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Case Report

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Rapid Resolution of Delayed Facial Palsy in Miller Fisher Syndrome With Steroid Therapy

Nithisha Thatikonda[a, b], Alexandru Lerint[a], Vijaya Valaparla[a], Chilvana Patel[a]

Abstract

Miller Fisher syndrome (MFS), a variant of Guillain-Barre syndrome (GBS), is characterized by the classic triad of ataxia, areflexia, and ophthalmoplegia. Approximately 20% of MFS patients experience facial weakness, with a subset developing delayed facial palsy (DFP) after other neurological symptoms have peaked or begun to improve. Initially, DFP was considered a natural progression of MFS, leading to recommendations against additional treatment. However, DFP persisted for more than 50 days without additional treatment in some patients, prompting additional steroid therapy, resulting in quicker resolution of DFP. We describe an MFS patient who presented with the classic triad of MFS and subsequently developed DFP. The patient was treated with methylprednisolone pulse therapy (1,000 mg/day for 3 days) followed by oral prednisolone (60 mg/day) with a gradual taper, resulting in rapid and complete resolution of DFP, suggesting an alternative mechanism behind DFP, opening avenues for further research and insights into this matter. MFS-DFP is rarely reported in the literature. In addition to this case, we aim to provide a comprehensive literature review on MFS-DFP, to further expand the existing knowledge on the current concepts of DFP-MFS.

Keywords: Delayed facial palsy; Miller Fischer syndrome; Methylprednisolone

Introduction

Miller Fisher syndrome (MFS) is a variant of Guillain-Barre syndrome (GBS), characterized by the triad of ataxia, areflexia, and ophthalmoparesis [1]. The global incidence of GBS is approximately 1 - 2 per 100,000 annually, with MFS accounting for about 5% of GBS cases in Western countries and 15-25% in Asian countries. MFS can affect individuals of all ages, with a slight male predominance [1-3]. The primary risk factor for MFS is a preceding infection, typically of the upper respiratory tract or gastrointestinal system. The most common pathogens involved are *Campylobacter jejuni* and *Haemophilus influenzae* [1, 3]. These infections trigger an immune response through molecular mimicry, leading to the production of autoantibodies against the GQ1b ganglioside found in the myelin sheath, particularly affecting the oculomotor nerves (III, IV, and VI cranial nerves) and dorsal root ganglia, resulting in myelin sheath destruction [1, 2].

MFS is generally self-limiting, with treatment aimed at accelerating recovery and preventing neuromuscular respiratory failure [4, 5]. The main treatments are plasmapheresis and intravenous immunoglobulin (IVIG), both of which are effective in reducing disease duration [1, 2, 4, 5]. Although no studies have compared the effectiveness of these treatments directly, the choice often depends on side effect profiles and administration methods [4]. The prognosis for MFS patients is typically excellent, with symptoms resolving within 6 months, and relapses occurring in less than 3% of cases [4, 5].

Beyond the symptoms of clinical triad, a variety of other symptoms and signs have been reported in MFS [2]. About 20% of MFS patients experience facial weakness, with a few developing delayed facial palsy (DFP) after other neurological symptoms reach nadir or start to improve [2-5]. DFP, reported in 6-16% of MFS cases, is defined as facial palsy occurring 5 - 21 days after symptom onset [6, 7]. Initially, DFP was considered a natural progression of MFS, leading many earlier reports to advise against additional treatment [3, 6-13]. However, more recent studies challenge this view, suggesting that facial palsy should only be considered part of MFS progression if it occurs alongside other symptoms [14-18]. In some patients, DFP, although self-limiting, persisted for more than 50 days without additional treatment [3, 11-13]. This persistence prompted some authors to treat DFP with pulse dose steroids followed by a prednisone taper, resulting in a quicker DFP resolution [17, 18]. It remains unclear whether DFP in MFS patients is due to a shared immunological phenomenon [3, 6, 7], a secondary effect of MFS treatment [14], or simply a coincidence. This case report describes an MFS patient with DFP who benefited from steroid therapy, reviewing existing literature and potential underlying mechanisms of DFP.

Case Report

A 37-year-old man presented with a 1-week history of diplopia, dysphagia, and unsteadiness following an upper respirato-

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[a]Department of Neurology, University of Texas Medical Branch Galveston, Galveston, TX 77550, USA
[b]Corresponding Author: Nithisha Thatikonda, Department of Neurology, University of Texas Medical Branch Galveston, Galveston, TX 77550, USA. Email: nithatik@utmb.edu

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ry tract infection (URTI). On presentation, neurological examination revealed bilateral ophthalmoplegia (more pronounced in horizontal eye movements), dilated and sluggishly reactive pupils, ataxia, diffuse areflexia, and dysesthesias in all four limbs, but no limb muscle weakness or facial weakness. Cerebrospinal fluid (CSF) analysis showed white cell count of 2/μL (reference range 0 - 5/μL) and protein level of 31 mg/dL (reference range 15 - 45 mg/dL) without albumino-cytologic dissociation. Active infection was ruled out with negative viral respiratory pathogens panel, CSF viral meningitis/encephalitis panel, gram stain and bacterial cultures from both CSF and blood samples. Serum IgG antibodies against gangliosides GQ1b (3200) and GD1b (3200) confirmed MFS. He began (IVIG therapy (0.4 g/kg/day for 5 days) on the day of presentation. By day 4 of admission, his symptoms of dysphagia and ophthalmoplegia began to improve. However, on day 7 of his admission, he developed left lower motor neuron type facial palsy. Other neurological symptoms did not worsen. Additional tests for connective tissue disorders, mycoplasma, cytomegalovirus (CMV), varicella zoster virus (VZV), herpes simplex virus (HSV), Epstein-Barr virus (EBV), human immunodeficiency virus (HIV), hepatitis C virus (HCV), Lyme titers, hemoglobin A1c, and angiotensin-converting enzyme (ACE) levels were negative. Brain magnetic resonance imaging (MRI) revealed no abnormalities. He received methylprednisolone pulse therapy (1,000 mg/day for 3 days) starting on day 7, followed by oral prednisolone (60 mg/day) with gradual tapering over 3 weeks. His facial palsy improved by day 12 and resolved completely by day 15. He was discharged on admission day 15 with improving ataxia, ophthalmoplegia, and dysesthesias. At a follow-up visit 2 months later, he was asymptomatic except for mild dysesthesias in his lower limbs.

Discussion

We present a patient who initially exhibited the classic triad of MFS and subsequently developed peripheral type-DFP as other neurological symptoms began to improve. Remarkably, treatment with pulse dose steroids followed by an oral prednisone taper led to a rapid and complete resolution of DFP. This outcome is particularly intriguing, given that steroids are typically considered ineffective and potentially harmful in cases of GBS and its variants.

DFP is characterized by the onset of facial weakness after other neurological symptoms have reached nadir or begun to improve. A recent study further defined DFP as facial palsy occurring between 5 and 21 days after symptom onset [7]. Our patient reached the nadir of symptoms by day 9, showed improvement in ataxia and ophthalmoplegia by day 11, and developed facial palsy by day 14, meeting the criteria for DFP.

The reporting of DFP dates back to initial description of MFS, where one patient developed unilateral facial palsy after resolving ataxia on day 13 of the illness [8]. Since then, numerous case reports and small case series have documented DFP in MFS, with an incidence ranging from 6% to 16% [6, 8]. We reviewed the existing literature on DFP in MFS and summarized the clinical features of previous reports alongside our current patient (Table 1) [3, 6-18]. Some studies have suggested that the development of DFP may be attributed to plasmapheresis, which could reactivate certain immunological mechanisms in MFS [14, 19]. However, our patient did not receive plasmapheresis, consistent with other studies reporting DFP in MFS patients who also did not undergo plasmapheresis [7, 11, 12]. This raises the possibility that DFP may be an inherent part of MFS pathophysiology due to unclear reasons, warranting further discussion. To date, there have been no reported cases attributing DFP to treatment with IVIG.

The precise mechanism of DFP in MFS remains unclear. A "descending paralysis pattern", where involvement of cranial nerves III, IV, and VI leads to ophthalmoplegia before facial palsy, was described by Kim et al [7]. This pattern is supported by the higher distribution of GQ1b-type ganglioside in the oculomotor axolemma compared to the facial nerve, making ophthalmoplegia the most frequent symptom of MFS [7, 20]. Immunohistochemical studies with anti-GQ1b monoclonal antibody also show prominent staining in the oculomotor, trochlear, and abducens nerves, but not the facial nerve [5, 21]. However, this does not explain the presence of bulbar symptoms at disease onset in our patient. Moreover, bulbar symptoms have been reported in 17-26% of MFS patients, regardless of DFP occurrence [2, 4]. Some studies have also found an association between positive anti-GQ1b antibody and isolated cranial neuropathies without the classic MFS triad [22]. This suggests a mechanism beyond the disproportional distribution of anti-GQ1b antibody among cranial nerve axolemmas.

The existence of other undetectable antibodies is another explanation, especially in seronegative MFS patients [7, 11]. One study found that two patients with typical MFS were positive for IgG anti-LM1 (lipid-modified) antibodies [23]. It has also been suggested that other antibodies against ganglioside complexes exist in MFS patients who are seronegative [24, 25]. Therefore, another undetectable antibody, other than anti-GQ1b, might be associated with MFS pathophysiology [7, 23-25].

Another explanation for DFP involves conduction block and the facial nerve's length. A study using transcranial magnetic stimulation found conduction block in the proximal segments of the facial nerve in MFS patients without facial palsy [26], suggesting an initial subclinical conduction block that progresses to facial palsy when a threshold is reached. Kim et al proposed a "dying-forward" pattern starting from proximal facial nerve lesions. Given the facial nerve's length (approximately 5.5 cm from the internal acoustic meatus to the stylomastoid foramen), it takes longer to affect the entire nerve, delaying the onset of clinical symptoms. This suggests that clinically apparent facial palsy may take more time to develop [3, 26].

Current guidelines and opinions recommend against treating DFP in MFS due to its spontaneous resolution [3, 8, 9, 11-13, 26]. Previous case reports show untreated DFP resolves in 21 - 85 days (patients 6.1 - 6.3, 13.1 in Table 1). Some authors prefer treating DFP with additional immunotherapy. For instance, a study reported a patient (patient 14.1 in Table 1) who received an extra dose of plasmapheresis, resolving DFP in 45 days, similar to untreated cases [14]. In an another study, five out of 11 patients with DFP were treated with repeat IVIG,

Table 1. Summary of Current and Previous Case Reports on the Clinical Features of Delayed Facial Palsy in Miller Fisher Syndrome

Case	Age/sex	Antecedent illness	Immunotherapy/time of initiation (days)	Nadir of symptoms (days)	Improvement in symptoms (days)	Peripheral type-facial palsy			Additional therapy	Antibody profile
						Appearance (days)	Resolution (days)	Side		
Our case	37/M	URTI	IVIG/7	9	11	14	22	U/l	oPSL	GQ1b
Tan et al [3]										
3.1	52/M	N/a	IVIG/7	9	12	14	66	U/l	-	Negative
3.2	45/M	URTI	PLEX/6	6	14	12	35	B/l	-	GQ1b, GT1a GD1b
3.3	21/F	URTI	N/a	4	7	8	64	B/l	-	GQ1b, GT1a
3.4	57/F	URTI	PLEX/6	6	9	14	43	U/l	-	GQ1b, GT1a
Tatsumoto et al [6]										
6.1	35/M	URTI	N/a	5	6	15	21	U/l	-	GQ1b
6.2	32/M	URTI	IVIG /N/a	6	10	10	21	B/l	-	GQ1b
6.3	18/M	URTI	IVIG/N/a	6	8	10	21	B/l	-	N/a
6.4	29/F	URTI	N/a	10	18	11	21	U/l	-	GQ1b
Kim et al [7]										
7.1 - 7.11 (mean values for 11 cases)	34/(M: 7; F: 4)	URTI: 6; diarrhea:4	IVIG/N/a	10	13	12	<30	U/l in 4; B/l in 7	IVIG in five patients	GQ1b in 7
Fisher et al [8]										
8.1	45/M	URTI	-	5	8	13	42	U/l	-	Negative
Umekawa et al [9]										
9.1	47/M	N/a	IVIG/4	6	11	14	N/a	B/l	-	GQ1b
Yamamoto et al [10]										
10.1	55/M	URTI	IVIG/6	6	11	13	42	U/l	-	GQ1b
Jung et al [11]										
11.1	23/M	N/a	IVIG/N/a	7	11	16	60	B/l	-	N/a
11.2	50/M	N/a	IVIG/N/a	4	7	10	60	U/l	-	GQ1b
11.3	57/M	N/a	IVIG/N/a	9	20	15	60	U/l	-	N/a
Kwon et al [12]										
12.1	25/M	N/a	IVIG/6	N/a	N/a	12	42	U/l	-	N/a
12.2	48/F	N/a	IVIG/7	N/a	N/a	16	84	U/l	-	N/a
12.3	52/M	N/a	IVIG/3	N/a	N/a	11	84	B/l	-	N/a
12.4	56/M	N/a	IVIG/3	N/a	N/a	8	84	U/l	-	N/a
Port et al [13]										
13.1	20/F	N/a	IVIG/N/a	N/a	N/a	10	85	B/l	-	GQ1b
Chida el al [14]										

Table 1. Summary of Current and Previous Case Reports on the Clinical Features of Delayed Facial Palsy in Miller Fisher Syndrome - *(continued)*

Case	Age/sex	Antecedent illness	Immunotherapy/time of initiation (days)	Nadir of symptoms (days)	Improvement in symptoms (days)	Peripheral type-facial palsy			Additional therapy	Antibody profile
						Appearance (days)	Resolution (days)	Side		
14.1	65/M	URTI	PLEX/10	8	14	17	45	B/I	PLEX	GQ1b
14.2	60/M	N/a	PLEX/4	4	10	12	36	B/I	MP	GQ1b
Sakel et al [15]										
15.1	67/M	N/a	IVIG/N/a	N/a	N/a	10	N/a	B/I	IVIG	N/a
Liu et al [16]										
16.1	40/M	URTI	IVIG/5	4	9	10	33	B/I	MP	GM1
Nanatsue et al [17]										
17.1	70/M	URTI	IVIG/2	5	N/a	7	30	U/I	oPSL	GQ1b
Watanabe et al [18]										
18.1	35/M	URTI	PLEX/6	10	11	12	24	U/I	MP f/b oPSL	GQ1b, GT1a
18.2	46/F	URTI	IVIG/4	4	5	9	31	U/I	MP f/b oPSL	GQ1b
18.3	21/F	Diarrhea	IVIG/3	3	5	9	33	U/I	MP f/b oPSL	GQ1b, GT1b

oPSL: oral prednisolone; MP: methylprednisolone; IVIG: intravenous immunoglobulin; PLEX: plasmapheresis; U/I: unilateral; B/I: bilateral; N/a: not available; URTI: upper respiratory tract infection; GM1: ganglioside-monosialic acid; GQ1b: gangliosdie-Q1b.

noting no difference in resolution time between treated and untreated patients [7]. Recently, some authors questioned DFP as part of MFS progression since facial weakness appeared after other neurological symptoms improved [17, 19]. They argue that facial palsy should occur while other symptoms are present to be considered part of MFS. Additionally, DFP persisted for over 50 days without extra immunotherapy [18]. Consequently, some authors treated DFP with pulse dose steroids for 3 days followed by an oral prednisone taper or oral prednisone alone [17, 18]. The resolution time for steroid-treated DFP was 24 - 33 days (patients 18.1 and 18.3 in Table 1), a narrower range compared to untreated cases. While the minimum resolution time is similar, the maximum time differs significantly (33 vs. 85 days). Based on these findings, authors recommend additional steroid therapy for MFS-DFP despite the unknown mechanism [18]. In our patient, DFP resolved in 22 days with additional steroids, the shortest time compared with other reported cases in the literature thus far.

MFS-DFP is rarely reported in the literature, with our patient being the first documented case in the United States. This rarity may be due to the lower incidence of MFS in Western countries compared to Asian countries. Another possibility is the underestimation of its incidence due to its delayed onset after patient discharge. Our case report is the most comprehensive literature review on MFS-DFP, including 40 patients from 15 case reports and series. Limitations of our case report include the lack of electrophysiological studies, such as blink reflex tests.

Conclusions

Steroids are ineffective in treating GBS and may even worsen the condition. However, our patient's rapid resolution of DFP with steroids suggests that DFP might not be a progression of MFS. Instead, a different mechanism could be at play, and its occurrence after MFS onset might be coincidental. Most cases of facial palsy are self-limiting and improve within 6 weeks. However, complications such as corneal ulceration, vision loss, and permanent facial paralysis can occur if the facial palsy persists. While a few cases in the literature report prompt DFP resolution with treatment, it remains inconclusive whether additional steroid therapy effectively reduces the duration of MFS-DFP. Factors like age, severity of facial palsy, and presence of autoantibodies might contribute to a longer duration of DFP in untreated patients. More studies are needed to elucidate the pathophysiology of DFP, identify predictive factors, and determine the role of steroid therapy in a larger patient cohort. In conclusion, not all MFS-DFP cases improve promptly without additional therapies, and steroids might be effective in accelerating DFP recovery.

Learning points

A small subset of MFS patients develop DFP. Although DFP typically resolves on its own, additional steroid therapy may expedite recovery, thereby reducing the risk of complications

from prolonged DFP. This novel therapeutic approach is particularly noteworthy given that steroids are generally ineffective in treating GBS. Therefore, further investigation into the use of steroids for MFS-DFP is warranted.

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Financial Disclosure

None to declare.

Conflict of Interest

None to declare.

Informed Consent

Written informed consent was obtained from the patient.

Author Contributions

Nithisha Thatikonda: study design, literature search, and writing manuscript. Alexandru Lerint: literature search and writing manuscript. Vijaya Valaparla: writing and editing manuscript. Chilvana Patel: study design and manuscript editing. All the authors approved the final manuscript for submission.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Abbreviations

DFP: delayed facial palsy; MFS: Miller Fisher syndrome; GBS: Guillain-Barre syndrome; IVIG: intravenous immunoglobulin; ACE: angiotensin-converting enzyme; oPSL: oral prednisolone; MP: methylprednisolone; PLEX: plasmapheresis; U/l: unilateral; B/l: bilateral; N/a: not available; URTI: upper respiratory tract infection; GM1: ganglioside-monosialic acid; GQ1b: ganglioside-Q1b; LM1: lipid-modified

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