Variants of Guillain-Barré Syndrome: Miller Fisher Syndrome, Facial Diplegia and Multiple Cranial Nerve Palsies

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ABSTRACT: We report the experience at a large teaching hospital over a 10 year period with Miller Fisher Syndrome, facial diplegia, and multiple cranial nerve palsies. In these patients, absence of drowsiness on examination, normal cranial CT scans, albumino-cytological dissociation on CSF examination and slowing of nerve conduction, all suggest that a peripheral nerve dysfunction is the underlying mechanism. Pertinent literature is reviewed, in an attempt to separate these probable variants of Guillain-Barré Syndrome from brainstem encephalitis, with which they may be confused.

RÉSUMÉ: Variantes du syndrome de Guillain-Barré: le syndrome de Miller-Fisher, la diplégie faciale et les paralysies multiples des nerfs crâniens. Nous rapportons l'observation de patients atteints du syndrome de Miller-Fisher, de diplégie faciale et de paralysies multiples des nerfs crâniens, ayant consulté dans un hôpital d'enseignement sur une période de dix ans. Chez ces patients, l'absence de somnolence à l'examen, un CT-scan crânien normal, une dissociation albumino-cytologique à l'examen du LCR et une conduction lente à l'étude de la conduction nerveuse, suggèrent que la dysfonction au niveau du nerf périphérique en est le mécanisme sous-jacent. Nous revoyons la littérature pertinente, en tentant de séparer ces variantes probables du syndrome de Guillain-Barré de l'encéphalite du tronc cérébral, entité avec laquelle elles peuvent être confondues.

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The syndrome of ataxia, areflexia and ophthalmoplegia (AAO), initially described by Bogaert et al in 1938,[1] became widely recognized after Miller Fisher's classic description of three patients with AAO in 1956.[2] Often called Miller Fisher syndrome (MFS), it is considered rare and mentioned only in passing in most neurological text books.[3-6]

The syndrome of multiple cranial nerve palsies, as a variant of Guillain-Barré Syndrome (GBS) was initially described by Guillain in Brussels in 1937,[8] but has only rarely been since reported.[9] Facial diplegia with minimal associated limb weakness or sensory deficits, another reversible neuropathy with a good prognosis has also been considered a variant of GBS.[10]

The pathogenesis of MFS has traditionally been considered to be similar to that of GBS[2,11,12,13] as most patients have a preceding viral illness, albumino-cytological (A-C) dissociation on cerebrospinal fluid (CSF) examination, and a favorable outcome. This view has however recently been challenged by reports that consider brainstem encephalitis as the underlying cause for the AAO.[14,15]

We report here our analysis of patients admitted over a ten year period with AAO, multiple cranial nerve dysfunction and facial diplegia. Our results will be compared with reports from the literature where the underlying etiology was considered to be brainstem encephalitis in an attempt to separate two distinct entities with different underlying mechanisms and prognosis.

METHODS

We reviewed the charts of all patients admitted to the Foothills Hospital (Calgary, Alberta) between 1975 and 1985 with a diagnosis of GBS,[7] infectious polyneuropathy, MFS, or multiple cranial nerve palsies. The cases were included if there was no primary cause for neuropathy such as diabetes or intoxications, and if the patients recovered with a minimum of sequelae. A-C dissociation and slow nerve conduction velocities were seen in some patients, and were helpful in making a diagnosis, but patients with normal CSF protein and normal nerve conduction

velocities were not excluded if the clinical course was typical. Drowsiness or signs of upper motor neuron involvement were not present in any of our cases. Out of a total of 60 patients, 15 met diagnostic criteria for MFS, multiple cranial nerve dysfunction or facial diplegia and are presented here. The remaining 45 patients, all with a diagnosis of GBS, will not be discussed further. These 15 patients were divided into the following three groups.

1) AAO, with minimal weakness and/or sensory symptoms (MFS).

2) Facial diplegia, ataxia and areflexia.

3) Multiple cranial nerve dysfunction, with or without associated ataxia or areflexia.

PATIENT ANALYSIS

1) Ataxia, Areflexia and Ophthalmoplegia

Seven patients had AAO. Clinical features are shown in Table 1A. Ataxia and ophthalmoplegia were present in all cases, but the degree of ophthalmoplegia was quite variable. One patient had bilateral ptosis only, 3 patients showed incomplete third nerve palsies, two patients had involvement of third and sixth nerves and one patient showed complete external ophthalmoplegia. Pupillary involvement was noted in two cases. In addition to ataxia, intention tremor was said to be present in one case and truncal titubation was seen in one patient. Preced-

Table 1A: Patients with Ataxia, Areflexia and Ophthalmoplegia: Clinical Features

Patient No.	Preceding Viral Illness	Ophthal-moplegia	Other Cranial Nerve Involve-ment	Gait Ataxia	Deep Tendon Reflexes Absent
1.	URTI	+	—	+	Hyporeflexia
2.	—	+	—	+	+
3.	URTI	+	—	+	+
4.	URTI	+	7, 9, 10	+	+
5.	URTI	+	—	+	+
6.	URTI	+	—	+	+
7.	URTI	+	—	+	Hyporeflexia

URTI (Upper Respiratory Tract Infection)
(—)   (Absent)

Table 1B: Laboratory Investigations

Patient No.	CSF Protein (GMS/L)	CSF WBCs (Cells/mm^3)	CT	EEG	Nerve Conduction Study
1.	1.40	5	Normal	—	Absent H-Reflex
2.	.69	0	Normal	—	Slowed-Conduction
3.	1.80	9	Normal	—	Slowed-Conduction
4.	.42	1	—	—	Slowed-Conduction
5.	2.25	2	—	—	Normal
6.	.28	0	Normal	Normal	—
7.	2.00	0	Normal	—	Absent H-Reflex

(—) Not done

ing viral illness was present in six patients. Reflexes were absent in five patients and decreased in two patients. Involvement of cranial nerves other than 3, 4, & 6 was seen in only one patient. No patients had upper motor neuron signs and none of the patients were drowsy. Hospital stay was less than 15 days in all cases, and all were showing signs of recovery prior to discharge. All patients were followed for at least 2-3 months, and during this time no relapses occurred.

Results of nerve conduction studies varied widely from patient to patient. Two patients showed normal motor and sensory conduction velocities, but H reflexes were absent. Three patients showed slowed nerve conduction velocities. These ranged from very mild in some patients to motor conduction velocities as low as 27 meters per second in others. Sensory action potential amplitudes in the median and ulnar nerves were markedly reduced in several patients.

Cranial computed tomography (CT) was done in five patients and was normal in all. CSF examination (Table 1B) showed only minimal white blood cell count elevations, but CSF protein was increased in five patients. An EEG was done in only one patient, and was normal.

2) Ataxia Areflexia and Facial Diplegia

There were three patients in this group (Table 2A). Preceding viral illness was present in only one patient. Ataxia and areflexia or hyporeflexia were present in all patients. The reason for neurological consultation was facial diplegia in two cases and ataxia in the third. Symptoms developed over three days in two patients and ten days in the third. In the only case where the facial diplegia was not symmetrical, paralysis began first in the more severely involved side followed by facial weakness on the opposite side three days later. Facial weakness was of lower motor neuron type in all cases and taste was affected bilaterally in one patient. Reflexes were absent in one patient, and markedly reduced in the other two. Muscle strength was normal in all patients on admission, but two patients later developed mild hip flexor weakness. Distal lower limb numbness was present in one patient and this patient also showed decreased position and vibration sense in the feet. Romberg's sign was absent in all patients. Limb ataxia was absent in two patients, and present to a very mild degree in the third.

Hospital stay was less than ten days in all cases, with some recovery by the time of discharge. Two patients had returned to normal within 12 weeks, while the third patient required bilateral blepharoplasty for persistent facial palsy one year later. CSF protein (Table 2B) was increased markedly in all three patients, and CSF white blood cell counts were not increased. Nerve conduction studies were done during the acute illness in

Table 2A: Patients with Ataxia, Areflexia and Facial Diplegia: Clinical Features

Patient No.	Preceding Viral Illness	Facial Diplegia	Ataxia	Deep Tendon Reflexes Absent
1.	URTI	+	+	+
2.	—	+	+	Hyporeflexia
3.	—	+	+	Hyporeflexia

URTI (Upper Respiratory Tract Infection)
(—)   Not present

two patients, and these patients both showed absent or markedly reduced sensory action potential amplitudes in the median and ulnar nerves, and moderately slowed motor nerve conduction velocities. Nerve conduction studies were done only at 18 months after clinical presentation in the third patient, and both motor and sensory conduction studies were normal. EMG studies in this patient however were abnormal, with reduced motor unit recruitment and many large polyphasic motor unit potentials in the left tibialis anterior, left vastus medialis, and left deltoid muscles. Brain CT scan was normal in the one patient in whom this test was performed.

3) Multiple Cranial Nerve Palsies with or without Ataxia or Areflexia

This group consisted of five patients (Table 3A). A preceding viral illness was present in one patient. Symptoms were variable, with most patients having at least four cranial nerves involved. The third cranial nerve (CN) was affected in four of five cases, and involvement varied from decreased adduction to complete paralysis of all muscles supplied by this nerve. The seventh CN was affected in four cases, and the severity of involvement varied from mild to almost complete paralysis of all facial muscles. Bilateral involvement was present in three cases.

The fourth CN and sixth CN were each involved in three patients. The twelfth CN was affected in three cases, with bilateral involvement in two of these. The eighth CN was affected in two patients, with both showing only unilateral involvement. Both patients showed hearing loss without dizziness. The fifth CN was affected in two patients and this involvement was unilateral in both cases. The eleventh CN was affected in only one patient, with severe bilateral involvement. Muscle strength in the limbs was normal in all patients. Sensory examination revealed decreased light touch in one patient and decreased vibration sensation in another. Two patients were ataxic and two were hyporeflexic. No patients showed evidence for upper motor neuron signs or drowsiness.

Table 2B: Laboratory Investigations

Patient No.	CSF Protein (GMS/L)	CSF WBCs (Cells/mm³)	CT	EEG	Nerve Conduction Study
1.	1.20	1	—	—	Abnormal
2.	5.20	0	Normal	—	Abnormal
3.	1.52	0	—	—	Abnormal

(—) Not done

Table 3A: Multiple Cranial Nerve Palsies: Clinical Features

Patient No.	Preceding Viral Illness	Cranial Nerves Involved	Ataxia	Deep Tendon Reflexes	Strength (Limbs)	Sensory Signs
1.	—	3,4,6,7	+	Normal	Normal	Normal
2.	—	3,4,6,10,11	—	Normal	Normal	+
3.	—	3,5,7,8,12	—	Decreased	Normal	+
4.	+	3,4,5,6,7,12	+ +	Decreased	Normal	Normal
5.	—	5,7,8,12	—	Normal	Normal	Normal

(—) Not present

The CSF was examined in four patients, and all showed increased CSF protein (Table 3B). CSF white blood cell count elevations were minimal except in one patient with a traumatic lumbar puncture. Brain CT scans were done in four patients and were normal. Nerve conduction studies were done in three patients and were normal except for absent H reflexes in one patient.

Time from initial symptom to maximum disability varied from two to seven days, and the duration of hospital stay was one to five weeks. At the time of discharge, all patients were showing significant improvement. A follow-up was obtained in four patients. Three patients had returned to normal within three month's of discharge from hospital. One patient developed multiple myeloma one year after discharge, and showed mild bilateral facial weakness and mild unilateral tongue weakness three years after discharge.

DISCUSSION

Although Miller Fisher syndrome is characterized by the triad of Ataxia, Areflexia, and Ophthalmoplegia, cases reported in the literature frequently show involvement of other cranial nerves in addition to 3, 4, & 6[2,12,13] and occasionally also show mild weakness and sensory changes in the limbs.[2,11,16] In these patients with more extensive neurological involvement, the diagnosis of MFS is made because these associated symptoms are over-shadowed by the AAO.

Ophthalmoplegia has been considered essential for the diagnosis of MFS, but, as in our patients, has varied from bilateral ptosis only[17] to complete external and internal ophthalmoplegia.[2,13,18-20,30] Some reported clinical findings might suggest brainstem lesions. These include the preservation of Bell's phenomenon despite paralysis of upward gaze,[2,11,15,19,21,22] mild ptosis in the presence of severe ophthalmoplegia[2,15,19,22] and horizontal dissociated nystagmus.[15,19,23,24,25] As similar disorders of eye movement have however been reported in conditions that do not affect the brainstem directly, including myasthenia gravis, botulism, intracranial aneurysm and pituitary tumor; Ropper has stated that all the eye movement disorders seen in MFS can be explained by cranial nerve dysfunction.[26] Furthermore, it is difficult to conceive that inflammation could involve multiple cranial nerves in the brainstem and totally spare adjacent structures,[27] so that no changes occur in level of consciousness.

The ophthalmoplegia that occasionally occurs with typical GBS is clinically indistinguishable from that reported with MFS,[28]

Table 3B: Laboratory Investigations

Patient No.	CSF Protein (GMS/L)	CSF WBCs (Cells/mm³)	CT	EEG	Nerve Conduction Study
1.	.56	5	—	—	Absent H-Reflex
2.	.64	1	Normal	—	Normal
3.	.76	0	Normal	—	—
4.	1.50	26*	Normal	—	Normal
5.	—	—	Normal	—	—

(—) Not done
* CSF Bloodstained (Traumatic Tap)

and some patients present with AAO only to develop profound peripheral weakness later.[29] A recent autopsy report of a patient with GBS and ophthalmoplegia showed no evidence of brainstem inflammation or demyelination, but did show widespread demyelination of cranial nerves.[30] The prominent ataxia seen in MFS has been considered by some to be due to lesions in cerebellar outflow pathways in the brainstem.[14] In his original report Miller Fisher considered a "unique and widespread attack on the sensory neurons underlying postural adjustment", to be responsible for the ataxia,[2] and recent physiological studies have shown evidence for a selective Ia afferent nerve fiber dysfunction in MFS.[13] A disparity between proprioceptive information from muscle spindles and kinesthetic information from joint receptors has also been suggested as a cause for the underlying ataxia.[32] Although involvement of sensory tracts in the spinal cord has been reported in autopsy series of GBS[33] including "ataxic" GBS,[34] these changes may all be due to severe involvement of spinal nerve roots. In our experience, the ataxia in MFS improves quickly, with almost complete recovery in 5 of our 7 cases at the time of discharge from hospital within 15 days of admission.

Five of our patients with MFS had abnormal nerve conduction studies. Similar findings have been reported previously[12,13,23,35,36] and support the hypothesis that the pathogenesis of MFS is similar to that of GBS.

Normal nerve conductions have also been reported in MFS,[37] but this does not rule out peripheral nerve involvement as the underlying mechanism as 9 to 41% of patients with typical GBS also show normal nerve conduction studies.[38]

In addition to MFS, other variants of GBS likely share a similar pathogenesis. Reports of multiple cranial nerve dysfunction as a variant of GBS have been infrequent.[9] Our 5 patients with multiple cranial nerve dysfunction, showed many features in common with GBS. All patients remained alert, 4 of 5 had A-C dissociation in the CSF, and all showed a good recovery where follow-up was available. Cranial nerves 3, 4 & 6 were the most frequently involved nerves, similar to MFS, but only 2 out of 5 cases showed ataxia. In contrast to MFS, 3 out of 5 patients showed normal stretch reflexes. All of our 5 patients had 4 or more cranial nerves involved, and with the exception of one patient, involvement was always bilateral.

Our 3 patients with facial diplegia, ataxia and areflexia may represent a further variant of GBS. Two patients showed slowed nerve conduction velocities, and a third patient, studied eighteen months after admission, showed neurogenic changes on EMG studies. All showed a marked increase in CSF protein levels. A brain CT scan done in one patient showed no evidence of CNS involvement. Although ophthalmoplegia was not present, the ataxia and areflexia in the absence of marked sensory abnormalities and weakness might suggest a relationship to MFS. Of interest, in patients with MFS with involvement of additional cranial nerves, the 7th cranial nerve is the one most frequently involved.[23] The favourable outcome in all 3 patients is in keeping with peripheral neuropathy, as the underlying mechanism. To our knowledge, such patients have not been reported previously.

As in most previous reports, none of our 7 patients with MFS died, reflecting the good prognosis for this syndrome. As a result we have no autopsy data. A previously reported autopsy of a patient with MFS showed no evidence for lesions in the brainstem or cerebellum.[31] Cranial nerves, 7, 9 and 11 showed

demyelination and lymphoctyic infiltration. Unfortunately only the proximal portions of cranial nerves 3, 4 and 6 were available for neuropathological examination and were normal, although more peripheral pathology in these nerves could not be excluded. Brainstem pathology with demyelination and lymphocytic infiltration has been described in patients diagnosed or referred to later as MFS, but a careful review of these cases suggests that their clinical findings were not typical for MFS. In the 2 patients with inflammatory cells, astrocytic proliferation, and swelling in the brainstem as autopsy reported by Bickerstaff[39] and Al-Din et al[14] the clinical findings prior to death included drowsiness, headache, confusion and extensor plantar responses in one patient and drowsiness, headache, confusion, incontinence of urine and extrapyramidal tremor in the other. Another autopsy report of a patient with ophthalmoplegia by Bignami and Servi[40] showed swelling and chromatolysis of cells in the 3rd cranial nerve nucleus, but such changes may have been secondary to axonal damage in the 3rd cranial nerve peripheral to the brainstem. The 3rd cranial nerve was described as edematous and showed perivascular lymphocytic infiltration. This patient had involvement of multiple cranial nerves (3, 4, 6, 7, 10 and 12) and no mention was made of ataxia or areflexia, so he should not be referred to as a case of MFS.

In the 18 clinical cases reported by Al-Din[14] it is worth noting that 4 patients with AAO who showed no drowsiness or upper motor neuron signs (cases 3, 5, 15, 16) also showed where available normal CT scan and EEG findings. All 4 cases recovered. However, patients that were drowsy (cases 1, 2, 4, 6, 7, 8, 9, 10, 11, 12, 13, 14, 17) or had normal or brisk reflexes (cases 1, 4, 7, 8, 10, 12) associated with some cases with Babinski's sign showed abnormal EEGs where this test was done, and some in addition showed brain CT scan abnormalities. Many of these patients also recovered completely. We would suggest that this report may be dealing with two separate groups of patients, one group with typical MFS resulting from peripheral nerve pathology, and another group with brainstem encephalitis with or without additional peripheral nerves involvement. Clinically, however, it may be impossible to completely separate the two groups, as illustrated by case 18 in the series by Al-Din et al.[14] This patient had AAO with A-C dissociation without drowsiness or long tract signs, but her brain CT scan and EEG were both abnormal. A patient with multiple brain metastases who presented with facial diplegia, oculomotor paresis, ataxia and spinal fluid A-C dissociation without drowsiness has also been reported.[41] Barontini and Sita[15] reported EEG abnormalities in 2 of 6 cases with presumed MFS. Both patients with EEG abnormalities were drowsy and one had an abnormal CT scan as well. Abnormal EEGs suggestive of brainstem pathology have however been reported in children with otherwise typical MFS.[23] The significance of this finding is unclear.

According to Bickerstaff,[42] the clinical features of brainstem encephalitis include prominent early drowsiness, headache and CSF pleocytosis. Parkinsonism or pseudobulbar symptoms are seen frequently during recovery. All these findings are absent in most patients with typical MFS. Patients reported by Al-Din et al[14] and Barontini and Sita[15] who were drowsy or had upper motor neuron signs may in fact have had brainstem encephalitis. Grouping together patients with typical MFS and patients with signs and symptoms more suggestive of brainstem encephalitis can only lead to confusion. We would suggest that the term MFS be restricted to patients with AAO (complete or incomplete)

without drowsiness or upper motor neuron signs. In our experience CT scans are usually normal in such patients, and CSF shows A-C dissociation. These patients have an excellent prognosis, with no deaths attributable to this syndrome to our knowledge.

Patients with involvement of other cranial nerves but without drowsiness or upper motor neuron signs also have a good prognosis, and show clinical, laboratory, and radiological findings similar to GBS. These patients may or may not show ophthalmoplegia, ataxia or areflexia. We speculate that the pathological changes in these patients may also be restricted to the peripheral nerves, the cell bodies of neurons with axons in peripheral nerves, and the central processes of peripheral sensory neurons. These patients are in many ways similar to MFS, and whether or not they should be included in this nosological entity is a matter of debate. In the past many authors have included such patients within the spectrum of MFS.[9,10] Juncos and Beal,[43] however, reported 14 patients with idiopathic multiple cranial nerve palsies and concluded that only one of their patients might have had an "oligosymptomatic" form of GBS. Because many of their patients presented with facial pain and responded to corticosteroid treatment, these authors concluded that their patients had a condition related to Tolosa-Hunt syndrome. It is of interest that, similar to our patients with multiple cranial nerve palsies, the oculomotor nerves were the cranial nerves most commonly involved, followed by cranial nerves 5 and 7.

Patients with drowsiness or upper motor neuron signs on admission in addition to AAO and ataxia frequently show EEG and CT scan abnormalities. These patients likely represent examples of brainstem encephalitis. The prognosis in such patients may not be as favorable as in MFS. From our experience and a review of the literature we conclude that MFS and brainstem encephalitis, both of which may cause ophthalmoplegia and ataxia, are two distinct syndromes. Failure to recognize this had lead to confusion and controversy regarding the underlying pathological mechanisms.

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