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Treatment of Bell's Palsy Using Monochromatic Infrared Energy: A Report of 2 Cases

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Abstract

Objective: The purpose of the study is to describe the use of monochromatic infrared energy (MIRE) therapy in the management of 2 patients with Bell's palsy.

Clinical features: Two patients presented to a chiropractic clinic with Bell's palsy that was diagnosed by a medical physician. Both patients were treated using MIRE. The acute patient was a 32-year-old male. He presented with left facial palsy 1 day before the consultation. He was unable to puff the left cheek and close the left eyelid. He had difficulty raising the left eyebrow. The chronic case was a 46-year-old lady. Prior to the first consultation, she was treated with corticosteroid and electro-acupuncture for one and a half years, with incomplete recovery. When first seen, the left corner of mouth drooped and she had difficulty raising her left eyebrow.

Intervention and outcome: Monochromatic infrared energy therapy, emitting 890 nm infrared light, was placed on the post-auricular area, pre-auricular area, the temple and mandibular area of the affected side. Each treatment lasted 30 minutes. Photographs were taken every week to document changes. The acute case received 19 treatments in 6 weeks. He reported an improvement of 95%. The chronic case received a total of 45 treatments in 9 months. She rated an improvement of 50%. At the conclusion of treatment, she was able to close her left eyelid and puff her left cheek but still could not raise her left eyebrow.

Conclusion: These 2 patients seemed to respond to a different degree to the MIRE therapy. As 71% of patients with Bell's palsy recover uneventfully without any treatment, the present study describes the course of care but cannot confirm the effectiveness of MIRE therapy in the management of Bell's palsy.

Introduction

Bell's palsy is a sudden peripheral palsy involving the facial nerve. Recent studies have shown that it may be due to herpes simplex infection [1] or reactivation of latent herpes simplex virus 1 (HSV1) in the facial ganglion. [2–6]

A study of the facial canal anatomy in children showed that the facial nerve fills nearly 35% to 65% of the bony canal; the remaining portion is filled with extraneural blood vessels and connective tissues without leaving any empty space. [7] Along the facial canal, the labyrinth segment is the narrowest part. [1]

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Table 1 Infrared Laser of Different Wavelength and Power has Been Found to be Effective in the Management of Bell's Palsy; the Present Study Used MIRE with Wavelength and Power Between that Used in the Studies by Marques et al[25] and Rubis[25]

Year	Studies	Type of Photo-energy	λ (nm)	Power (mW)	Energy (Joules or Joules/cm^2)	Conditions Treated
2010	Marques et al[25]	Soft infrared laser	790	40 mW	26-29 J/cm^2	Bell's palsy
2013	Rubis[25]	Cold infrared laser	910	Peak pulse 100,000 mW	47.6 × 10 J	Bell's palsy
Present Study		MIRE	890	60 mW/cm^2	108 J/cm^2	Bell's palsy

MIRE, monochromatic infrared energy.

Herpes simplex infection was believed to result in inflammation and swelling of the facial nerve, as evidenced by operative,[1,8] magnetic resonance imaging[1,9] and histopathology studies.[10,11] An intraoperative study showed swelling of facial nerve proximal to meatal foramen in patients with Bell's palsy.[8] Entrapment of swollen facial nerve was found at meatal foramen and labyrinth segment. Magnetic resonance imaging studies similarly showed signs of inflammation of facial nerve within the facial canal.[1,9,12,13] Also, a histopathological study of the facial nerve 1 week after the onset of Bell's palsy showed infiltration of inflammatory cells, with myelin breakdown, axonal changes and marked edema.[14] Response of Bell's palsy to steroid treatment also suggested that inflammation and edema of facial nerve play a part in the pathogenesis of Bell's palsy.[15]

When facial nerve inflames, pressure builds up in the facial canal,[1] especially in the narrow labyrinth segment where vascularity is poor.[16] Compression of the facial nerve, with attending axonal changes and myelin breakdown is believed to contribute to Bell's palsy.

Study showed that 85% of patients function returned within 3 weeks of onset, without any intervention.[17] In 71% of patients, normal facial mimicry function was obtained, with the rest having slight to severe sequelae.[17] As there are at present no methods to determine which cases will resolve uneventfully and which will not, treatment is provided upon presentation.

Various treatments have been used to treat Bell's palsy. Steroid and antiviral agents are standard medical treatments. Clinical trials using steroid and antiviral agents alone or in combination yielded mixed results.[15,18–20] Some studies showed better results with addition of antiviral agents to steroid[18] and others showed addition of antiviral agents had little effects.[15,20] Chiropractic treatment,[21,22] acupuncture[23] and physiotherapy treatments[24] have also been used to treat Bell's palsy, with varying degree of success. Recently, a case of Bell's palsy was reported to be treated successfully by low level infrared laser with improvement of facial weakness.[25]

Some patients are aware of the side effects of corticosteroid and are reluctant to take the medication. Also, some are scared of needling or cervical manipulation. Infrared laser may provide an alternative to treatment of Bell's palsy. The treatment, however, requires accurate localization of facial nerve.

Monochromatic infrared energy (MIRE) therapy is an array of 60 gallium aluminum arsenide light emitting diodes (LED). The diode array must be placed in direct contact with the target skin. As it can irradiate a large area simultaneously when compared with the infrared laser. This reduces the need for very accurate localization of facial nerve. Application is thus easier. Also, the wavelength (890 nm) and energy level lie between that of low level infrared laser reported to be effective in the treatment of Bell's palsy (Table 1).[25,26] At present, there are no case reports of the use of MIRE for patients with Bell's palsy. Therefore, the purpose of the study is to describe the use of MIRE therapy in the management of 2 patients with Bell's palsy.

Case Reports

Two consecutive patients with Bell's palsy were treated by monochromatic infrared light. Case A was an acute case with 1 day onset and case B was a chronic case for around 2 years. Both cases were diagnosed by a medical physician prior to being seen. The labelled indication for monochromatic infrared lights is for increasing circulation and decreasing pain. Both patients gave consent for their information to be included in this case report.

Case A

A 32-year-old man presented with an acute left facial palsy with 1 day onset. On the day prior to the onset, he felt feverish and experienced slight twitching of his left face and dripping of saliva. He was seen by a medical practitioner and was diagnosed with Bell's palsy. He was prescribed a course of corticosteroid, which the patient said he did not take.

Upon presentation, he was unable to puff the left cheek and close the left eyelid. There was lack of corrugation of left forehead when attempting to raise the left eyebrow. Food tended to be lodged in the left cheek. Drooping of left corner of mouth was evident. The patient did not complain of altered taste sensation nor increased sensitivity to sound. The patient had no prior history of herpes infection nor respiratory complaints. He was not taking any medication. He had no history of hypertension nor diabetes mellitus. Also, there was no history of accidents nor surgery.

Cranial nerve tests were performed, except cranial nerve I. Cranial nerves II, III, IV, V, VI, VIII, IX, X, XI and XII were intact. Weakness of left facial expression muscles was evident, including weakness of frontal belly of occipito-frontalis. He was unable to raise the left eyebrow. This ruled out the possibility of central causes of the condition, as upper motor neurone lesions tend to affect only the lower face. The upper face muscles are usually spared as innervation to the muscles of upper face originates bilaterally from the brain.

Physical examination showed that the patient had bilateral anterior rotated shoulders and a forward head posture. Motion palpation of the cervical and thoracic spine, however, did not reveal any motion restriction. Radiographs of the cervical spine were not taken.

The patient consented to our taking photographs to track his progress on a weekly basis. He was treated with MIRE therapy. The 4 LED pads were placed on the post-auricular area, pre-auricular area, temple and mandible of the affected side (Fig). The pads in front and behind the ear directed the infrared light to the facial canal. The other 2 pads were placed on the ipsilateral temple and mandible to irradiate the temporal, zygomatic, buccal and mandibular branches of the facial nerve. Treatment was set at 40 minutes as it was found that increased blood flow following irradiation by low level infrared laser leveled off at 25 to 30 minutes.[27] The treatment was warm and soothing. As some heat was generated during the treatment, the treatment areas were not covered to enable dissipation of heat. The protocol was used during the entire treatment period. He was advised not to receive any other treatments, including Chinese medicine and acupuncture.

He was treated daily in the first 2 weeks, except the weekend. In the third to fifth week, he was treated twice weekly. He was thereafter treated once weekly (Table 2). After the first 2 treatments, the patient did not report any changes in symptoms. After the third treatment, which was the fifth day after the onset of the facial palsy, the patient reported an increased ability to close the left eyelid. There was a reduced tendency for food to lodge in the left cheek. He was, however, still unable to puff the left cheek nor raise the left eyebrow. As a result of the asymmetry in facial appearance, he was withdrawn and he resigned from his job as a retail manager. At the tenth treatment, which was 11 days from the onset, the patient reported less tightness of his left face and was able to raise the left eyebrow and puff the left cheek. The face became more symmetrical. Also, there was no more dripping of saliva. Some weakness of left facial muscles was, however, still evident. He was then treated twice a week for a further 3 weeks. After a total of 19 treatments which lasted for 6 weeks, the patient reported symmetry of facial expression. He reported an improvement of 95%, with mild weakness in closure of left eyelid.

Fig. Placement of pads for monochromatic infrared energy therapy.

A telephone call was made to the patient 8 months after the last treatment. He reported complete recovery with no residual facial weakness nor residual symptoms.

Case B

A 46-year-old lady, with a long history of systemic lupus erythematosus (SLE), presented with discomfort of her neck and left periorbital and perioral areas. Two years prior to consultation, she complained of vertigo with nausea and vomiting and was hospitalized. One week after, her left face suddenly became paralyzed when having a meal. She was diagnosed with Bell's palsy and was treated with corticosteroid for 10 days in the hospital. After being discharged, she consulted a private medical practitioner, who again diagnosed her with Bell's palsy and treated her with corticosteroid injection for 8 times. There was little improvement. She then sought treatment from a Chinese medical practitioner and received electro-acupuncture for about 1 year. The

treatment brought about little improvement, and moderate weakness of left facial muscles was still present.

On the day of first consultation, the patient could not raise her left eyebrow, blow her left cheek nor close the left eyelid completely. There were no symptoms relating to taste or hearing. Also, there were no signs of synkinesis. She had systemic lupus erythematosus for over 20 years and had history of hypertension. There was no history of trauma. She had surgical excision of necrotic fat in the left breast and surgery of intestinal obstruction. She took prednisolone tablet (7.5 mg), azathioprine (50 mg, Douglas Pharmaceuticals Ltd, New Zealand), azathioprine (25 mg, Sandoz, Germany) and simvastatin (10 mg, Teva Pharmaceutical Works, Debrecen, Hungary) once daily. She also took 5 mg of ramipril (Sanofi Aventis, Italy) twice a day.

Physical examination revealed a lower motor neurone type facial weakness of the left side. The rest of the physical examination including the central nervous examination was within normal limits. Motion palpation of the neck revealed fixation between occiput and C1, with mild tenderness on the left suboccipital area. Movements of the neck were not painful. Flexion and extension movements of the cervical spine were full. Cervical rotation and lateral flexion to left were slightly limited. Radiographs of the cervical spine were not taken.

The patient agreed to have photographs taken to document the progress. During the first consultation, the fixed occiput-C1 was manipulated in the supine position using chiropractic diversified technique with very slight force. Care was exercised in view of the fact that the patient had been taking corticosteroid for over 20 years. 4 LED pads were then applied on the left face, according to the protocol detailed above.

In the second visit, no fixation between occiput-C1 was found and the patient was not given spinal manipulation. Also, in order not to confound the MIRE treatment, the patient was not manipulated thereafter. She received only MIRE treatment from the second visit onward. She was treated twice a week. After the 10th treatment, she was seen once every week (Table 3).

After the first treatment, the patient felt that the left corner of the mouth was slightly more relaxed. After 4 MIRE treatments, the patient felt slight movement on the left perioral area when smiling, but she still could not raise her left eyebrow. After the seventh treatment, the left nasolabial fold became more obvious and more dentition was seen when smiling. However, when the patient attempted to frown, very little corrugation of left forehead was seen. After the tenth treatment, patient could close her left eyelid completely. She could evenly close her mouth when blowing up her left cheek. The condition of the patient reached a plateau thereafter. At this point, the patient was requested if she agreed for us to continue the treatment for investigative purpose. Upon consent of the patient, the MIRE treatment was continued. Improvement, however, was insignificant.

On the 22nd visit, which was the fourth month, the patient had rash on her left face. Her dermatologist prescribed extra dose of steroid. Despite this, there was no significant improvement of the left facial palsy. On the 31st treatment, which was 6 months from the date of first consultation, her SLE flared up and her medical practitioner increased the steroid dosage. In the following 2 weeks, the patient reported an improvement. She could raise her left eyebrow more. The improvement, however, did not persist.

Table 2 The Clinical Presentation, Intervention and Progress of Case A. Date 0 was the First Date of Consultation

No of Tx	Date	Clinical presentation/intervention/outcome
1	0	Unable to blow the left cheek, incomplete closure of left eyelid, unable to raise the left eyebrow; treated by MIRE therapy; no cervical adjustment
2	1	MIRE therapy × 40 min
3	2	MIRE therapy × 40 min
4	4	Able to close the left eyelid better; unable to blow nor raise the left eyebrow; MIRE therapy × 40 min
5	5	MIRE therapy × 40 min
6	6	MIRE therapy × 40 min
7	7	MIRE therapy × 40 min
8	8	MIRE therapy × 40 min
9	9	MIRE therapy × 40 min
10	11	Able to raise the left eyebrow and blow the left cheek; left face felt less tight; MIRE therapy × 40 min
11	13	MIRE therapy × 40 min
12	14	MIRE therapy × 40 min
13	17	Subjective rating: Improved by 70%; can blow the left cheek; increased ability to raise the eyebrow, left eyelid closure still incomplete; MIRE therapy × 40 min
14	21	MIRE therapy × 40 min
15	24	Subjective rating: Improved by 80%; increased strength of raising the left eyebrow and closure of left eyelid; MIRE therapy × 40min
16	28	MIRE therapy × 40 min
17	31	MIRE therapy × 40 min
18	35	Subjective rating: over 90% improvement; MIRE therapy × 40 min
19	40	Subjective rating: around 95% improvement; mild incomplete closure of left eyelid; otherwise, there is complete facial symmetry; MIRE therapy × 40 min; discharged

MIRE, monochromatic infrared energy.

Table 3 The Clinical Presentation, Intervention and Progress of Case B; Date 0 was the First Date of Consultation

No of Tx	Date	Clinical presentation/intervention/outcome
1	0	Unable to blow the left cheek, close the left eyelid and raise the left eyebrow; history of SLE for over 20 years; on prednisolone 7.5 mg, azathioprine 75 mg, ramipril 10 mg and simvastatin 10 mg daily; MIRE therapy × 30 min; rotary break of O/C1
2	1	Left corner of mouth slightly relaxed; MIRE therapy × 30 min
3	3	No obvious improvement; MIRE therapy × 30 min
4	7	Left corner of mouth more relaxed; no other improvement; MIRE therapy × 30 min
5	8	When smiling, the left corner of mouth moved slightly; MIRE therapy × 30 min
6	10	Increased ability to move the left corner of mouth; MIRE therapy × 40 min
7	14	Improvement in closure of left eyelid; MIRE therapy × 40 min
8	17	Slight improvement in closure of left eyelid; cheeks more even when puffing; left nasolabial fold became more obvious; MIRE therapy × 40 min
9	21	No significant improvement; MIRE therapy × 40 min
10	31	No improvement; MIRE therapy × 40 min
11	38	Better able to close the left eyelid; MIRE therapy × 40 min
12	45	Less deviation of mouth to left; left eyelid could close almost completely; MIRE therapy × 40 min
13	52	More dentition shown on the left when smiling; MIRE therapy × 40 min
14	59	Mild synkinesis evident; when closing the left eye, the left corner of mouth twitched; MIRE therapy × 40 min
15	66	No more improvement; MIRE therapy × 40 min
16	73	No improvement; mild skin rash on left face; MIRE therapy × 45 min
17	80	No improvement; MIRE therapy × 45 min
18	94	No significant improvement; MIRE therapy × 45 min
19	101	Improvement slow; left eyelid can close but not completely; could not raise the left eyebrow; MIRE therapy × 45 min
20	108	No significant improvement; MIRE therapy × 45 min
21	115	No significant improvement; MIRE therapy × 45 min
22	122	Slight improvement of left eyelid closure; more dentition shown when smiling; skin rash on the left face; MIRE therapy × 45 min
23	129	No significant improvement, steroid was prescribed by dermatologist to treat the skin rash; MIRE therapy × 45 min
24	135	No significant improvement; increased lacrimation of left eye; skin rash improved; MIRE therapy × 45 min
25	142	Slight relaxation of left corner of mouth for 2 days; still on steroid; MIRE therapy × 45 min
26	149	Slight relaxation of left corner of mouth; twitching of left corner of mouth when closing eyes; MIRE therapy × 45 min
27	156	Slight swelling of left eyelid; skin rash on left face; MIRE therapy × 45 min
28	163	Left eyebrow can be raised slightly; MIRE therapy × 45 min
29	170	Left eyebrow can be raised slightly; MIRE therapy × 45 min
30	177	Very little improvement; MIRE therapy × 45 min
31	184	Left eyelid could be closed more completely; SLE more serious, steroid dosage stepped up; MIRE therapy × 45 min
32	191	Could raise the left eyebrow more; open the left eye more; MIRE therapy × 45 min
33	198	Increase in left eye secretion; no other improvement; MIRE therapy × 45 min
34	205	Increase in left eye secretion; no other improvement; MIRE therapy × 45 min
35	212	Increase in left eye secretion; no other improvement; MIRE therapy × 45 min
36	219	Increase in left eye secretion; no other improvement; could not raise the left eyebrow completely; cheeks symmetrical when puffing; MIRE therapy × 45 min
37	226	Left eye secretion still present; no other improvement; MIRE therapy × 45 min
38	240	Left eye secretion still present; no other improvement; mouth less deviated to left; MIRE therapy × 45 min
39	247	Could not raise well the left eyebrow; could close the left eyelid more; Able to puff both cheeks symmetrically; MIRE therapy × 45 min
40	254	Could not raise well the left eyebrow; could close the left eyelid more; Able to puff both cheeks symmetrically; left eye secretion still present; improvement of 50%; MIRE therapy × 45 min
41	261	No further improvement; MIRE therapy × 45 min
42	268	No further improvement; MIRE therapy × 45 min
43	275	Could not raise the left eyebrow; left eyelid could be closed completely; able to puff both cheeks relatively symmetrically; chewed gum daily; MIRE therapy × 45 min
44	282	No further improvement; MIRE therapy × 45 min
45	289	Subjective rating: improvement of 50%; able to puff both cheeks relatively symmetrically; able to close the left eyelid; unable to raise the left eyebrow; mild synkinesis present (when closing the eyes, the left corner of the mouth twitched); MIRE therapy × 45 min

MIRE, monochromatic infrared energy; *SLE*, systemic lupus erythematosus.

The patient was discharged after a total of 45 treatments, which spanned over a period of 9.5 months. At the time of discharge, the patient was able to close her left eyelid, puff the left cheek and close the mouth symmetrically. She, however, still could not raise the left eyebrow. She reported an improvement of 50% as compared to initial presentation.

Five months after the last consultation, the patient reported that the condition was stable. There was no deterioration of the condition. Mild synkinesis was present. The left corner of the mouth twitched slightly when she attempted to close the left eyelid.

Discussion

MIRE has a wavelength of 890 nm and a power density of 60 mW/cm^2,[27] the parameters of which are similar to that of the low level infrared laser used to treat Bell's palsy.[25,26] It can penetrate up to 5 cm through bony and soft tissues[28] and can theoretically reach the facial canal when applied transcutaneously. Also, it is safe to apply transcranially. Transcranial application of LED infrared light with energy level of 250 mW/cm^2 is not harmful.[29] It causes negligible heat and no physical damage.[29]

The mechanism of actions of MIRE is unknown. The physiological effects of MIRE on biological tissues are thought to be due to photochemical reactions in the skin similar to low level infrared laser therapy.[30]

In both cases reported in this study, the treatment started during the first consultation, as studies have shown that early application favorably modulates inflammation[31,32] and nerve regeneration.[33] When laser irradiation was applied immediately after carrageenan-induced inflammation, the animals did not suffer from traumatic injury or vascular damage.[31,32] Early application of laser also favorably affects nerve regeneration.[33,34] Transcutaneous application of Ga-As laser (903 nm, 27 mW) 3 minutes daily for 1, 3, 5 and 7 weeks was found to improve the morphology and function of damaged sciatic nerve fibers in rats.[35] Increase in myelinated axons, decrease in degenerated axons and increase in nerve conduction velocity were significant in the irradiated group of rats as compared to the control group in the third and fifth weeks of treatment.[35] The effects were proportional to the time of treatment.[35] The difference between the treatment and control groups, however, disappeared at seventh week of treatment as natural repair of injured myelinated nerve fibers developed vigorously around the sixth to seventh week after injury. This suggested that laser irradiation improves the regeneration of nerve fibers in the early stage.[35] Another study comparing the effects of different wavelengths on the regeneration of crushed facial nerve showed that transcutaneous phototherapy when carried out daily over the first postoperative week, led to a significant increase in the rate of regeneration of facial nerve axons in comparison to untreated animals,[34] suggesting that early treatment with laser irradiation is important in enhancing regeneration of damaged nerve fibers.

MIRE exerted its effects possibly through improvement in microcirculation and regeneration of nerve fibers. MIRE of 890 nm was found to significantly increase the capillary blood cell velocity and superficial blood flow as compared to the placebo and control groups.[27] The increase in microcirculation was 20% to 30% in normal subjects. It was reasoned that similar increase in microcirculation within the facial canal would take place, despite the presence of demyelination and axonal changes within the facial nerve,[14] as vasodilatation following light irradiation was not nerve dependent.[36] It was theorized that the initial phase of vascular dilatation was related to nitric oxide and the late phase of the vascular dilatation was due to a reduction of intracellular calcium in microvascular smooth muscle cells.[36] As the labyrinth segment of the facial canal is narrow and poorly vascularized,[16] increase of microcirculation may speed up the reduction of inflammation and edema which compromise the facial nerve.

Apart from the improvement in microcirculation, the MIRE may also exert its effects through influence on viral activities and stimulation of nerve regeneration. Previous studies have shown that low level infrared laser irradiation has no direct inactivating effects on HSV1 culture.[37] Instead, it possibly acted by inhibiting HSV1 replication.[38] Laser irradiation enhances nerve regeneration in animal[34,35,39] and human studies.[40] Low-level infrared laser irradiation has been found to increase the functional activity of the injured peripheral nerve,[41] maintain functional activity of the injured nerve over time,[42] increase the rates of axonal growth and myelination[35,39,42] and accelerate regeneration of the injured nerve. Low level infrared laser irradiation was also found to improve motor function in patients with incomplete nerve damage.[40] In a clinical randomized double blind, placebo-controlled study, 780 nm laser was applied on patients with incomplete peripheral nerve and brachial plexus injuries for 6 months up to several years.[40] Most of these patients were discharged by orthopedic surgeons, neurosurgeons and plastic surgeons without treatment. Low level infrared laser irradiation was applied transcutaneously for 5 hours daily for 21 consecutive days; 3 hours to the injured areas of the peripheral

nerve and 2 hours on the corresponding segments of the spinal cord. At the end of 6 months, motor function showed significant improvement in the treated group as compared to the placebo group. Sensory function in the treated group did not improve as fast, It was only until the sixth month when the difference from the placebo group reached statistical significance.[40]

LED emits incoherent narrow spectrum light, whereas laser emits coherent light. Infrared light from both LEDs and lasers disperse once the light contacts body tissue. Infrared light output from LEDs possess all the attributes of laser radiation except coherence.[28] The physiological effects of MIRE therapy on biological tissues are thought to be similar to that of the low level infrared laser.[30] Nevertheless, MIRE is not low level infrared laser. Their effects on viral replication and nerve regeneration may differ. Extrapolation of the effects of low level infrared laser on viral replication and nerve regeneration to MIRE may not be appropriate.

Precaution and Contraindication

MIRE therapy produces mild heat. During application, the area should not be covered. Also, the skin contact should be even, especially on areas with bony prominence to avoid local increase in temperature, which can cause blister. The infrared light should not be used over cancerous lesions. Other than that, MIRE therapy is a safe treatment. LED array has achieved non significant risk status for human trials by the Food and Drug Administration (www.clinicaltrials.gov).[29] Also, there have been no reports of adverse effects from MIRE treatment. Recent studies have shown that transcranial LED infrared light modulates neurobiological functions and improves psychological disorders.[29]

Limitations

The current study is limited by only 2 patients and no control subjects. Around 71% of patients with Bell's palsy recover normal function of the facial muscles without treatment. People who recover usually do so quickly, with 85% of them reporting some improvement in the first 3 weeks.[17] Therefore it is impossible to determine if the response of these 2 cases was due to the intervention or to the natural history of the condition. The steroid and the azathioprine intake by the chronic case also confounds the outcome. In addition, the diagnosis of the chronic case may be incorrect.

Conclusion

MIRE therapy was used to treat an acute and a chronic case of Bell's palsy. Though the 2 cases responded to a varying extent to the therapy, the present study cannot state effectiveness of MIRE therapy in the management of Bell's palsy.

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No funding sources or conflicts of interest were reported for this study.

References

1. Kim IS, Shin SH, Kim J, et al. Correlation between MRI and operative findings in Bell's palsy and Ramsay Hunt syndrome. Yonsei Med J 2007;48:963–8.
2. Adour KK, Bell DN, Hilsinger Jr RL. Herpes simplex virus in idiopathic facial paralysis (Bell palsy). JAMA 1975;233:527–30.
3. Burgess RC, Michaels L, Bales Jr JF, Smith RJ. Polymerase chain reaction amplification of herpes simplex viral DNA from the geniculate ganglion of a patient with Bell's palsy. Ann Otol Rhinol Laryngol 1994;103:775–9.
4. Furuta Y, Fukuda S, Chida E, et al. Reactivation of herpes simplex virus type 1 in patients with Bell's palsy. J Med Virol 1998;54:162–6.
5. McCormick DP. Herpes-simplex virus as a cause of Bell's palsy. Lancet 1972;299:937–9.
6. Murakami S, Mizobuchi M, Nakashiro Y, Doi T, Hato N, Yanagihara N. Bell's palsy and herpes simplex virus: identification of viral DNA in endoneurial fluid and muscle. Ann Intern Med 1996;124:27–30.
7. Sato H, Takeda T, Kishimoto S. Facial nerve to facial canal cross sectional area ratio in children. Laryngoscope 1992;102:1172–6.
8. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve. Pathologic findings in Bell's palsy. Arch Otolaryngol 1972;95:335–41.
9. Yetiser S, Kazkayas M, Altinok D, Karadeniz Y. Magnetic resonance imaging of the intratemporal facial nerve in idiopathic peripheral facial palsy. Clin Imaging 2003;27:77–81.
10. Gacek RR, Gacek MR. Idiopathic facial paralysis (Bell's palsy). Otol Neurotol 2002;23(4):617–8.
11. Liston SL, Kleid MS. Histopathology of Bell's palsy. Laryngoscope 1989;99:23–6.
12. Schwaber MK, Larson III TC, Zealear DL, Creasy J. Gadolinium enhanced magnetic resonance imaging in Bell's palsy. Laryngoscope 1990;100:1264–9.
13. Suzuki F, Furuta Y, Ohtani F, et al. Herpes virus reactivation and gadolinium-enhanced magnetic resonance imaging in patients with facial palsy. Otol Neurotol 2001;22:549–53.
14. Linder T, Bossart W, Bodmer D. Bell's palsy and herpes simplex virus: fact or mystery. Otol Neurotol 2005;26:109–13.
15. Engström M, Berg T, Stjernquist-Desatnik A, et al. Prednisolone and valaciclovir in Bell's palsy: a randomized, double-

blind, placebo-controlled, multicentre trial. Lancet Neurol 2008;7:993–1000.
16. Gebraski SS, Telian SA, Niparko JK. Enhancement along the normal facial nerve in the facial canal: MR imaging and anatomic correlation. Radiologe 1992;183:391–4.
17. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl 2002;549:4–30.
18. Hato H, Yamada H, Matsumoto D, Honda N, Gyo K. Furuta Yet al. Valacyclovir and prednisolone treatment for Bell's palsy: a multicenter, randomized, placebo-controlled study. Otol Neurotol 2007;28:408–13.
19. Hato N, Murakami S, Gyo K. Steroid and antiviral treatment or Bell's palsy. Lancet 2008;371:1818–20.
20. Tyler KL. Prednisolone—but not antiviral drugs—improve outcome in patients with Bell's palsy. Neurol 2009;5(2):74–5.
21. Alcantara J, Plaugher G, Van Wyngarden DL. Chiropractic care of a patient with vertebral subluxation and Bell's palsy. J Manipulative Physiol Ther 2003;26(4):253.
22. Cotton BA. Chiropractic care of a 47 year-old woman with chronic Bell's palsy. A case study. J Chiropr Med 2011;10:288–93.
23. Wang Y, Yang J. Clinical observation on treatment of acupuncture for different stages of Bell's palsy. Zhonguo Zhen Jiu 2010;30(1):23–6.
24. Shrode LW. Treatment of facial muscles affected by Bell's palsy with high-voltage electrical muscle stimulation. J Manipulative Physiol Ther 1993;16(5):347–52.
25. Marques AMC, Soares LGP, Nascimento CM, Neto AAPV, Marques RC, Pinherro ALB. Laser phototherapy in Bell's palsy. A case report. Laser 2010;3:12–3.
26. Rubis LM. Chiropractic management of Bell palsy with low level laser and manipulation: a case report. J Chiropr Med 2013;12:288–91.
27. Mak MC, Cheing GL. Immediate effects of monochromatic infrared energy on microcirculation in healthy subjects. Photomed Laser Surg 2012;30(4):193–9.
28. http://www.anodynetherapy.com/infrared_therapy.html.
29. Rojas JC, Gonzalez-Lima F. Neurological and psychological applications of transcranial lasers and LEDs. Biochem Pharmacol 2013;86:447–57.
30. Fishman SM, Ballantyne JC, Rathmell JP. Bonica's management of pain. 4th ed. Williams and Wilkins; 2009.
31. Aimbire F, Albertini R, Pacheco MTT, Castro-Faria-Neto HC, Leonardo PSL, Iversen VV. Low-level laser therapy induces dose-dependent reduction of TNF-alpha levels in acute inflammation. Photomed Laser Surg 2006;24(1):33–7.
32. Albertini R, Aimbire FSC, Correa FI, et al. Effects of different protocol doses of low power gallium-aluminum-arsenate (Ga-Al-As) laser radiation (650 nm) on carrageenan induced rat paw oedema. J Photochem Photobiol B 2004;74:101–7.
33. Shin DH, Lee E, Hyun JK, et al. Growth-associated protein-43 is elevated in the injured rat sciatic nerve after low power laser irradiation. Neurosci Lett 2003;344:71–4.
34. Anders JJ, Borke RC, Woolery SK, Van de Merwe WP. Low power laser irradiation alters the rate of regeneration of the rat facial nerve. Lasers Surg Med 1993;13:72–82.
35. Bae CS, Lim SC, Kim KY, et al. Effect of Ga-As laser on the regeneration of injured sciatic nerves in the rat. In Vivo 2004;18:489–96.
36. Maegawa Y, Itoh T, Hosokawa T, Yaegashi K, Nishi M. Effects of near-infrared low-level laser irradiation on microcirculation. Lasers Surg Med 2000;27:427–37.
37. Körner R, Bahmer F, Wigand R. The effect of infrared laser rays on herpes simplex virus and the functions of human immunocompetent cells. Hautarzt 1989;40(6):350–4.
38. Donnarumma G, De Gregorio V, Fusco A, et al. Inhibition of HSV-1 replication by laser diode-irradiation: possible mechanism of action. Int J Immunopathol Pharmacol 2010;23(4):1167–76.
39. Mohammed IFR, Mustawfi NA, Kaka LN. Promotion of regeneration processes in injured peripheral nerve induced by low level laser therapy. Photomed Laser Surg 2007;25(2):107–11.
40. Rochkind S, Drory V, Alon M, Nissan M, Ouaknime GE. Laser phototherapy (780 nm), a new modality in treatment of long-term incomplete peripheral nerve injury: a randomized double-blind placebo-controlled study. Photomed Laser Surg 2007;25(5):436–42.
41. Rochkind S, Nissan M, Lubart R, Avram J, Bartal A. The in vivo nerve response to direct low-energy laser irradiation. Acta Neurochir (Wien) 1988;94:74–7.
42. Rochkind S, Barr-Nea L, Razon N, Bartal A, Schwartz M. Stimulatory effect of He-Ne low dose laser on injured sciatic nerves of rats. Neurosurgery 1987;20:843–7.