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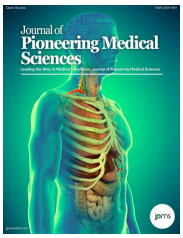

Hyperbaric Oxygen Therapy in the Management of Bell's Palsy: A Case Study

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Abstract **Objectives:** Bell's palsy (BP) is an acute, unilateral facial nerve paralysis that can lead to significant facial muscle dysfunction and considerable impact on quality of life. Standard treatment includes corticosteroids and antiviral medications, but some patients experience incomplete recovery. Hyperbaric oxygen therapy (HBOT) has been proposed as a complementary treatment due to its neuroregenerative and anti-inflammatory properties. **Case Presentation:** A healthy 31-year-old male presented with acute right-sided facial pain, numbness and muscle weakness, consistent with BP (Grade IV House-Brackmann classification). Diagnostic evaluations, including magnetic resonance imaging and electroneuromyography (ENMG), confirmed the diagnosis. Initial treatment involved prednisone and vitamin B complexes. The patient then underwent HBOT (2.5 ATA, 80 minutes daily, over 18 sessions). Significant clinical improvements were observed following HBOT, with recovery in facial muscle function and reduction of symptoms. Follow-up ENMG showed restoration of nerve conduction. At a 12-month evaluation, the patient achieved near-complete recovery, classified as Grade I on the House-Brackmann scale. **Conclusion:** This case highlights the potential benefits of integrating HBOT with conventional therapy for BP, suggesting its role in enhancing neuroregeneration and accelerating recovery. Beyond individual outcomes, this case underscores the broader implications of utilising HBOT as an adjunct in conditions with acute nerve injury. Although limited by a paucity of high-quality evidence, HBOT holds promise as a valuable addition to BP management, particularly in cases where conventional treatment yields suboptimal results. These findings warrant further clinical trials to establish its efficacy, optimise protocols and ensure cost-effectiveness for broader implementation.

Key Words Adjunctive therapy, case reports, diving medicine, facial nerve paralysis, house-brackmann classification, hyperbaric oxygen, nerve regeneration

INTRODUCTION

Facial nerve paralysis, also called Bell's palsy (BP), is an acute and unilateral facial weakness, with an estimated incidence of 20 cases per 100,000 [1]. BP evolves rapidly within two days and symptoms may include ear discomfort, sensitivity to loud noise and reduced tear production. This condition can occasionally lead to complete paralysis, manifesting with noticeable changes in facial appearance and mimicry, affecting the buccal region and the elevator palpebrae superioris muscle [2].

BP diagnosis is primarily clinical, supported by Magnetic Resonance Imaging (MRI), which can highlight contrast accumulation in the affected nerve and electroneuromyography (ENMG), which demonstrates altered nerve conduction and the degree of impairment [3,4]. However, BP is a diagnosis of exclusion, ruling out other causes of acute facial palsy, such as infections or tumours. Emerging evidence suggests viral aetiology in many cases, particularly Herpes Simplex Virus, Herpes Zoster, Cytomegalovirus and Epstein-Barr Virus [5,6].

Regardless of the cause, the underlying pathophysiology points to nerve hypoxia and swelling within the facial canal of the temporal bone, where inflammation and ischemia lead to reversible neuropathy and axonal injury due to Wallerian degeneration [7].

The cornerstone of BP treatment remains corticosteroids, often combined with antiviral therapy in cases of virological positivity [8]. However, the prognosis is variable and many patients experience incomplete recovery or prolonged symptoms, underscoring the need for adjunctive therapies. Hyperbaric oxygen therapy (HBOT) has been suggested as a potential treatment due to its ability to enhance tissue oxygenation, reduce inflammation and support neuroregeneration. While current evidence is limited and of low quality, HBOT's proposed mechanism, improving perfusion and promoting healing of ischemic nerves, provides a strong theoretical rationale for its use [9].

This case report presents a healthy young man with BP, highlighting the integration of HBOT into a multidisciplinary treatment strategy. By addressing the research gap surrounding HBOT's role in neuro lesions, this report aims to stimulate further exploration of its potential benefits in enhancing recovery and influencing broader clinical practice, particularly for patients unresponsive to conventional treatments.

Case Presentation

Patients Information and Clinical Assessment: The present case study was reported according to CARE guidelines. A healthy 31 years-old man came to the emergency department (AOU "San Giovanni di Dio e Ruggi d'Aragona", Salerno, Italy), reporting pain and numbness of the right facial region, with an associated slight deviation of the buccal fissure, eyelid ptosis, solids and liquid dysphagia. The patient reported a physically active lifestyle, with no significant medical history or comorbidities and did not smoke or consume alcohol. He expressed significant emotional distress related to the sudden onset of facial asymmetry, including concerns about social interactions.

Emergency neurological evaluation excluded acute stroke and the diagnostic process continued with ear-nose-throat (ENT) specialist evaluation. The endoscopic study of the upper airways and digestive tracts did not highlight motility deficits, showing adequate swallowing reflex.

The MRI study revealed a soft impregnation of the distal right facial nerve, characterised by contrast enhancement. No mass lesions, structural abnormalities, or pathological enhancement patterns suggestive of these conditions were detected. The ENMG demonstrated a reduction in the amplitude of the compound muscle action potentials on the right side compared to the unaffected side, indicating partial axonal involvement. Additionally, mild prolongation of distal latency was observed, consistent with demyelination secondary to inflammation. While these findings were consistent with Bell's palsy, they also required the exclusion of other neuropathies (e.g., Guillain-Barré syndrome or diabetes-associated neuropathy), which were ruled out based on the patient's history and clinical context. Furthermore, blood chemistries were negative for storage disease and viral infections. Together with the absence of systemic signs or atypical progression, the clinical findings were ultimately compatible with a BP diagnosis (grade IV of the House-Brackmann classification, Table 1) [10].

Table 1: The modified House Brackmann facial grading system

Grade	Description	Characteristics
I	Normal	Normal facial function in all nerve branches
II	Slight	Gross: slight weakness on close inspection At rest: normal tone and symmetry Motion: forehead slight weakness, eye complete closure with minimum effort, mouth slight asymmetry Synkinesis: absent or mild
III	Moderate	Gross: obvious facial weakness At rest: normal tone or subtle asymmetry (mild) Motion: forehead moderate weakness, eye complete closure with effort, mouth slight weakness with maximum effort Synkinesis: moderate and /or hemifacial spasm may develop
IV	Moderately severe	Gross: severe facial weakness At rest: asymmetrical facial appearance (moderate) Motion: forehead severe weakness, eye incomplete closure, mouth asymmetrical with maximum effort
V	Severe	Gross: barely noticeable movement At rest: asymmetrical facial appearance (severe) Motion: forehead no movement, eye incomplete closure, mouth slight movement
VI	Total	No facial movement

Therapeutic Interventions

The patient initially began medical therapy with prednisone 30 mg/day per os and group B vitamin complexes. However, no significant clinical improvement was observed after one week of corticosteroid therapy. This lack of progress exacerbated the patient's concern about his condition, particularly regarding the potential for long-term facial dysfunction. As a result, he was referred to our Diving Medicine Center for further evaluation and consideration of hyperbaric oxygen therapy (HBOT).

Contraindications were further assessed through a detailed medical history, including exclusion of pneumothorax, severe chronic obstructive pulmonary disease and active middle ear infections, which are known risk factors for HBOT complications.

According to our local protocol, HBOT was prescribed at 2.5 atmosphere absolute (ATA) for 80 minutes, once a day, for 18 cycles. This protocol is based on evidence suggesting that pressures between 2.0 and 2.5 ATA are optimal for enhancing oxygen delivery to ischemic and inflamed tissues while minimising the risk of oxygen toxicity. The choice of 18 cycles reflects the balance between achieving clinical efficacy and limiting patient burden.

Before starting the complete treatment, the patient performed a trial HBOT (2.2 ATA for 40 minutes), allowing

Figure 1: Clinical evolution. Panel A illustrates the patient's clinical condition before initiating HBOT, showing significant motor impairment of the right facial muscles and asymmetry in the buccal region. Panel B demonstrates the patient's condition after completing 18 sessions of HBOT, highlighting notable improvements in muscle function, symmetry restoration and near-complete recovery as graded by the House-Brackmann scale (Grade II at the end of therapy, progressing to Grade I at 12 months follow-up)

Table 2: The table outlines the key phases of the patient's diagnostic and therapeutic journey, highlighting the progression from initial presentation and standard therapy to the integration of HBOT and the subsequent clinical outcomes

Phase	Description	Outcome
Initial presentation	Right-sided facial pain and weakness (House-Brackmann IV)	Bell's palsy diagnosis confirmed by ENMG and MRI
Initial Therapy	Prednisone 30 mg/day and B-complex vitamins	No significant improvement
HBOT	18 sessions (2.5 ATA, 80 minutes/day)	Marked motor and symptomatic improvement
12-Month Follow-up	Clinical evaluation (House-Brackmann I)	Near-complete recovery

him to familiarise with the hyperbaric chamber environment and evaluate the occurrence of poor psychological tolerability to the treatment (i.e., unknown claustrophobia). After passing the trial, the patients started the scheduled HBOT.

While HBOT was chosen as an adjunctive therapy, other advanced therapies for facial nerve recovery (e.g., transcutaneous electrical nerve stimulation) were considered. However, these approaches were deemed less suitable in this early stage due to the patient's significant inflammatory component, where HBOT's anti-inflammatory and neuroregenerative effects were prioritised.

Outcome and Follow-up

After the first eight cycles of HBOT, the patient reported a subjective improvement in the motor component and objectively, there was an increase in wrinkling of both the facial muscles and a significant elevation of the eyelid. By the end of HBOT (after 18 cycles), the patient showed a marked improvement in symptoms and signs, reporting the absence of pain and reduced numbness and near-complete restoration of muscular functions, achieving Grade II on the House-Brackmann scale (Figure 1).

At a 30-day follow-up, a new ENMG was performed, which highlighted the reappearance of the R1 component with the recovery of the direct R2 latencies, indicative of significant neurophysiological recovery. Patient-reported outcome measures were also collected at this time, showing improved quality of life and functional satisfaction as measured by the Facial Disability Index [11], with a score of 90% in the physical function domain and 85% in the social/well-being domain.

At the 12-month evaluation, the patient achieved Grade I of the House-Brackmann classification, indicating near-complete recovery. Notably, the patient reported sustained improvements without recurrence of symptoms or functional decline, suggesting the durability of nerve function recovery post-treatment.

The patient's diagnostic and therapeutic journey is summarised in Table 2, highlighting key milestones from initial presentation to follow-up.

DISCUSSION

As reported in this case report, BP diagnosis and treatment are based on a multi-disciplinary approach involving different medical figures and diagnostic tools (MRI and ENMG). BP diagnosis is by elimination. Clinical examination should prioritise the fine analysis of facial motricity to confirm the peripheral nature of the palsy, with complete neurologic and ENT examination [12]. As in our case, MRI showed characteristic but non-specific enhancement of the facial nerve ipsilateral to the neurologic lesion. ENMG was fundamental for assessing the clinical recovery profile after medical and HBOT treatment [3,4].

Once the diagnosis is confirmed, the treatment strategy is based on drugs and HBOT. HBOT represents a non-invasive and usually well-tolerated treatment by patients. The ratio for HBOT derives from the cascade activation of molecular signals that lead to the regeneration of peripheral nervous tissue induced by oxygen. The molecular pathways appear to lead to a reduction of apoptosis through inhibition of the genes that promote it, such as c-fos and c-jun, as well as a consensual reduction of caspase activation. Hyperbaric oxygen therapy also promotes the expression of neurotrophic factors such as NGF and NT-3 [13]. Vilela *et al.* [14] in a study evaluating the effect of HBOT on the histological

pattern of the facial nerve in rabbits exposed to a nerve crush injury, showed a more advanced process of axonal regeneration, confirmed by measurement of the external axonal diameters (axon plus myelin sheath), which was statistically significant in comparison with the control group [14].

Although preclinical studies justify the rationale for HBOT to treat axonal degeneration, data in the literature conducted on human populations are scarce. Racic *et al.* [15] performed the only single-blinded randomised clinical trial (RCT) on the topic. The study compared 42 patients treated with HBOT and placebo with 37 treated with placebo HBOT and prednisone. The study found a recovery of facial function in 95% of the patients treated with HBOT compared to 76% in the prednisone group, with a significant difference in favour of HBOT (RR 1.26, 95% CI 1.04 to 1.53).

Other evidence derived from observational studies. Litavrin *et al.* [16] in a comparative analysis, compared 42 participants with BP treated with HBOT against 29 participants with a similar disease treated conventionally. When added to standard treatment, they reported improved facial nerve outcomes and recovery speed with HBOT. Makishima *et al.* [17] in a non-controlled case series in which 12 participants with BP and 11 participants with Ramsay Hunt syndrome were treated with HBOT, stellate ganglion block, an oral vasodilator (kallidinogenase), vitamins and mecobalamine, showed that ten patients had a "satisfactory clinical result" and five of these had complete recovery in facial motor function. However, the study did not compare other treatments. Still, Sadiq *et al.* [18] in a prospective observational study, showed that the combination of HBOT and steroids is beneficial in treating BP. The study showed that 90% of patients fully recovered after the treatment, while 10% experienced partial recovery. Factors such as the severity of the disease and the time between symptom onset and treatment initiation were statistically significant in influencing the treatment outcomes.

Despite these findings from the literature, Holland *et al.* [9], in a Cochrane review, concluded that HBOT may be an effective treatment for moderate to severe Bell's palsy with low-quality evidence, advocating for further RCT on the topic. Ultimately, the French Society of ENT guidelines for managing acute BP did not recommend HBOT as treatment (Grade C) [8].

However, although the clinical validity of HBOT for the treatment of BP is debated, severe adverse effects (central neurological toxicity, seizure, pulmonary oxygen toxicity and pneumothorax) are very rare and there were no reported complications in the literature studies.

In conclusion, this case highlights the potential benefits of integrating HBOT into treating BP. The significant functional recovery observed in this patient, along with improved quality of life and reduced residual disability, suggests that HBOT could be a promising therapeutic option, particularly in cases resistant to conventional treatments.

Despite the limited availability of high-quality clinical data in the current literature, the underlying biological mechanisms, including nerve regeneration and inflammation reduction, provide a strong theoretical rationale. However, large-scale RCTs are needed to confirm HBOT efficacy, optimise protocols and evaluate cost-effectiveness. While awaiting further evidence, HBOT should be cautiously encouraged in selected cases, given its low incidence of severe adverse effects. This case supports the use of HBOT in BP and underscores the importance of a multidisciplinary approach to improving clinical outcomes in acute nerve injury conditions.

Patient perspective

Before treatment, the patient was apprehensive about his appearance. Currently, he reported no motor or sensory deficits.

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This study was conducted without external funding. No collaborations with external institutions influenced this research's design, conduct, or reporting.

Ethical statement

The patient gave written informed consent to the publication of this case report. Patient data were anonymised and the study adhered to the General Data Protection Regulation (GDPR) and local ethical guidelines to ensure confidentiality.

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REFERENCE

[1] Rowlands, S., *et al.* "The epidemiology and treatment of bell's palsy in the UK." *European Journal of Neurology*, vol. 9, no. 1, January 2002, pp. 63-67. http://dx.doi.org/10.1046/j.1468-1331.2002.00343.x.

[2] Holland, N Julian and Graeme M Weiner. "Recent developments in bell's palsy." *BMJ*, vol. 329, no. 7465, September 2004, pp. 553-557. http://dx.doi.org/10.1136/bmj.329.7465.553.

[3] Kinoshita, T., *et al.* "Facial nerve palsy: Evaluation by contrast-enhanced MR imaging." *Clinical Radiology*, vol. 56, no. 11, November 2001, pp. 926-932. http://dx.doi.org/10.1053/crad.2001.0730.

[4] Khedr, Eman M., *et al.* "Prognostic role of neurophysiological testing 3–7 days after onset of acute unilateral bell's palsy." *Neurophysiologie Clinique*, vol. 48, no. 2, April 2018, pp. 111-117. http://dx.doi.org/10.1016/j.neucli.2018.02.002.

[5] Schmidbauer, M., *et al.* "Presence, distribution and spread of productive varicella zoster virus infection in nervous tissues." *Brain*, vol. 115, no. 2, December 1991, pp. 383-398. http://dx.doi.org/10.1093/brain/115.2.383.

[6] Vrabec, Jeffrey T. and Deborah A. Payne. "Prevalence of herpesviruses in cranial nerve ganglia." *Acta Oto-Laryngologica*, vol. 121, no. 7, January 2001, pp. 831-835. http://dx.doi.org/10.1080/00016480152602285.

[7] Fisch, U. and H. Felix. "On the pathogenesls of bell's palsy." *Acta Oto-Laryngologica*, vol. 95, no. 5, January 1983, pp. 532-538. http://dx.doi.org/10.3109/00016488309139438.

[8] Fieux, M., *et al.* "French society of ENT (SFORL) guidelines. management of acute bell's palsy." *European Annals of Otorhinolaryngology, Head and Neck Diseases*, vol. 137, no. 6, December 2020, pp. 483-488. http://dx.doi.org/10.1016/j.anorl.2020.06.004.

[9] Holland, N Julian, *et al.* "Hyperbaric oxygen therapy for bell's palsy." *Cochrane Database of Systematic Reviews*, vol. 2016, no. 7, February 2012. http://dx.doi.org/10.1002/14651858.cd007288.pub2.

[10] House, John W. and Derald E. Brackmann. "Facial nerve grading system." *Otolaryngology–Head and Neck Surgery*, vol. 93, no. 2, April 1985, pp. 146-147. http://dx.doi.org/10.1177/019459988509300202.

[11] Özden, Fatih, *et al.* "Psychometric properties of the facial disability index in patients with facial palsy: A systematic review and meta-analysis." *Neurological Sciences*, vol. 43, no. 7, April 2022, pp. 4157-4165. http://dx.doi.org/10.1007/s10072-022-06066-z.

[12] Diego, Juan I. De, *et al.* "Seasonal patterns of idiopathic facial paralysis: A 16 year study." *Otolaryngology–Head and Neck Surgery*, vol. 120, no. 2, February 1999, pp. 269-271. http://dx.doi.org/10.1016/s0194-5998(99)70418-3.

[13] Shinomiya N, Asai Y. *Hyperbaric oxygenation therapy: Molecular mechanisms and clinical applications*. 1st Edn., Springer, 2019, Pages: 152. https://doi.org/10.1007/978-981-13-7836-2.

[14] Vilela, Daniela Salgado Alves, *et al.* "Effects of hyperbaric oxygen therapy on facial nerve regeneration." *Acta Oto-Laryngologica*, vol. 128, no. 9, January 2008, pp. 1048-1052. http://dx.doi.org/10.1080/00016480701827525.

[15] Racic, G. *et al.* "Hyperbaric oxygen as a therapy of Bell's palsy." Undersea and Hyperbaric Medicine, vol. 24, no. 1, 1997, pp. 35-38. https://pubmed.ncbi.nlm.nih.gov/9068154.

[16] Litavrin, A.F. *et al.* "Hyperbaric oxygenation in the treatment of facial neuritis." *Zh Nevropatol Psikhiatr Im S S Korsakova*. Vol. 85, no. 4, 1985, pp. 528-531.

[17] Makishima, K., *et al.* "Hyperbaric oxygenation as a treatment for facial palsy." *Advances in Oto-Rhino-Laryngology*, edited by N. Yanagita, T. Nakashima, Basel, KARGER, 1998, pp. 110-118. http://dx.doi.org/10.1159/000059046.

[18] Sadiq, Dr. Imran, *et al.* "Hyperbaric oxygen therapy (HBOT); an "adjuvant" to bell's palsy treatment." *The Professional Medical Journal*, vol. 23, no. 10, October 2016, pp. 1252-1257. http://dx.doi.org/10.17957/tpmj/16.3504.