnosis of suspected Guillain-Barre syndrome (GBS) and control EMG with improvement. He completes the 5 days of

immunoglobulins (35g per day) with good tolerance and RHB is started with progressive improvement in EE strength and disappearance of dysphagia. At discharge, weakness persists in the pelvic girdle that prevents him from standing, but according to the physio and RHB, he will benefit more from ambulatory RHB.

Reason for discharge: Clinical improvement of the patient diagnosed with AIDP.

Conclusion

In this patient the following are observed: Delayed motor latencies, conduction velocities also show delays, the F responses of motor nerves of both MMII have latency increases greater than 120% of the ULN, there are conduction blocks that translate tomocular demyelization at the level of the:

- Right motor peroneal nerve. and left with altered F,
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Electrophysiological data compatible with acute demyelinating inflammatory polyradiculoneuritis (AIDP-Guillain Barre Syndrome), predominantly motor, with incipient involvement of the motor trunks of both MMII, of acute course (10 - 11 days of evolution) without data of acute or subacute denervation to level of the proximal or distal muscles in the lower limbs.

EMG control 02/02/2020: Electrophysiological data compatible with the persistence of an acute demyelinating inflammatory polyradiculoneuritis (AIDP-Guillain Barre Syndrome), predominantly motor, with involvement of the motor trunks of both MMII, CPE + bilateral ICC, of subacute course (18 days of evolution) where there is still no evidence of axonal degeneration, no evidence of acute or subacute denervation at the proximal or distal muscle level of both MMII, neurographic changes are evidenced towards improvement clinical and neurographic of the patient. The conduction block and delays are less marked, the answers F and H show data of improvement with respect to the last EMG. There is still no evidence of acute or subacute denervation.

Moderate distal chronic polyradiculopathic pattern selective to right L5 roots and left + at the MMII level. In line with possible old lumbosacral radiculopathies that are not related to the current acute picture.

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