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Multidisciplinary Management and Physical Therapy Outcomes in a Patient with Ramsay Hunt Syndrome: A Case Report

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ABSTRACT

Introduction: Ramsay Hunt syndrome (RHS) is an uncommon peripheral facial neuropathy caused by reactivation of latent varicella-zoster virus (VZV) within the geniculate ganglion, and it carries a worse prognosis for facial nerve recovery than idiopathic Bell's palsy[1,2]. Few reports detail the specific physical therapy decision-making - including the staged use of facial neuromuscular re-education and the deliberate avoidance of aggressive stimulation to prevent synkinesis - that accompanies pharmacological treatment of this condition. This case adds a structured, outcome-measure-driven account of physiotherapy management across the acute, paretic, and synkinesis-prevention phases of recovery.

Main Symptoms and Important Clinical Findings: A 54-year-old male presented with a 3-day history of severe right-sided otalgia followed by acute right peripheral facial palsy, incomplete eye closure, and vesicular eruptions over the right pinna. Examination revealed House-Brackmann Grade V facial palsy with absent voluntary movement of the frontalis, orbicularis oculi, orbicularis oris, and zygomaticus major, accompanied by reduced taste sensation and diminished lacrimation on the affected side.

Main Diagnoses, Therapeutic Interventions, and Outcomes: The clinical triad of otalgia, vesicular rash, and lower motor neuron facial palsy, supported by VZV-positive vesicle fluid PCR and MRI enhancement of the facial nerve, confirmed the diagnosis of Ramsay Hunt syndrome. Management combined oral antiviral therapy, corticosteroids, and ocular protection with a phased physiotherapy protocol progressing from gentle circulatory massage and eye protection in the acute flaccid phase to mirror-guided neuromuscular re-education and EMG biofeedback as paresis resolved.

Conclusion: Early antiviral and corticosteroid therapy combined with a carefully staged, low-effort facial re-education program - rather than early aggressive stimulation - may support functional recovery while minimizing the risk of synkinesis in patients with Ramsay Hunt syndrome.

Keywords: Ramsay Hunt syndrome; facial nerve palsy; varicella-zoster virus; facial neuromuscular re-education; EMG biofeedback

INTRODUCTION

Ramsay Hunt syndrome (RHS), also termed herpes zoster oticus, results from reactivation of latent varicella-zoster virus

within the geniculate ganglion of the facial nerve. It accounts for a notable proportion of acute peripheral facial palsy presentations and classically manifests as a triad of otalgia, herpetic vesicular eruption of the external ear or auditory canal, and ipsilateral lower motor neuron facial weakness.

Compared with idiopathic Bell's palsy, RHS is associated with more severe nerve involvement, a higher likelihood of concurrent cranial nerve VIII dysfunction (tinnitus, hearing loss, vertigo), and a substantially lower rate of complete recovery [1,2,3]. Early initiation of antiviral and corticosteroid therapy within the first 72 hours of onset is widely recognized as a key determinant of facial nerve outcome [3,4].

While the pharmacological management of RHS is well described, published accounts of the corresponding physical therapy pathway — including staged neuromuscular re-education, mirror feedback training, and the rationale for avoiding early aggressive facial exercise or electrical stimulation — remain limited. This case report describes the clinical course, multidisciplinary management, and detailed physiotherapy intervention of a patient with RHS, with the aim of illustrating an evidence-informed, phase-based rehabilitation framework and standardized outcome measurement that can inform clinical practice and physiotherapy training.

Patient Information

A 54-year-old male presented to the outpatient department with a 3-day history of severe, throbbing right-sided ear pain (otalgia), followed by the sudden onset of right facial drooping noticed upon waking. He reported an inability to close his right eye and the appearance of fluid-filled blisters over the right pinna concurrent with the onset of weakness. His childhood history was significant for chickenpox; there was no history of recent upper respiratory infection, trauma, or prior episodes of facial palsy. He had no relevant family history of neurological or autoimmune disease and no history of immunosuppression.

Primary Concerns and Symptoms

- Marked facial asymmetry with visible drooping of the right side of the face
- Escape of food and saliva from the right angle of the mouth during eating
- Severe pain behind the right ear, rated 8 out of 10 on the Visual Analogue Scale (VAS)
- Incomplete closure of the right eye with associated ocular dryness and irritation
- Difficulty with mastication and impaired verbal articulation
- Psychological distress related to altered facial appearance

Relevant History and Past Interventions

The patient had no prior episodes of facial palsy and had not received any treatment for this condition before presentation. No extremity weakness, cognitive alteration, or lower cranial nerve dysfunction (swallowing or vocal changes) was reported, and these red flags were specifically screened for and excluded on initial assessment.

Clinical Findings

Observation (Static and Dynamic)

- Asymmetry at rest: flattening of the right nasolabial fold, drooping of the right angle of the mouth, widened right palpebral fissure, and loss of forehead wrinkling on the right
- No synkinesis (abnormal involuntary associated movement) observed at this acute stage
- Integumentary findings: clustered, crusting erythematous vesicular lesions over the right concha and external auditory meatus

Motor Function Assessment (Voluntary Movement Testing)

- Frontalis: inability to elevate the right eyebrow or wrinkle the forehead
- Orbicularis oculi: incomplete eye closure with a positive Bell's phenomenon (upward and outward rolling of the eyeball on attempted closure)

- Orbicularis oris: inability to pucker the lips, whistle, or prevent air leakage during cheek puffing
- Zygomaticus major: inability to retract the right angle of the mouth on attempted smiling

Sensory and Autonomic Findings

- Reduced taste sensation over the anterior two-thirds of the tongue on the right side
- Markedly reduced lacrimation in the right eye, raising risk of exposure keratopathy

Timeline

The chronological sequence of clinical presentation, diagnostic evaluation, and physiotherapy/medical management is summarized in Table 1.

Table 1. Chronological timeline of clinical presentation, diagnostic evaluation, and management.

Day	Event
Day 1–3	Onset of severe right-sided otalgia (VAS 8/10)
Day 4	Sudden-onset right facial weakness noted on waking; vesicular eruptions appear over right pinna; patient presents for evaluation
Day 4	Clinical examination, viral PCR swab, contrast-enhanced MRI, and audiometry/ENG performed; House-Brackmann Grade V and Sunnybrook score of 18/100 recorded at baseline
Day 4	Oral valacyclovir and prednisolone initiated; ocular protection regimen started
Week 1–2	Acute flaccid phase physiotherapy: patient education, eye protection, gentle effleurage massage
Week 3 onward	Transition to facial neuromuscular re-education: mirror feedback training and EMG biofeedback introduced as paresis begins to resolve

Diagnostic Assessment

Diagnostic Testing

- Viral swab PCR of fluid collected from right auricular vesicles: positive for varicella-zoster virus DNA
- Contrast-enhanced MRI (brain and internal acoustic canal): enhancement and edema of the right facial nerve within the labyrinthine and geniculate segments, with no space-occupying lesion or brainstem infarct
- Audiometry and electronystagmography (ENG), performed for reported mild tinnitus: mild sensorineural hearing loss on the right; no vestibular nystagmus on ENG

Diagnostic Challenges

No significant barriers to diagnostic access were encountered; testing was completed within the critical treatment window, allowing pharmacological therapy to begin promptly.

Differential Diagnosis

The clinical triad of right otalgia, herpetic vesicles over the pinna and external auditory canal, and lower motor neuron facial nerve palsy was used to differentiate RHS from its principal mimics, summarized in Table 2.

Table 2. Differential diagnosis matrix for Ramsay Hunt syndrome.

Diagnostic Entity	Overlapping Features	Distinguishing Clinical Features	Distinguishing Investigations
Ramsay Hunt syndrome	LMN facial palsy; ear pain	Painful vesicular rash on pinna/EAC; frequent CN VIII	VZV PCR positive from vesicle fluid; MRI shows geniculate

		involvement (tinnitus, vertigo)	ganglion enhancement
Bell's palsy	Idiopathic unilateral LMN facial palsy; retroauricular ache	No vesicular eruption or severe herpetic neuralgia; isolated CN VII involvement	VZV PCR negative; secondary viral causes excluded
Otitis externa	Severe otalgia; localized erythema	Tenderness on tragal traction; no acute LMN facial paralysis	Otoscopy shows external auditory canal swelling with intact cranial nerve function
Vestibular schwannoma	Progressive facial weakness (late); tinnitus; hearing loss	Insidious onset over months; no vesicles or acute severe pain	Brain MRI (IAC protocol) reveals cerebellopontine angle mass

Diagnosis

The combination of clinical triad, VZV-positive vesicle PCR, and MRI findings of facial nerve enhancement confirmed a diagnosis of Ramsay Hunt syndrome.

Prognosis

Severe initial House-Brackmann grading (Grade V) and the presence of cochleovestibular involvement are recognized as adverse prognostic indicators for complete facial nerve recovery in RHS[2,5], and the patient was counselled accordingly regarding an anticipated prolonged recovery course relative to Bell's palsy.

Therapeutic Intervention

Medical Management

Pharmacological treatment was initiated within the critical 72-hour window from symptom onset.

- Antiviral therapy: oral valacyclovir 1 g three times daily for 7 days (acyclovir 800 mg five times daily as an alternative), to halt viral replication
- Corticosteroid therapy: oral prednisolone 1 mg/kg/day for 5 days, followed by a tapering protocol over 6 days, to reduce neural edema within the bony facial canal
- Ophthalmic care: artificial tear drops every 2 hours during the day, lubricating ointment at night, and nocturnal eye patching to prevent corneal abrasion from exposure keratitis.

Physiotherapy Management Protocol

Physiotherapy aimed to promote neural recovery while minimizing the risk of abnormal axonal misrouting and resultant synkinesis, following a staged approach consistent with current facial rehabilitation principles[6,7].

Sessions were delivered on a 1:1, therapist-supervised basis throughout the entire course of care (no unsupervised clinic-based treatment); a qualified neurophysiotherapist was present for every session and directly administered or supervised all manual therapy, mirror feedback, and EMG biofeedback techniques. Physiotherapy was scheduled 3 times per week (alternate days) for both Phase 1 and Phase 2/3, with each supervised session lasting approximately 30-40 minutes. The overall course was planned for 8-10 weeks of supervised care, with continuation and phase progression governed by the objective, criterion-based indicators described below rather than by a fixed calendar endpoint.

Phase 1 — Acute Flaccid Phase (Weeks 1–2)

- Patient education: explicit instruction against forceful or aggressive grimacing exercises and against the use of electrical stimulation, both of which are associated with an increased incidence of synkinesis
- Manual therapy: gentle effleurage circulatory massage directed superiorly to maintain facial muscle tone and tissue elasticity

- Continued ocular protection in coordination with the medical team

Phase 2 — Paresis/Initiation Phase and Phase 3 — Synkinesis Prevention (Week 3 Onward)

- Facial neuromuscular re-education (NMR) introduced as voluntary movement began to return
- Mirror feedback training: small, symmetrical, slow, isolated voluntary movements performed simultaneously on the affected and unaffected sides, with emphasis on low-effort recruitment (for example, a barely perceptible micro-smile that avoids co-activating the eye muscles)
- EMG biofeedback used to down-regulate overactive muscle groups and to assist the patient in precisely isolating weak facial movements (Figure 1)

Home Exercise Program (HEP)

A structured home exercise program supplemented supervised sessions throughout the rehabilitation course, with adherence checked and reinforced by the treating therapist at each visit.

- Phase 1 (acute flaccid phase): self-administered gentle effleurage massage in the same superior direction taught by the therapist, performed for 5 minutes, twice daily; explicit avoidance of any facial exercise, grimacing, or self-applied electrical stimulation at home; ocular protection measures (artificial tears, nocturnal patching) continued as prescribed
- Phase 2/3 (once voluntary movement returned): mirror-guided practice of small, symmetrical, low-effort movements for 5-10 repetitions per targeted muscle group, twice daily, with explicit instruction to stop if fatigue, overflow movement, or twitching (early synkinesis) was noticed; a home exercise diary was used to record repetitions and any adverse observations for review at the next supervised session

Figure 1. *Clinical photograph from a supervised facial neuromuscular re-education session using a computerized electrotherapy/biofeedback unit, with the treatment probe positioned at the right perioral region and a return electrode secured over the forearm. (Photograph obtained with patient consent; authors to confirm exact device/modality and session phase depicted.)*

Progression Criteria for Phase Transition

Advancement between rehabilitation phases was criterion-based rather than time-based, guided by the following objective indicators assessed at each supervised session:

- Transition from Phase 1 (acute flaccid) to Phase 2 (paresis/initiation) required the emergence of the first clinically visible or palpable voluntary muscle contraction in any of the four target muscle groups (frontalis, orbicularis oculi, orbicularis oris, zygomaticus major), corresponding to an improvement to House-Brackmann Grade IV or better, together with absence of synkinesis on structured clinical observation and medical clearance from the treating physician
- Within Phase 2, progression from mirror feedback alone to combined mirror feedback with EMG biofeedback required the patient to demonstrate a consistent, isolated voluntary contraction (without visible mass movement or co-contraction of adjacent muscle groups) on at least two consecutive sessions
- Entry into Phase 3 (synkinesis-prevention emphasis) was triggered by the appearance of any early synkinesis sub-score on the Sunnybrook Facial Grading System or by observed involuntary co-activation (for example, eye narrowing during volitional smiling), prompting an immediate shift toward inhibitory, low-effort retraining and a reduction in repetition intensity
- Planned criteria for tapering supervised session frequency and progressing to an independent home program were a plateauing or ceiling-level Sunnybrook composite score, a stable Facial Disability Index score across two consecutive assessments, and no new synkinesis on serial reassessment

Changes in Intervention

No deviation from the planned pharmacological course was required; the patient tolerated the full course of antiviral and corticosteroid therapy without adverse effect. Physiotherapy progressed from Phase 1 to Phase 2/3 techniques in a criterion-based manner, triggered by the emergence of voluntary motor activity rather than by a fixed timepoint, consistent with the principle of avoiding premature high-effort facial exercise. Specifically, physiotherapy advanced from Phase 1 to Phase 2 once the first visible voluntary movement was detected (consistent with the criteria above), rather than at a pre-set week, and progression from mirror feedback to combined mirror feedback with EMG biofeedback occurred once isolated, symmetrical contraction without mass movement was consistently demonstrated.

Follow-up and Outcomes

Clinician- and Patient-Assessed Outcomes

Standardized, validated outcome measures were used to track recovery objectively over the course of treatment (Table 3)[8]:

Table 3. Baseline physical therapy outcome measures used to quantify facial nerve function and disability at presentation.

Outcome Measure	Baseline Finding
House-Brackmann Facial Nerve Grading System (HBGS)	Grade V (severe dysfunction)
Sunnybrook Facial Grading System (SFGS)	18/100 — resting symmetry, voluntary movement symmetry, and synkinesis sub-scores recorded
Facial Disability Index (FDI)	Administered to assess physical function (eating, speech articulation) and social well-being subscales

Important Follow-up Findings

Serial reassessment using the House-Brackmann and Sunnybrook scales was planned to track the trajectory of facial nerve recovery and to identify early signs of synkinesis, which would prompt a shift in physiotherapy emphasis toward inhibitory, low-effort retraining techniques.

Intervention Adherence and Tolerability

The patient adhered fully to the prescribed antiviral and corticosteroid regimen and attended all scheduled physiotherapy sessions. He tolerated manual therapy and neuromuscular re-education techniques well, with no reported discomfort during mirror feedback or EMG biofeedback sessions. Adherence to the ocular protection regimen was reinforced at each visit given the risk of exposure keratopathy.

Adverse and Unanticipated Events

No adverse events were observed in relation to either the pharmacological or physiotherapy interventions during the reported course of care.

DISCUSSION

This case illustrates the multidisciplinary management of Ramsay Hunt syndrome, integrating early antiviral and corticosteroid pharmacotherapy with a staged, evidence-informed physiotherapy pathway. The clinical presentation and investigative findings were consistent with previously described features of RHS, including the characteristic triad of otalgia, vesicular rash, and lower motor neuron facial palsy, as well as the association with cochleovestibular symptoms such as tinnitus and mild sensorineural hearing loss [1,2,9].

A central feature of the physiotherapy approach in this case was the explicit avoidance of aggressive facial exercise and electrical stimulation during the acute flaccid phase. This is consistent with the broader literature on facial nerve rehabilitation, which links early high-effort or non-selective stimulation to an increased risk of synkinesis through aberrant axonal regeneration and cross-innervation of facial muscle groups [6,7]. Mirror-guided neuromuscular re-education and EMG biofeedback, introduced once voluntary movement began to return, are similarly supported as techniques that promote selective, symmetrical muscle recruitment while limiting compensatory mass movement patterns [7,8].

Severity at presentation — reflected here by an initial House-Brackmann Grade V and a low baseline Sunnybrook score — together with cochleovestibular involvement, are recognized prognostic indicators associated with a less favorable and more protracted recovery in RHS compared with Bell's palsy [2,5,9]. This underscores the value of early, structured physiotherapy involvement and serial standardized outcome measurement, including the Facial Disability Index, to capture both physical and psychosocial dimensions of recovery [8].

Strengths and Limitations

This case report provides a detailed, phase-specific account of physiotherapy decision-making in RHS, supported by standardized, validated outcome measures (House-Brackmann, Sunnybrook, and Facial Disability Index scores), which strengthens the reproducibility and clinical applicability of the described approach. As a single case, however, findings cannot be generalized, and the report describes baseline assessment and the initiation of a treatment pathway rather than long-term outcome data; longitudinal follow-up over the typical 6- to 12-month recovery window would be required to confirm functional and synkinesis outcomes.

CONCLUSION

Early initiation of antiviral and corticosteroid therapy, combined with a carefully staged physiotherapy program that prioritizes ocular protection and gentle massage in the acute phase before progressing to low-effort, mirror-guided neuromuscular re-education and EMG biofeedback, represents a rational, evidence-

informed approach to the management of Ramsay Hunt syndrome. Deliberately avoiding early aggressive facial stimulation may reduce the risk of synkinesis and support more favourable long-term functional recovery.

Declaration by Authors

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