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Case Report

About a Case of Miller Fisher, in a 10-Year-Old School, in Our Hospital

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Abstract

Miller Fisher syndrome (SMF) 6%. It is a type of acute inflammatory demyelinating polyradiculoneuropathy, a variant of Guillain Barre syndrome (GBS). There is demyelination in its etiopathogenesis. It is very rare in childhood, hence the interest in this specific case since it occurs in a 10-year-old schoolgirl with a benign and self-limited subsequent evolution. The annual incidence is 0.09% per 100,000 inhabitants. In series published in children, it accounts for approximately 9% of cases of acute inflammatory polyradiculoneuritis [2]. As with GBS, it is frequently triggered by certain strains of *Campylobacter jejuni* that induce the activation of the immune system and the generation of anti-GQ1b anti-ganglioside antibodies. *Haemophilus influenzae* and *Mycoplasma pneumoniae* infections have also been described. There are antibodies, for example, GQ1b that not only act on *C. jejuni*, but also on the outer part of the membrane of paranodal Schwann cells where these gangliosides (GQ1b) exist similar to those existing in the outer layer of *C. jejuni*, activating complement with the development of paranodal myelin vacuolization of oculomotor cranial nerves (III, IV and VI), spinal roots, motor and sensory nerves, mainly of the lower extremities, followed by disintegration of myelin phagocytosed by invading macrophages the affected area and after infiltration with lymphocytes [1,7,8]. The inflammatory reaction is rarely so intense that the axon that has lost its myelin may undergo degeneration, but its etiopathogenesis is demyelinating in nature and therefore there may be subsequent axonal degeneration. These Ab recognize epitopes that are specifically expressed in the nodal regions of the oculomotor nerves, in the dorsal ganglia and in cerebellar neurons, structures responsible for the symptoms of MFS. It is a selective autoimmune attack on the gangliosides of the myelin sheath. With special predilection in the cranial nerves (III, IV and VI) and the spinal roots and sensory nerves [1,7]. Attack of cells sensory ganglia and cerebellum neurons. It generates a classic triad of ataxia areflexia and ophthalmoplegia. There is demyelination of cranial nerves and spinal roots. There is no axonopathy in the etiopathogenesis. We present this case of this 10-year-old girl, where, thanks to the treatment, the evolution and the prognosis were good.

Keywords: *Miller-Fisher; Demyelinating Polyneuropathy; Antiganglioside Antibodies; Autoimmunity; Cranial Nerves; Ataxia; Areflexia; Ophthalmoplegia*

Introduction

Miller Fisher's syndrome (SMF) 6%. It is a type of acute inflammatory demyelinating polyradiculoneuropathy, a variant of Guillain-Barre syndrome (GBS). There is demyelination in its etiopathogenesis. It is very rare in childhood, hence the interest in this specific case given that it occurs in a 10-year-old schoolgirl with a subsequent benign and self-limited evolution.

The annual incidence is 0.09% per 100,000 inhabitants. In the few series published in children, it accounts for approximately 9% of cases of acute inflammatory polyradiculoneuritis. (two). As with GBS, it is frequently triggered by certain strains of *Campylobacter jejuni*

that induce the activation of the immune system and the generation of antiganglioside anti-GQ1b antibodies. Cases of infections by *Haemophilus influenzae* and *Mycoplasma pneumoniae* have also been described. There are antibodies, for example, GQ1b that not only act on *C. jejuni*, but also on the outer part of the membrane of paranodal Schwann cells where there are these gangliosides (GQ1b) similar to those in the outer layer of *C. jejuni*, activating the complement with the development of paranodal myelin vacuolation of the oculomotor cranial nerves (III, IV and VI), roots, spinal sensory and motor nerves, mainly of the lower extremities, followed by disintegration of the myelin phagocytosed by macrophages that invade the affected area and after infiltration with lymphocytes [1,7,8].

The inflammatory reaction is rarely so intense that the axon that has lost its myelin may experience degeneration, but its etiopathogenesis is demyelinating in nature and therefore axonal degeneration may follow.

These Ab recognize epitopes that are specifically expressed in the nodal regions of the oculomotor nerves, in the dorsal ganglia and in cerebellar neurons, structures responsible for the symptoms of FMS. It is a selective autoimmune attack on the gangliosides of the myelin sheath. With special predilection in the cranial nerves (III, IV and VI) and the spinal roots and sensory nerves [1,7]. Attack sensory ganglia cells and cerebellar neurons. It generates a classic triad of ataxia areflexia and ophthalmoplegia. There is demyelination of cranial nerves and spinal roots. There is no axonopathy in the etiopathogenesis. We present this case of this 10-year-old girl, where, thanks to the treatment, the evolution and prognosis were good.

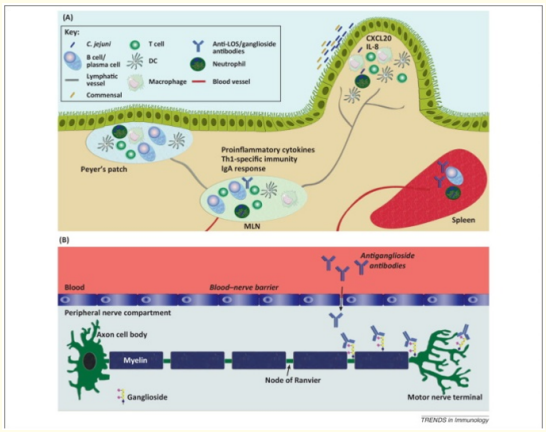


Figure 1

Reason for consultation

She was admitted to the hospital due to gait instability, non specific alterations of the ocular cranial nerves and arreflexia for 10 days. Ataxia under study. He refers diplopia and blurred vision to the left.

Clinical Case Presentation

Family background

Mother: 41 years old. Fury. Father: 51 years old. Healthy. Healthy 4, 12 and 21 year old sisters.

Personal history

Her mother's pregnancy controlled without incident, normal prenatal ultrasounds. Correct vaccinations. Psychomotor and weight-bearing development referred to as normal. No medical-surgical history of interest for the case.

Current illness

Consultation for a 10-year-old girl who came to the emergency room due to progressive weakness in the lower limbs of 6 days of evolution associated with dysarthria (nasal voice) and headache. Diplopia of the same evolution that prevents reading. One month before, they reported an episode of diarrhea that was self-limited.

Physical exploration

Weight: 39.5 kg. Temperature: 35.6ºC. TA: 103/71. HR: 99bpm. $SatO_2$: 99%.

(Upon admission): Stable PTSD. BEG. NRL: Glasgow 15/15, poorly reactive middle pupils, left palpebral ptosis with correct opening if requested to open the eyes. EOMs preserved, with pain on extreme lateralization, without nystagmus. Centered cranial nerves, no facial asymmetries in front or mouth corner. Strength in MMSS 5/5, MMII 5/5. Sensitivity apparently preserved she notices changes in temperature when removing her clothes. I don't know they get patellar ROTs, yes right bicipital (via in left flexure). Spontaneous mobility of all limbs, she sits and gets up from the stretcher alone. When walking she presents slight nonspecific instability, with normal Baran and Unterberger, normal tandem gait. No falls. Negative meningeal signs. Normal ENT. Normal ACP. Normal abdomen. Skin without lesions.

Complementary explorations

- Blood test (09/18/2021): leukocytes: 10090 (7140N, 2080L, 370M). Platelets: 318000, Hemoglobin: 13.4 g/dL, hematocrit: 39.3%. Glucose: 93, urea: 20, Cr: 0.33, bilirubin: 0.64, CRP: < 0.06 mg/dL. Na: 141, K: 4.6, Cl: 105 mEq/L. GOT: 19, GPT: 14, GGT: 14, FA: 321, CK: 76, LDH: 199, Amylase: 64IU/L. TP: 75%, APTT: 37.8s.
- CSF analysis (09/18/2021) cerebrospinal fluid study: 1 leukocyte/field. Glucose: 58, proteins: 22.1 mg/dL. Normal values normally fluctuate as follows:
    - Pressure: from 70 to 180 mm H_2O.
    - Appearance: transparent, colorless.
    - Total protein in CSF: 15 to 60 mg/100 mL.
    - Gamma globulin: 3% to 12% of the total protein.
    - CSF glucose: 50 to 80 mg/100 mL (or greater than two-thirds of the blood sugar level).

CSF cerebrospinal fluid study: Examination in cases of MFS may show albuminocytological dissociation, although it may be normal initially. And after the seventh day of onset of the symptoms, discreet hyperprotenorachia, as in the case of our patient, with its maximum expression at 4 - 6 weeks, the CSF examination is not a parameter in which the diagnosis can be supported, although it can serve to support you [16].

AcAntiganglioside IgM and IgG: Positive in blood, pending in CSF. Later they also tested positive in CSF cerebrospinal fluid study.

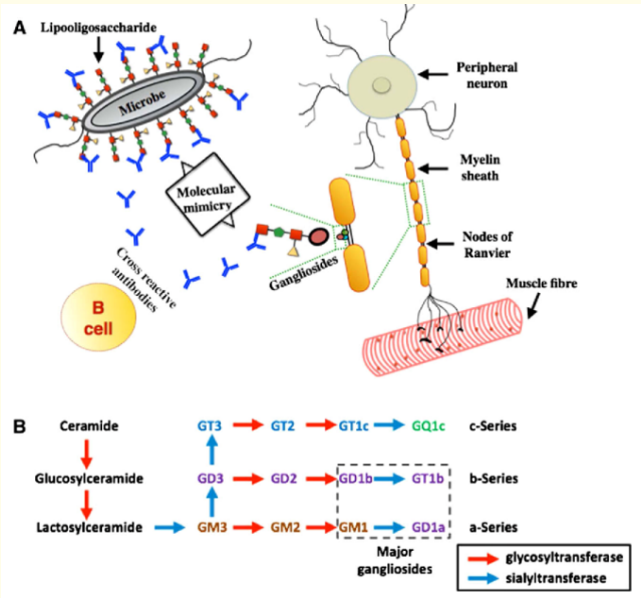


Figure 2

Toxic in urine (18.09.2021): Negative for all drugs tested.

Serology (20.09.2021): RPR, hepatitis A, hepatitis B, hepatitis C, HSV, HIV, CMV, EBV, Chagas, *Brucella*, *Borrelia*, negative. IgM *M. pneumoniae* low positive, IgG positive. Pending *Coxiella, Chlamydia, Bartonella*, *Francisella*.

PCR virus (09/18/2021): Negative for enterovirus, HSV, VHZ, EBV, CMV, HHV 6 and 7.

Cranial MRI (09/23/2021): No significant findings.

ITC to ophthalmology (09/20/2021): The patient reported constant horizontal binocular diplopia. In the distant cover test, hypertropia and esotropia of the left eye were observed. A close-up cover test shows exophoria and subtle hypertropia of the left eye. In the lateral versions (right and left) she presents a low frequency broad nystagmus, with evident limitation to levoabduction and more moderate limitation to dextroabduction. She had no impressions of limitations in the rest of the MOEs (external ocular motors) although she reported diplopia in the position of both superior obliques and presented strong ocular pain upon supraversion.

EMG (21.09.2021): Data suggestive of demyelinating sensory-motor polyradiculoneuropathy, in acute stage (10 days) with involvement in upper body limbs and lower limbs of the body, absence of responses in the trigeminal-facial reflex. These findings are similar to those described in Miller-Fisher syndrome (MFS). CMAP latency delay and slight drop in amplitude of the left facial. Regarding the right. Left facial paresis.

Procedures

- Conduction speeds made with surface electrodes.
- EMG performed with a concentric needle electrode.

EMG description

Study is requested for suspected Miller-Fisher syndrome.

It has been explored:

- Sensitive electroneurography (ENG) of sural N, superficial peroneal N, superficial radial N, and right median nerve (partialized) N.
- Motor ENG of bilateral peroneal N and posterior tibial N (left distal), median N, ulnar N (partialized) and right facial N.
- Answer F on peroneal N, posterior tibial N, median N and right ulnar N.
- Bilateral trigeminal-facial reflex.

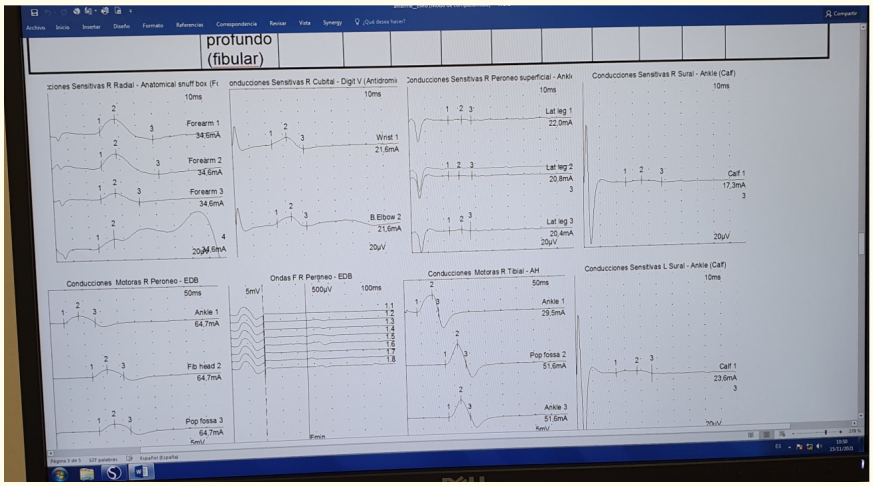
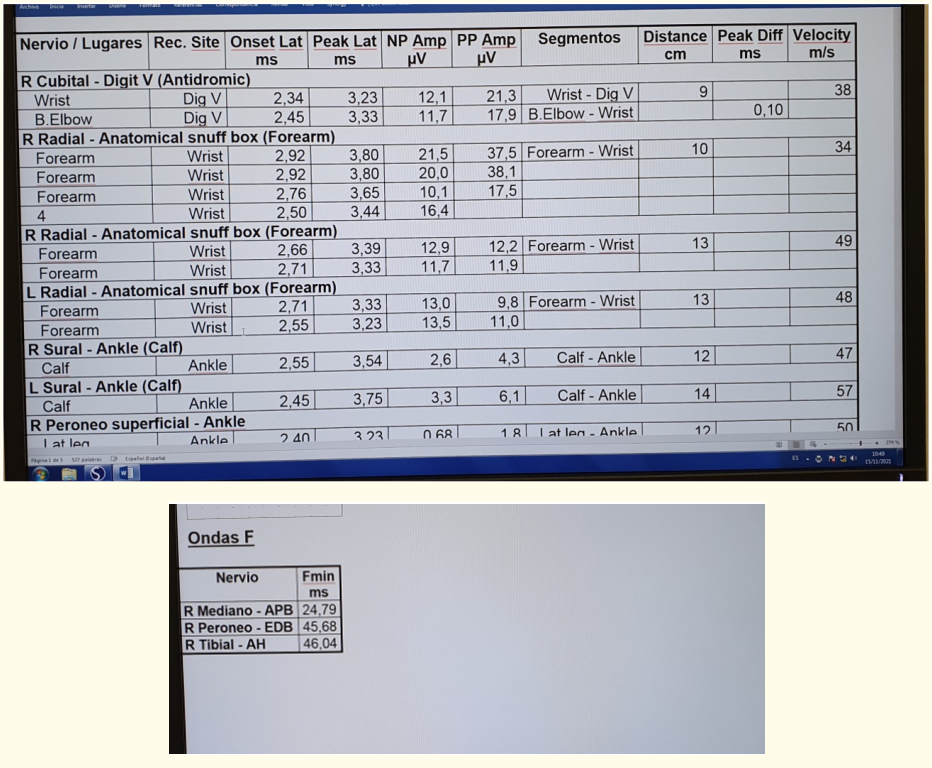


Figure 3

Sensitive ENG: Absence of assessable PRS (sensitive potential) in right and left N sural and superficial peroneal N. (the patient correctly reports the irradiation of the stimulus). The low amplitude of the SNAP is striking, as well as its temporal dispersion, low amplitude PRS and widening in superficial radial N. and fall in sensory amplitude. PRS (sensitive potential) of low amplitude and dispersed in medium N, without clear slowing in the palm-wrist path. VCS in low limits or somewhat slowed down. Drop in the amplitude of the PRS (sensitive potential) of both radial and right ulnar. Distal sensitive amplitude reduction and absence due to the moment of acute denervation at the distal muscualture level. (10 days of the process) temporary dispersion of the sensitive potentials. CV from 34 radial, 38 ulnar up to 50 m/sec.



Nervio / Lugares	Rec. Site	Onset Lat ms	Peak Lat ms	NP Amp µV	PP Amp µV	Segmentos	Distance cm	Peak Diff ms	Velocity m/s
R Cubital - Digit V (Antidromic)									
Wrist	Dig V	2,34	3,23	12,1	21,3	Wrist - Dig V	9		38
B.Elbow	Dig V	2,45	3,33	11,7	17,9	B.Elbow - Wrist		0,10	
R Radial - Anatomical snuff box (Forearm)									
Forearm	Wrist	2,92	3,80	21,5	37,5	Forearm - Wrist	10		34
Forearm	Wrist	2,92	3,80	20,0	38,1				
Forearm	Wrist	2,76	3,65	10,1	17,5				
4	Wrist	2,50	3,44	16,4					
R Radial - Anatomical snuff box (Forearm)									
Forearm	Wrist	2,66	3,39	12,9	12,2	Forearm - Wrist	13		49
Forearm	Wrist	2,71	3,33	11,7	11,9				
L Radial - Anatomical snuff box (Forearm)									
Forearm	Wrist	2,71	3,33	13,0	9,8	Forearm - Wrist	13		48
Forearm	Wrist	2,55	3,23	13,5	11,0				
R Sural - Ankle (Calf)									
Calf	Ankle	2,55	3,54	2,6	4,3	Calf - Ankle	12		47
L Sural - Ankle (Calf)									
Calf	Ankle	2,45	3,75	3,3	6,1	Calf - Ankle	14		57
R Peroneo superficial - Ankle									
Lat leg	Ankle	2,40	3,23	0,68	1,8	Lat leg - Ankle	12		50

Ondas F

Nervio	Fmin ms
R Mediano - APB	24,79
R Peroneo - EDB	45,68
R Tibial - AH	46,04

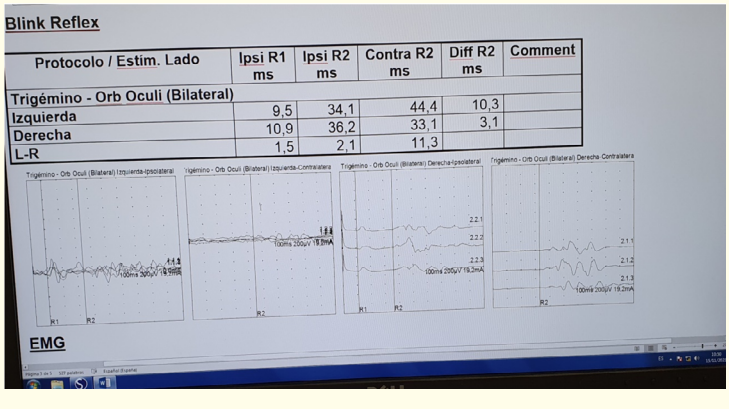
Figure 4

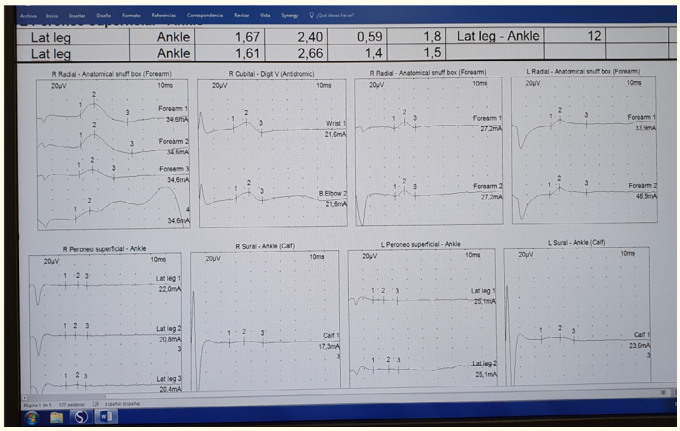
Motor ENG: CMAP increased distal latency in median N. Slowness of the MCV of the ulnar N at the elbow. VCM somewhat slow in MMSS. Rest within normality.

Answer F: Normal minimum latency and persistence in MMII. Presence of axonal, low persistence and minimal latency increase in MMSS.

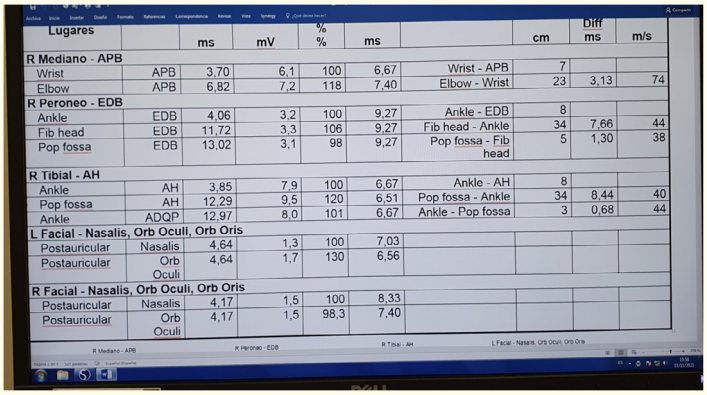
Trigeminal-facial reflex: Absence of assessable responses on the left side. Delay and amplitude drop of R1 and R2 left.

CMAP latency delay and slight drop in amplitude of the left facial. Regarding the right. Left facial paresis.





Lat leg	Ankle	1,67	2,40	0,59	1,8	Lat leg - Ankle	12
Lat leg	Ankle	1,61	2,66	1,4	1,5		



Lugares		ms	mV	%	%	ms		cm	Diff ms	m/s
R Mediano - APB										
Wrist	APB	3,70	6,1	100		6,67	Wrist - APB	7		
Elbow	APB	6,82	7,2	118		7,40	Elbow - Wrist	23	3,13	74
R Peroneo - EDB										
Ankle	EDB	4,06	3,2	100		9,27	Ankle - EDB	8		
Fib head	EDB	11,72	3,3	106		9,27	Fib head - Ankle	34	7,66	44
Pop fossa	EDB	13,02	3,1	98		9,27	Pop fossa - Fib head	5	1,30	38
R Tibial - AH										
Ankle	AH	3,85	7,9	100		6,67	Ankle - AH	8		
Pop fossa	AH	12,29	9,5	120		6,51	Pop fossa - Ankle	34	8,44	40
Ankle	ADQP	12,97	8,0	101		6,67	Ankle - Pop fossa	3	0,68	44
L Facial - Nasalis, Orb Oculi, Orb Oris										
Postauricular	Nasalis	4,64	1,3	100		7,03				
Postauricular	Orb Oculi	4,64	1,7	130		6,56				
R Facial - Nasalis, Orb Oculi, Orb Oris										
Postauricular	Nasalis	4,17	1,5	100		8,33				
Postauricular	Orb Oculi	4,17	1,5	98,3		7,40				

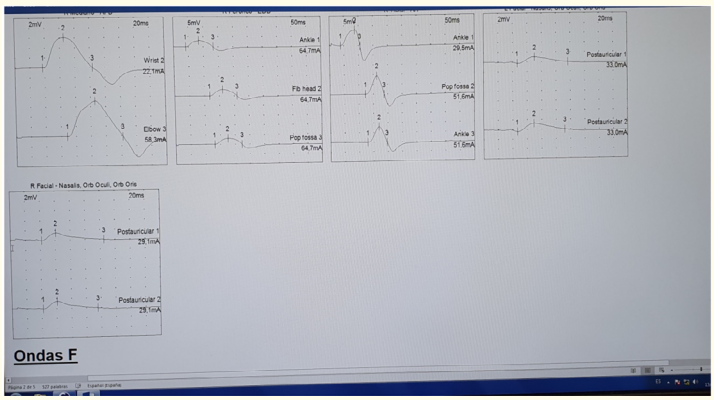


Figure 5

Principal diagnostic:

- Miller-Fisher Syndrome

Discussion

In the Miller Fisher syndrome the most common symptoms are:

- Diplopia (63%)
- Ataxia (33%)
- Sensory dysesthesias (17%)
- Ptosis (35-45%)
- Facial paralysis (32-35%)
- Pupillary abnormality in 42% of cases
- Muscle weakness (25%)
- Sensory alteration (52%) (15)
- Ophthalmoplegia (65% cases)
- Areflexia (33%)
- Bulbar paralysis 26%
- Dysesthesia 24%
- Weakness 20%
- Urinary incontinence 16%.

There are antibodies, for example, GQ1b that not only act on *C. jejuni*, but also on the outer part of the membrane of paranodal Schwann cells where these gangliosides exist (GQ1b), gangliosides are important components of peripheral nerves, there are 4 gangliosides GM1, GD1a, GT1a and GQ1b, and differ in the number and position of sialic acids, where M, D, T and Q represent mono, di, tri and quadri sialosil groups [11,12] similar to those existing in the outer layer of *C. jejuni*, activating the complement with the development of vacuolization of paranodal myelin of the oculomotor cranial nerves (III, IV and VI), roots, motor and sensory nerves, mainly of the lower extremities, followed of disintegration of myelin phagocytosed by macrophages that invade the affected area and after infiltration with lymphocytes. The Ig G autoantibodies directed to the gangliosides GQ1b, cross-react with GT1a; are strongly linked to MFS, its incomplete forms and its variant at the CNS level 8 Bickerstaff encephalitis, which includes acute ophthalmoplegia, ataxia, and altered consciousness after an infectious episode [1,7,8]. Studies have shown that bacteria isolated from patients with Guillain-Barre syndrome possess a lipopolysaccharide that closely mimics ganglioside GQ1b [13].

The ganglioside GQ1b is strongly expressed in the oculomotor, trochlear, and abductor nerves, as well as the muscle spindles of the extremities [8,9].

The glossopharyngeal and vagus nerves strongly express the ganglioside GT1a and GQ1b, causing dysphagia [10].

There is evidence of early complement activation, which is based on antibody binding to the outer surface of the Schwann cell and deposition of activated complement components, such activation initiates myelin vacuolization, macrophage invasion is observed 1 week after complement-mediated myelin damage occurs [6,10,15].

The inflammatory reaction is rarely so severe that the axon that has lost its myelin may degenerate. Therefore, in SMF there is no axonopathy in its etiopathogenesis, it is an attack on the myelin sheath. The paranodal myelin of the cranial nerves, roots, motor nerves, and sensory spinal ganglia is destroyed.

These Ab recognize epitopes that are specifically expressed in the nodal regions of the oculomotor nerves, in the dorsal ganglia and in cerebellar neurons, structures responsible for the symptoms of MFS [3,7,9,10].

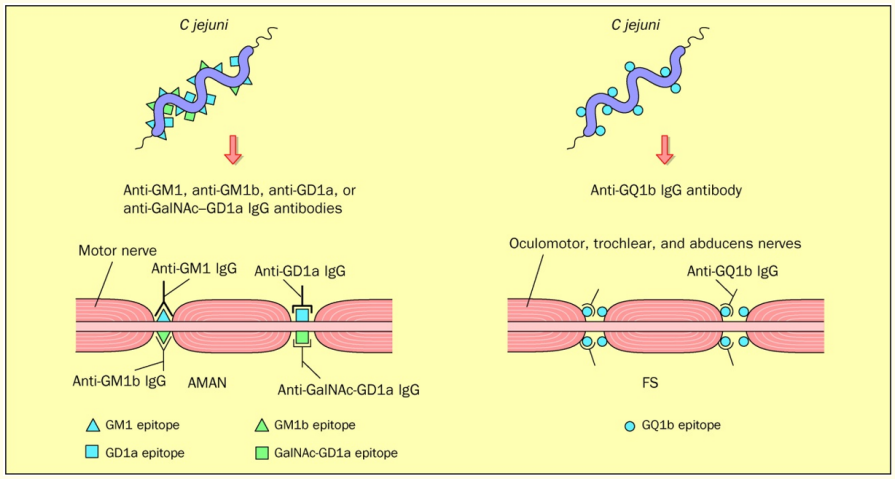


Figure 6

The infectious agents that precede the onset of Miller Fisher syndrome (MFS), the most related are *Campylobacter jejuni* (21%), *H. influenza* (8%), cytomegalovirus (3%) and *M. pneumoniae* (3%) [4] agents like him. *C. jejuni*, cause the formation of antibodies, for example, GQ1b that not only act on *C. jejuni*, but also on the outer part of the membrane of paranodal Schwann cells where these gangliosides (GQ1b) exist similar to those existing in the outer layer of *C. jejuni*, activating the complement with the development of vacuolization of the paranodal myelin of the oculomotor cranial nerves (III, IV and VI), roots, motor and sensory nerves, mainly of the lower extremities, followed by disintegration of myelin phagocytosed by macrophages that invade the affected area and after infiltration with lymphocytes. The inflammatory reaction is rarely so severe that the axon that has lost its myelin may degenerate [3,15].

These Ab recognize epitopes that are specifically expressed in the nodal regions of the oculomotor nerves, (ophthalmoplegia) in the dorsal ganglia (ataxia and areflexia polyneuropathy) and in cerebellar neurons (ataxia), structures responsible for the symptoms of MFS. The form that, although the most atypical, is considered to be the most frequent of the variants of Guillain-Barré syndrome (GBS); Other symptoms such as distal paresthesias, facial, oropharyngeal, or limb weakness are often associated, occasionally progressing to a classic form of GBS. There are not many neurophysiological studies of patients with MFS, but signs of peripheral polyneuropathy of the demyelinating or axonal type have been found, with a predominance of sensory, which may explain the ataxia as dysfunction of the peripheral sensory nerves (sensory polyneuropathy) and in proximal segments and radicular of the same, reason why its consideration within the spectrum of the GBS is justified.

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 [3,15]. 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 [5,10,15,17,18].

Reflex blink in GBS-Miller Fisher-usually presents with severe facial involvement.

Blink reflex affected uni or bilaterally.

Infectious agents that precede the onset of Miller Fisher syndrome (MFS), such as *C. jejuni*, provoke the formation of antibodies, for example, GQ1b that not only act on *C. jejuni*, but also on the outer part of the membrane of the lungs. Paranodal Schwann cells where these gangliosides (GQ1b) exist similar to those existing in the outer layer of *C. jejuni*, activating the complement with the development of vacuolization of the paranodal myelin of the oculomotor cranial nerves (III, IV and VI), roots, spinal motor and sensory nerves, mainly of the lower extremities, followed by disintegration of myelin phagocytosed by macrophages that invade the affected area and after infiltration with lymphocytes. The inflammatory reaction is rarely so severe that the axon that has lost its myelin may degenerate.

Most have IgG anti-GQ1b antibodies that always cross-react with GT1a (6%) syndrome Miller Fisher SGB variant Ataxia + areflexia ophthalmoplegia due to involvement of axonal sensory potentials. There is demyelination of cranial nerves and spinal roots. There is no axonopathy in the etiopathogenesis.

At the level of electroneurography and EMG in Miller Fisher Syndrome: it is usually found:

- Axonal sensory neuropathy.
- Persistence of the F. by proximal blocking of the roots.
- Areflexia: Enteric infection GQ1b anti-ganglioside antibodies that recognize epitopes expressed at the oculomotor nerve node. It is sharp.
- Course with axonal degeneration and there may be denervation.
- Course with instability to walk. Inability to wander Distal paresthesias. Diplopia Limitation for ocular mobility. Paresthesias both hands. MMSS and MMII weakness. Difficulty swallowing solids and liquids.
- Bilateral or unilateral facial paresis.
- There is usually a drop in sensitive potentials.

Asymmetric bilateral facial paresis. Antibodies in the oculomotor nerve node.

The Miller Fisher is usually self limited and of benign course. In this case the evolution was very good [17].

The facial nerve tends to drop in amplitude. And the blink reflex can be normal or show data from a PFP. In our patient there were clear data of left peripheral facial palsy.

F-wave persistence and temporal dispersion.

In all cases there was a marked reduction in distal sensory amplitude and frequent signs of axonal degeneration.

In Miller Fisher class Ig anti GQ 1b antibodies are present in up to 90% of cases.

These infectious agents, but also in the outer part of the membrane of paranodal Schwann cells where there are gangliosides (for example GQ1b) similar to those existing in the outer layer of these agents. These antibodies, by adhering to the gangliosides of the myelin sheath, especially in the axolema of Ranvier's nodules, cause the activation of the complement system, which in turn causes vacuolization of the paranodal myelin sheath, especially where these gangliosides they are more common: in the oculomotor cranial nerves (III, IV and VI), roots and spinal motor and sensory nerves (mainly of the lower extremities). This is followed by infiltration by macrophages attracted by the chemotactic components of complement, which adhere to the external aspect of the axolema and phagocytose the opsonized myelin. This opens the periaxonal spaces that are invaded by a greater number of macrophages, leading to axon retraction and in some cases to degeneration of the axon. Finally there is infiltration with lymphocytes. The agents mainly involved in the immune response are macrophages and T cells that cause areas of segmental demyelination often associated with signs of secondary axonal degeneration, which can be detected in small and large caliber nerves [5,10,15].

The end result of these autoimmune attacks on the nerves is the loss of myelin and as a consequence, the failure of both sensory and motor nerve conduction. An ischemic mechanism has also been proposed that could contribute to injury as a consequence of inflammatory edema of the nerve trunk when compressed with the inextensible epineurium and perineurium. Despite this, axonal function usually remains intact and recovery can be as rapid as remyelination occurs. However, if axonal degeneration is extreme, recovery occurs more slowly and there will be a greater degree of residual damage.

Serologically, in all the subtypes, there are variable degrees of different IgG and IgM antibodies against gangliosides of peripheral myelin or axolemum in Ranvier's nodules, although some predominate in one of the subtypes, as is the case with IgGantiGQ1b, which is elevated even in the 90% of patients with Miller Fisher syndrome (MFS).

Miller Fisher syndrome (MFS) was suspected and the necessary tests were ordered to confirm the diagnosis:

- ENG- EMG
- Lumbar puncture.
- MRI of the skull.
- Study of anti-ganglioside antibodies.

During admission, alterations compatible with Miller-Fisher syndrome were detected (ataxia with nystagmus in the lateral gaze, dysmetria and dysdiadochokinesia, areflexia and diplopia, although without frank ophthalmoparesis). MRI is performed, which is normal, and EMG and determination of antiganglioside antibodies with results compatible with the disease.

At discharge, the patient had two weeks of evolution, showing a progressive improvement in the last 4 - 5 days, with stable gait (although instability persists in tandem gait), without dysmetria or dysarthria and with occasional diplopia, which does not, however, prevent the reading, without having required specific treatment at any time given the mildness of the symptoms, which have not prevented ambulation at any time. Areflexia persists. It will follow reviews in CEX of Neuropediatrics. During admission, he presented occasionally

abdominal pain that has been attributed to constipation secondary to immobility and change of diet. No specific treatment is started at discharge.

At 6 months after the onset of the disease, after the onset of neurological symptoms, most patients have recovered from ataxia and ophthalmoplegia [4,18].

SMF is understood as a variant of GBS, predominantly in Asian-speaking countries. In the case presented here, it is a 10-year-old schoolgirl, with a history of gastrointestinal symptoms that preceded ataxia, ophthalmoplegia, diplopia 1 month before, which improves after 20 days, persisting instability in tandem gait. The most important diagnostic study is the serum determination of antiganglioside antibodies that in our patient were positive (Antiganglioside IgM and IgG: positive in blood and later in CSF) and that can be found in up to 90% of patients with MFS. This is a confirmed case of SMF in our hospital in Cartagena, Murcia. The diagnosis was made by the clinical examination of neurology, and the performance of laboratory studies, neuroimaging and ENG-EMG that allowed to rule out other entities within the differential diagnosis. confirmation was made by isolating the specific antiganglioside antibodies and the rest of the battery of tests.

Conclusion

Data suggestive of demyelinating sensory-motor polyradiculoneuropathy, in acute stage (10 days) with involvement of upper and lower limbs, absence of responses in the left side trigeminal-facial reflex. These findings are similar to those described in Millar-Fisher syndrome.

CMAP latency delay and slight drop in amplitude of the left facial. Regarding the right. Left facial paralysis.

Nerve conduction studies demonstrate demyelinating-type affection, electroneurography shows temporal dispersion of SNAPs, decreased amplitude of SNAPs, slowing of sensory and motor conduction speeds, CMAP motor conduction block, with prolongation of the distal latencies, as well as prolongation or absence of the F responses (which imply an affectation of the most proximal portions of the motor nerves and the motor roots) [18] the clinical characteristics added to the presence of anti-ganglioside antibodies of the Anti GQ1b type, and electroneurographic findings are associated with a diagnostic sensitivity of 90% [17,18].

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