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Facial Sparing Paralysis in Autoimmune Diseases: A Narrative Review

Yamuna Devi S[1], Mythili D[2], Kishore Kumar P[3]

[1]Assistant Professor, Madha College Of Physiotherapy, Kundrathur, Chennai, Tamilnadu, India
[2]Professor And Principal, Madha College Of Physiotherapy, Kundrathur, Chennai, Tamilnadu, India
[3]Assistant Professor, Madha College Of Physiotherapy, Kundrathur, Chennai, Tamilnadu, India

ABSTRACT:
Facial paralysis is among the most visible and functionally disruptive presentations in clinical neurology, and while idiopathic (Bell's) palsy accounts for the majority of cases, a substantial minority arise from identifiable autoimmune and immune-mediated diseases. A recurring diagnostic theme across this group of disorders is the pattern of "sparing" — which muscles, nerves, or facial zones remain unaffected — because sparing patterns carry strong localizing and diagnostic value. Forehead sparing distinguishes a central (upper motor neuron) lesion from a peripheral (lower motor neuron) one; ocular sparing helps separate isolated lower cranial nerve syndromes from Miller Fisher syndrome; and fatigable, non-dermatomal weakness raises suspicion for myasthenia gravis rather than a structural neuropathy. This narrative review synthesizes current literature on facial paralysis occurring in the context of autoimmune diseases — including Bell's palsy, Guillain-Barré syndrome and its Miller Fisher variant, myasthenia gravis, neurosarcoidosis, Melkersson-Rosenthal syndrome, Sjögren syndrome, systemic lupus erythematosus, and ANCA-associated vasculitis — with an emphasis on the clinical sparing patterns that differentiate them, their proposed autoimmune mechanisms, and an evidence-informed approach to diagnosis, pharmacological management, and physiotherapy led facial neuromuscular retraining. Furthermore, this review explores how the distinction between peripheral nerve involvement and central autoimmune pathogenesis informs the prognosis of acute bilateral facial presentations, which frequently serve as a hallmark of more complex systemic syndromes

KEYWORDS: Paralysis, Autoimmune Diseases, Facial Nerve.

INTRODUCTION

The estimated incidence of acute facial weakness is 15 to 30 per 100 000 per year. Idiopathic Bell’s palsy accounts for 60 to 75% of unilateral cases, but autoimmune and other inflammatory conditions are an important, often overlooked, differential, comprising a small but clinically decisive proportion of presentations.(Awantika Singh,2022) The long intracranial, intratemporal and extratemporal course of the facial nerve and its close anatomical and functional relationship with the trigeminal, vestibulocochlear and lower cranial nerves, the parotid gland and neuromuscular junction means that a wide variety of autoimmune processes can cause facial weakness by different mechanisms: demyelination of the nerve itself, granulomatous infiltration, neuromuscular junction blockade, vasculitic ischemia or central demyelination affecting corticobulbar pathways.(Zhang et al., 2019) The differentiation of these causes at

the bedside is based mainly on identifying structures that are spared, rather than only affected. An important principle that provides the organizing theme for this review.(Leonhard, et al., 2019)

ANATOMICAL BASIS OF "SPARING" PATTERNS IN FACIAL PARALYSIS:

The clinical benefit of sparing starts with the double cortical innervation of the upper face.Kosins et al. (2007) Because the forehead and orbicularis oculi are innervated by both cerebral hemispheres, a unilateral central (upper motor neuron) lesion, such as a stroke or demyelinating plaque, generally spares the forehead, allowing the patient to wrinkle the brow and close the eye on the affected side, while the lower face droops.(Kubota-Hanya et al. , 2024) A lesion of the facial nerve itself (eg, Bell's palsy, Guillain-Barré syndrome, or granulomatous or neoplastic process) is a peripheral (lower motor neuron) lesion, and thus equally affects the forehead and lower face, since it interrupts the final common pathway after convergence of the cortical inputs.(Leonhard et al. 2019)

This one sign - forehead sparing versus forehead involvement - is often the first branching point in the differential diagnosis of acute facial weakness and prompts neuroimaging and stroke workup when sparing is present and a peripheral, often autoimmune-oriented workup when it is not.(Li, et al., 2024) In addition to this classical central vs peripheral distinction, there are several other sparing patterns which have diagnostic weight and are discussed below in each disease section: ocular sparing (or lack thereof) in Guillain-Barré/Miller Fisher variants, extraocular muscle sparing in isolated facial presentations of myasthenia gravis, and the asymmetric, sequential, or alternating sparing seen in Melkersson-Rosenthal syndrome and neurosarcoidosis.(Kaminski, et al., 2002)

AUTOIMMUNE AND IMMUNE-MEDIATED CAUSES OF FACIAL PARALYSIS: BELL'S PALSY:

Bell's palsy is officially labeled "idiopathic," but a large amount of data favors an autoimmune or para-infectious mechanism, in which re-activation of latent herpesviruses in the geniculate ganglion is believed to stimulate an autoimmune reaction against peripheral nerve myelin, causing acute demyelination of the facial nerve.(Takahashi, 2001). The autoimmune hypothesis is indirectly supported by the response to therapy with glucocorticoids, which decrease the incidence of permanent paralysis and are considered the standard of care, usually combined with an antiviral agent if patients present within 72 hours of onset.(Gronseth and Paduga, 2012) Bell's palsy, which typically involves the whole hemiface including the forehead and lower face, and is distinguished from central causes, is a diagnosis of exclusion after the exclusion of red flags such as a gradual course, a course >3 months without improvement, or associated limb or bulbar weakness.(Loukas , 2014) If, however, the paralysis is bilateral or recurrent, it necessitates a more extensive workup as these presentations are rarely associated with idiopathic Bell's palsy and strongly suggest an underlying pathology such as Lyme disease, sarcoidosis, or Guillain-Barré syndrome (Patel & Levin, 2015). In particular, in cases of recurrent facial nerve involvement, Melkersson-Rosenthal syndrome should be considered in the differential diagnosis, which can be diagnosed by the characteristic triad of facial palsy, lingual plicata and recurrent orofacial edema (Heckmann et al., 2019). Moreover, assessment for systemic involvement is important because systemic diseases such as sarcoidosis and Sjogren's syndrome frequently show concomitant parotid gland swelling or uveitis, which require a comprehensive multisystem evaluation (Elnazeir et al., 2020; Finsterer, 2008). In those cases, the clinicians should focus on the exclusion of neoplastic processes, especially if the clinical course is characterized by a progressive deterioration or lack of recovery within a period of four months (Loukas,

2014). To diagnose these more complex, non-idiopathic causes, specific serological tests for autoantibodies or ganglioside markers are often needed, as well as an analysis of the cerebrospinal fluid, which may show albuminocytologic dissociation in Guillain-Barré syndrome (MacIntosh & Fay, 2018). Furthermore, when clinical features suggest a systemic etiology of bilateral facial palsy, prompt neuroimaging and investigations are necessary, as this presentation has a much greater likelihood of being secondary pathology rather than idiopathic (Cohen et al., 2016; Yang & Dalal, 2021). Moreover, unusual clinical presentations such as bilateral disease, recurrent attacks, or total paralysis necessitate thorough evaluation of the parotid gland and external auditory canal to exclude structural or infiltrative lesions that may masquerade as idiopathic cases (Moses et al., 2025; Pauna et al., 2023). Patients presenting with bilateral symptoms and even mild limb paraesthesia should be prioritised for urgent assessment as the prevalence of Guillain-Barré syndrome in these patients is often underestimated and misdiagnosed as Bell's palsy (Jain et al., 2006; Xu et al., 2024).

GUILLAIN-BARRÉ SYNDROME AND THE MILLER FISHER VARIANT:

Guillain-Barré syndrome (GBS) is an autoimmune polyradiculoneuropathy, which is characterized by demyelinating or axonal nerve injury due to molecular mimicry between peripheral nerve gangliosides and infectious antigens preceding the disease.Meena et al., (2011) The classical presentation of GBS is ascending, symmetric limb weakness and areflexia, but facial diplegia, bilateral, usually near-simultaneous facial paralysis, is a recognised and important presentation, at times the dominant or presenting feature even prior to limb involvement becoming apparent.(Al-Timimy et al., 2026) As bilateral facial palsy is rare outside GBS, its presence should lead to consideration of the diagnosis, with subsequently a lumbar puncture to look for albumin-cytologic dissociation.(Taftaf et al., 2024) Miller Fisher syndrome (MFS), a form of GBS, is characterized by the triad of ataxia, areflexia, and ophthalmoplegia and is closely associated with anti-GQ1b ganglioside antibodies.(Wang et al., 2026). Delay in the progression of bilateral facial nerve palsy may follow the initial ataxic and oculomotor presentations, sometimes following appreciation of the ophthalmoplegia. This is an important pattern, since the sequence and "sparing" of ocular versus facial involvement over time helps differentiate MFS from isolated bilateral Bell's palsy, or from anti-GQ1b antibody syndrome variants presenting with facial and trigeminal involvement alone, without ataxia. Intravenous immunoglobulin is the mainstay of treatment for both GBS and MFS, with recovery of facial function usually following general neurological recovery.(Bannai et al., 2026)

MYASTHENIA GRAVIS:

Myasthenia gravis (MG) is an autoimmune disorder of the postsynaptic neuromuscular junction predominantly mediated by autoantibodies against the acetylcholine receptor, with less common autoantibodies against muscle-specific kinase or LRP4.(Dresser et al., 2021) Facial involvement is common in generalized MG but the hallmark feature that differentiates it from a structural facial neuropathy is fatigability (ie, weakness that worsens with sustained activity and improves with rest) rather than a fixed non-fluctuating paresis.(Loukas, 2014) Isolated or bilateral facial weakness without extraocular muscle involvement is rare but is increasingly reported. There is case literature that describes MG presenting as unilateral Bell's palsy. Therefore, persistent or atypical fluctuation of facial weakness or bilateral facial palsy without a clear etiology should be screened for MG via acetylcholine receptor antibody testing and repetitive nerve stimulation.(Kaminski et. al., 2024) Since MG involves the

neuromuscular junction, not the nerve trunk, sensory symptoms, taste disturbance and hyperacusis (features typical with facial nerve lesions) are characteristically spared.(Kaminski et al., 2024)

NEUROSARCOIDOSIS AND HEERFORDT SYNDROME:

Sarcoidosis is a systemic granulomatous disease that may affect the peripheral and central nervous system; the facial nerve is the most commonly involved cranial nerve in neurosarcoidosis.(Nascimento et al., 2022) Heerfordt syndrome is a distinct acute presentation of sarcoidosis that consists of the triad of fever, anterior uveitis, parotid gland swelling, and facial nerve palsy, which occurs in a small percentage of all cases of sarcoidosis; facial palsy is reported in around a quarter to a half of cases of Heerfordt syndrome and is mostly unilateral but can be bilateral.(O. et al., 2026). The associated systemic and glandular findings (parotid swelling, uveitis, low-grade fever) that are not present in Bell's palsy are a key diagnostic pattern and suggest the granulomatous rather than purely neurotic process. Corticosteroids, sometimes in combination with steroid-sparing agents, usually result in good recovery.(Sakikawa et al., 2024)

MELKERSSON-ROSENTHAL SYNDROME

Melkersson-Rosenthal syndrome (MRS) is a rare granulomatous neuro-mucocutaneous disorder classically characterized by the triad of recurrent peripheral facial palsy, orofacial edema and fissured tongue, although the triad is present in only a minority of patients and facial palsy is often the presenting – and sometimes the only – feature, leading to frequent misdiagnosis of recurrent Bell's palsy.(Tidke et al., 2023) MRS facial palsy is clinically indistinguishable from Bell's palsy at the level of the nerve itself, but the recurrent, sometimes bilateral or alternating pattern, with orofacial swelling, should raise the suspicion of MRS, especially when a patient presents with more than one episode of facial palsy over time. (Raimondo, 2025)Corticosteroids remain the cornerstone of treatment. Steroid-sparing immunosuppressants are used in refractory or relapsing cases.(Campbell et al, 2025)

SJOGREN SYNDROME AND SYSTEMIC LUPUS ERYTHEMATOSUS

Cranial neuropathies such as facial nerve palsy are known albeit relatively rare manifestations of systemic autoimmune connective tissue disease.(Raju, et al. 2023) Cranial neuropathies in systemic lupus erythematosus (SLE) usually occur in the context of active disease, are frequently transient and generally respond to corticosteroid therapy. Facial nerve palsy is one of the most commonly reported patterns, in addition to third and sixth nerve palsies and trigeminal neuralgia. The proposed mechanisms are vasculitis and in some cases thrombosis associated with antiphospholipid antibodies.Lima et al. (2023) The same principle of distinguishing overlapping autoimmune and infectious mimics that pervades this review should be applied before attributing a new cranial neuropathy to neuropsychiatric SLE, and concurrent infectious and other autoimmune causes, including Lyme disease, sarcoidosis and myasthenia gravis, should be ruled out.(Jarius et al., 2023) Sjögren syndrome may also lead to cranial and peripheral neuropathy but trigeminal sensory neuropathy is more classically described than isolated facial motor palsy. Bilateral parotid gland swelling in Sjögren syndrome may also mimic the glandular component of Heerfordt syndrome adding another layer to the differential.(Makimoto et al., 2020; Hanaoka, 2025)

ANCA-ASSOCIATED VASCULITIS:

Facial nerve palsy may be caused by small-vessel vasculitis such as granulomatosis with polyangiitis by ischemic injury to the vasa nervorum or by direct granulomatous involvement of adjacent structures such

as the middle ear and mastoid. (Chao et al., 2024) Vasculitic facial palsy is typically associated with other systemic features — sinonasal disease, otologic involvement, renal or pulmonary manifestations, or positive ANCA titer — that are not present in isolated Bell's palsy and again illustrate how systemic, non-facial findings act as diagnostic "non-sparing" clues that guide the clinician away from an idiopathic diagnosis.(Heckmann et al., 2019; Kirchgassner et al., 2024)

DIAGNOSTIC APPROACH:

A systematic approach to facial weakness of suspected autoimmune origin should include investigation of the pattern of sparing at three levels: Central vs peripheral: is the forehead spared (suggesting a central, upper motor neuron lesion requiring urgent stroke work-up) or involved (suggesting a peripheral facial nerve lesion)?(Graus et al., 2016 Moses et al., 2025) Unilateral vs. bilateral and simultaneous vs. sequential: bilateral or rapidly sequential facial palsy markedly narrows the differential toward Guillain-Barré syndrome/Miller Fisher syndrome, myasthenia gravis, sarcoidosis, Lyme disease or bilateral Bell's palsy and should prompt a broader systemic and neurologic work-up.(Molinari et al. 2023) Fixed vs. fatigable – fatigable weakness suggests myasthenia gravis Isolated vs. syndromic – associated ataxia and ophthalmoplegia suggests Miller Fisher syndrome; associated orofacial edema and fissured tongue suggest Melkersson-Rosenthal syndrome; associated fever, uveitis and parotid swelling suggest Heerfordt syndrome[Noioso et al., 2023]. The severity and recovery are usually graded using the House-Brackmann scale (grades I-VI) or the Sunnybrook Facial Grading System, both of which are also helpful for monitoring the rehabilitation progress. Investigations should not be performed indiscriminately but guided by the clinical pattern, including serological testing (antinuclear antibody, ANCA, acetylcholine receptor antibodies, anti-GQ1b antibodies, Lyme serology), cerebrospinal fluid analysis, chest imaging or angiotensin-converting enzyme levels for suspected sarcoidosis, and MRI of the brain and internal auditory canals when a central lesion, tumour, or bilateral/recurrent pattern requires exclusion of structural causes.(Kullar and Maria, 2025)

MANAGEMENT:

PHARMACOLOGICAL AND DISEASE-SPECIFIC THERAPY:

Once identified, management targets the underlying process: oral corticosteroids (with or without antivirals) for Bell's palsy, intravenous immunoglobulin or plasma exchange for Guillain-Barré syndrome and Miller Fisher syndrome, acetylcholinesterase inhibitors, corticosteroids and other immunosuppressants for myasthenia gravis, corticosteroids with steroid-sparing agents for neuro-sarcoidosis and Melkersson-Rosenthal syndrome, and disease-modifying immunosuppression for SLE, Sjögren syndrome, and ANCA-associated vasculitis. Regardless of etiology, ocular protection (lubrication, taping, or temporary tarsorrhaphy if eye closure is compromised) remains a common priority to prevent exposure keratopathy.(Caprara & Rissardo, 2023)

PHYSIOTHERAPY AND FACIAL REHABILITATION:

Facial neuromuscular rehabilitation is a cornerstone of treatment, regardless of etiology, once the acute inflammatory or immune process is under medical management.(Shokri et al., 2020) Data on facial nerve recovery after nerve-sparing procedures support structured neuromuscular retraining. Functional gains are greatest in the first 6 months, but meaningful improvement can continue for a year or more with continued rehabilitation.(Molinari et al. 2024) Physiotherapy approaches such as mirror-guided facial exercises, soft-

tissue mobilization, biofeedback-assisted movement retraining, and management of synkinesis aim to restore symmetrical, coordinated movement and prevent maladaptive compensatory patterns, and should be tailored to the grade of paralysis and the muscles spared or affected in each underlying condition.(Hahnemann et al., 2025).

CONCLUSION:

Facial paralysis in autoimmune disease has rich diagnostic potential, as the pattern of sparedness – the forehead, ocular muscles, tongue, systemic organ involvement, or the fatigability of weakness itself – often reveals the underlying mechanism more clearly than the physical deficit itself. By recognizing the patterns of relative sparing in Bell's palsy, Guillain-Barré/Miller Fisher syndrome, myasthenia gravis, neurosarcoidosis, Melkersson-Rosenthal syndrome, and systemic autoimmune connective tissue disease, clinicians can rapidly progress from presentation to targeted investigation and therapy.(Bannai et al., 2026; MacIntosh & Fay, 2018).The best chance of functional recovery and psychosocial well-being in affected patients is provided by multidisciplinary care that combines timely immunomodulatory therapy with structured physiotherapy-led facial rehabilitation.(Steinhaeuser et al., 2022)

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