RECURRENT MILLER FISHER: A NEW CASE REPORT AND A LITERATURE REVIEW.

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Introduction

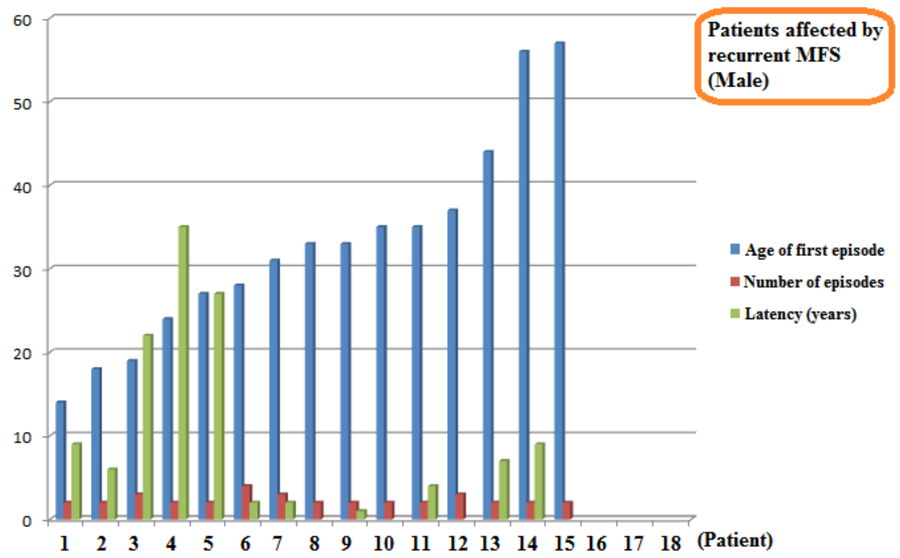
The clinical entity of ophthalmoplegia, ataxia and areflexia was first described by Collier and is named Miller Fisher syndrome (MFS) because of Fisher's report [1]. Besides the classical triad, additional signs and symptoms can accompany clinical condition such as sensory disturbances, bulbar palsy, limb weaknesses or even micturition disturbances [1].

Clinical Case

In 1998 a 37-year-old healthy woman developed, after four days of flu-like syndrome, diplopia, paraesthesias and gait instability. She presented ataxic gait, diffuse areflexia and left ophthalmoplegia. Brain MRI, neurophysiological evaluation and orbits ultrasound proved normal. Discharged from the hospital with diagnosis of anxiety disorder, in six months all symptoms remitted. In 2004 she newly experienced diplopia, left ptosis and gait instability. Considering previous diagnosis, she refused new neurological evaluations. Symptoms lasted 4 months and progressively improved. In 2010 patient again developed diplopia, ataxia and limb weakness, so she headed to hospital. All examinations, including brain MRI, neurophysiological evaluation, anti-AChR and anti-MuSK antibodies proved again normal. Subjected to a treatment with oral steroids for two months the symptoms gradually subsided.
The last episode, presenting with diplopia and generalized weakness, occurred on December 2014, two weeks after flu-like episode. Neurological examination showed bilateral ophthalmoplegia, right peripheral facial palsy, dysarthria, rhinolalia, dysmetria, ataxic gait and generalized areflexia with dysesthesias in four limbs. Miller-Fisher syndrome was diagnosed. Analysis of cerebrospinal fluid and neurophysiological examination were normal (Table 1). Serum anti-GQ1b antibodies were negative. Patient received therapy with IVIg (2g/Kg). Considering previous clinical episodes, she also started chronic therapy with prednisone (50mg/die). After three weeks there was complete clinical remission. Neurophysiological follow-up showed mild reduction of SNAP amplitude from lower limbs nerves and mild increase of mean F wave latency from upper and lower limbs (Table 1). Steroids was gradually tapered down after six months. Further neurophysiological evaluation showed normal results (Table 1). After two years clinical evaluation is still unremarkable.

Conclusion

Recent data show that recurrent MFS is higher than expected [1]. Anti-GQ1b in relapsing MFS are present in about 80% of patients, less commonly other antibodies [2]. Nerve conduction studies demonstrated in most of relapses no specific alterations, probably because they were made early in the disease course [3]. In our patient only a short-term follow-up study demonstrated mild alterations, and long-term examination was normal, underlying the importance of neurophysiological follow-up in these patients, such as clinical examination for diagnosis [4]. Up to now 34 cases of recurrent MFS were described (see graphics). Anti-GQ1b antibodies were found in 48% of cases; more than two relapses occurred in 19% of patients; 10 recurrent cases were treated with steroid with a good response in 88% of them (see graphics). IVIg or plasma exchange are used to treat MFS, and were successfully tested in recurrent MFS too [2, 6]. Steroid use is limited, but single reports suggest an efficacy in recurrent cases [4, 5]. In these, an overlap with CIDP with acute onset and relapsing-remitting course cannot be excluded [7]. Further studies are necessary to confirm this hypothesis.



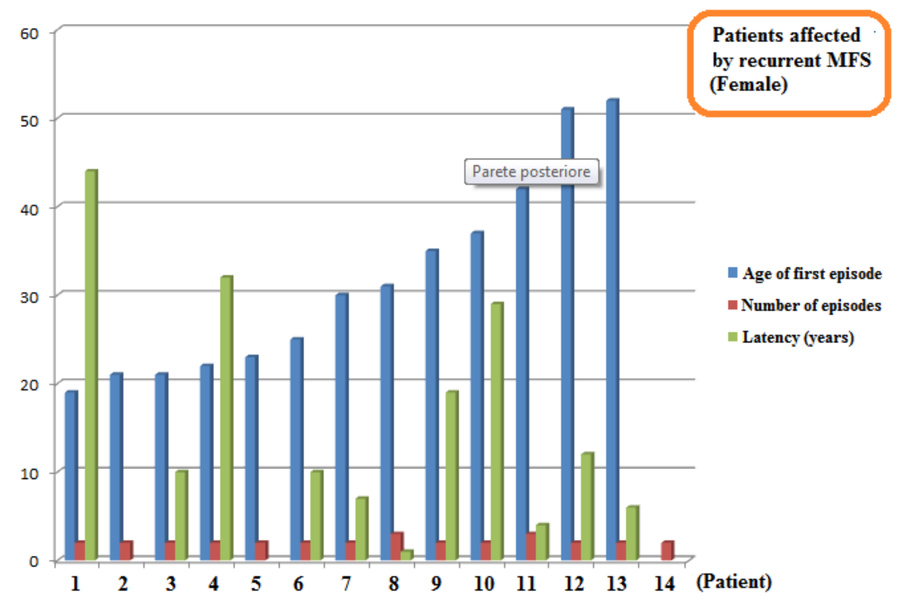
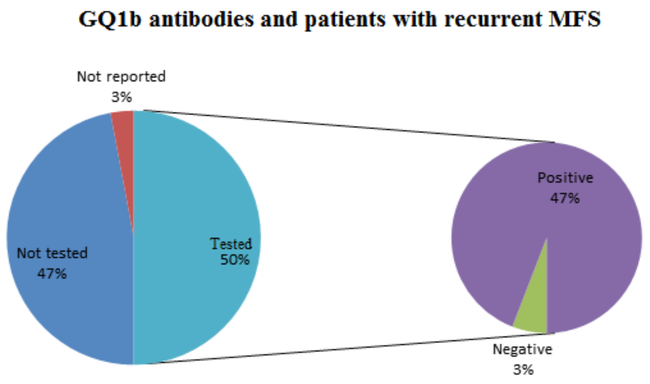
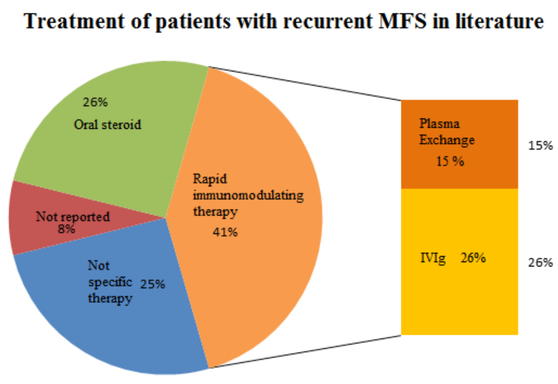


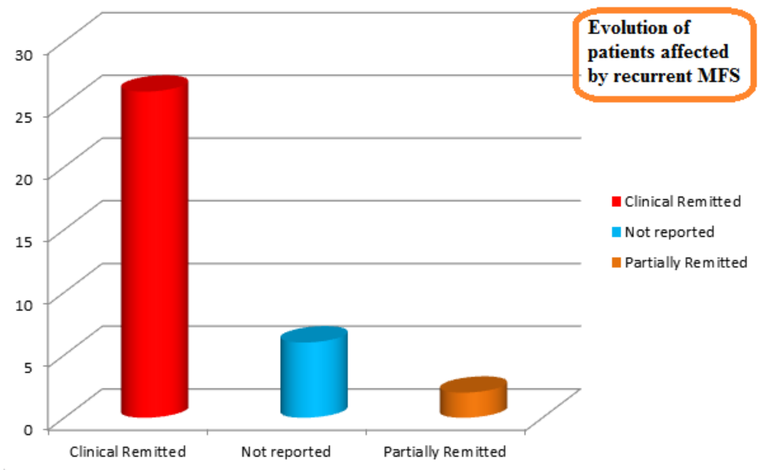
Table 1. Serial electrophysiological studies of the patient.

		First evaluation (admission)			Second evaluation (one month)			Third evaluation (six months)		
Nerve		*MCV (m/s)*	*dL (ms)*	*CMAP (mV)*	*MCV (m/s)*	*dL (ms)*	*CMAP (mV)*	*MCV (m/s)*	*dL (ms)*	*CMAP (mV)*
R Median	E-W	56	NE	8.0	54	NE	7.8	NE	55	8.0
	W-APB	NE	3.6	8.4	NE	3.5	8.0	NE	3.7	8.2
R Tibialis	LM-AH	NE	2.8	13.2	NE	3.1	15.6	NE	3.7	17.2
L Tibialis	LM-AH	NE	3.0	11.6	NE	3.5	13.8	NE	3.9	14.6
F wave			*mL (ms)*			*mL (ms)*			*mL (ms)*	
R Median			25.5			27.8			25.4	
R Tibialis			46.1			52.2			45.9	
L Tibialis			45.7			51.4			46.1	
Nerve		*SCV (m/s)*		*SNAP (µV)*	*SCV (m/s)*		*SNAP (µV)*	*SCV (m/s)*		*SNAP (µV)*
R Sural	A-SURA	68		20.0	43		7.1	56		14.5
L Sural	A-SURA	66		20.4	41		8.4	58		16.1
R. Peroneal	Lat. Leg-Ret	66		22	44		14.1	55		20.2

Legend: **MCV**, motor conduction velocity; **dL**, distal latency; **CMAP**, compound muscle action potential; **mL**, median latency; **SCV**, sensory conduction velocity; **SNAP**, sensory nerve action potential; **E**, elbow; **W**, wrist; **APB**, abductor pollicis brevis; **LM**, lateral malleolus; **AH**, abductor hallucis; **Lat**, lateral; **Ret**, retinaculum; **NE**, not examined.
Follow-up time of examination is given in brackets.







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