**MEDICAL REHABILITATION IN PATIENT WITH**

**LEFT BELL’S PALSY**

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**ABSTRACT**:Bell’s palsy is a complex neuromuscular facial disorder of unknown etiology commonly affecting the motor neurones of facial muscles receiving their neurological innervations from the seventh cranial nerve (the facial nerve).1 Bell's palsy is named after Sir Charles Bell (1774–1842), who first described the syndrome along with the anatomy and function of the facial nerve.2 The facial nerve not only carries motor fibers including fibers to the stapedius muscle but also supplies autonomic innervations of the lacrimal gland, submandibular gland, sensation to part of the ear, and taste to the anterior two thirds of the tongue via the chorda tympani.3 Bell’s palsy accounts for almost three quarters of peripheral facial palsies and the annual incidence is about 15-30 patients per 100.000 annually.The sexes are affected equally. The median age at onset is 40 years, but the disease may occur at any age. The right and left sides of the face are involved with equal frequency.4,5Treatment of Bell’s palsy varies, and no clear consensus exists. The aims of treatment in the acute phase of Bell's palsy include strategies to speed recovery and to prevent corneal complications. Most physicians prescribe corticosteroids as a primary treatment due to its potential to reduce swelling and inflammation. The addition of antiviral treatment (AVT) such as Acyclovir or Valacyclovir is aimed at eradication of HSV infection.6,7 The treatments at Medical Rehabilitation include facial exercise or facial neuromuscular re-education, electrostimulation, infrared rays, and acupuncture, as well as the need for eye-protective and further assessed with electroneurography or electromyography in the presence of complete facial paralysis.

**ABSTRAK:** Bell's palsy adalah gangguan wajah neuromuskular kompleks dengan etiologi yang tidak diketahui umumnya mempengaruhi neuron motorik otot wajah yang menerima persarafan neurologis dari saraf kranial ketujuh (saraf wajah).1 Bell's palsy dinamai Sir Charles Bell (1774-1842), yang pertama kali menggambarkan sindrom tersebut beserta anatomi dan fungsi nervus fasialis.2 Nervus fasialis tidak hanya membawa serabut motorik termasuk serabut ke otot stapedius tetapi juga mempersarafi persarafan otonom kelenjar lakrimal, kelenjar submandibular, sensasi ke bagian telinga, dan pengecapan pada dua pertiga anterior lidah melalui korda timpani.3 Bell's palsy merupakan hampir tiga perempat dari kelumpuhan wajah perifer dan kejadian tahunan sekitar 15-30 pasien per 100.000 per tahun. Jenis kelamin terpengaruh sama. Usia rata-rata saat onset adalah 40 tahun, tetapi penyakit ini dapat terjadi pada usia berapa pun. Sisi kanan dan kiri wajah terlibat dengan frekuensi yang sama.4,5 Pengobatan Bell's palsy bervariasi, dan tidak ada konsensus yang jelas. Tujuan pengobatan pada fase akut Bell's palsy termasuk strategi untuk mempercepat pemulihan dan untuk mencegah komplikasi kornea. Kebanyakan dokter meresepkan kortikosteroid sebagai pengobatan utama karena potensinya untuk mengurangi pembengkakan dan peradangan. Penambahan pengobatan antivirus (AVT) seperti Asiklovir atau Valasiklovir ditujukan untuk pemberantasan infeksi HSV.6,7 Perawatan di Rehabilitasi Medis meliputi senam wajah atau pendidikan ulang neuromuskular wajah, elektrostimulasi, sinar inframerah, dan akupunktur, serta kebutuhan akan pelindung mata dan dinilai lebih lanjut dengan electroneurography atau electromyography dengan adanya kelumpuhan wajah lengkap.

**INTRODUCTION**

Bell’s palsy is a complex neuromuscular facial disorder of unknown etiology commonly affecting the motor neurones of facial muscles receiving their neurological innervations from the seventh cranial nerve (the facial nerve).1 Bell's palsy is named after Sir Charles Bell (1774–1842), who first described the syndrome along with the anatomy and function of the facial nerve.2 The facial nerve not only carries motor fibers including fibers to the stapedius muscle but also supplies autonomic innervations of the lacrimal gland, submandibular gland, sensation to part of the ear, and taste to the anterior two thirds of the tongue via the chorda tympani. Bell’s palsy accounts for almost three quarters of peripheral facial palsies and the annual incidence is about 15-30 patients per 100.000 annually. The sexes are affected equally. The median age at onset is 40 years, but the disease may occur at any age. The right and left sides of the face are involved with equal frequency.5,6

The cause of Bell palsy remains unknown, though the disorder appears to be a polyneuritis with possible viral, inflammatory, autoimmune, and ischemic etiologies.6 It disproportionally attacks pregnant women, patients who have diabetes, influenza, a cold, some other respiratory alignment or have undergone tooth root extraction. Some patients report exposure to an air-condition outlet, or an open window before the attack.3 Bell’s palsy has been attributed to an inflammatory reaction involving the facial nerve near the stylomastoid foramen or in the bony facial canal. Increasing evidence incriminates reactivation of herpes simplex or varicella zoster virus infection in the geniculate ganglion at least in some instances.7

Bell’s palsy may begin with symptoms of pain in the mastoid region and produce full or partial paralysis of movement of one side of the face. The corner of the mouth droops, the creases and skin folds are effaced, the forehead is unfurrowed, and the eyelids will not close. Upon attempted closure of the lids, the eye on the paralyzed side rolls upward (*Bell’s phenomenon*). The lower lid sags and falls away from the conjunctiva, permitting tears to spill over the cheek. Food collects between the teeth and lips, and saliva may dribble from the corner of the mouth. The patient complains of a heaviness or numbness in the face but sensory loss is rarely demonstrable. If the lesion is in the middle-ear portion, taste is lost over the anterior two-thirds of the tongue on the same side. If the nerve to the stapedius is interrupted, there is hyperacusis (sensitivity to loud sound).6,8

Treatment of Bell’s palsy varies, and no clear consensus exists. The aims of treatment in the acute phase of Bell's palsy include strategies to speed recovery and to prevent corneal complications. Most physicians prescribe corticosteroids as a primary treatment due to its potential to reduce swelling and inflammation. The addition of antiviral treatment (AVT) such as Acyclovir or Valacyclovir is aimed at eradication of HSV infection.9,10 The treatments at Medical Rehabilitation include facial exercise or facial neuromuscular re-education, electrostimulation, infrared rays, and acupuncture, as well as the need for eye-protective and further assessed with electroneurography or electromyography in the presence of complete facial paralysis. Most patients who suffer from Bell palsy have neurapraxia or local nerve conduction block at electromyography study. These patients are likely to have a prompt and complete recovery of the nerve. Patients with axonotmesis, with disruption of the axons, have a fairly good recovery, but it is usually not complete.11,12,13,14 “ Neuropraxia” was defined as absence of pathologic, spontaneous fibrillation activity and decreased or lost voluntary activity. “Axonotmesis/neurotmesis” was defined as pathologic, spontaneous activity in one or more facial muscles and decreased or lost voluntary activity. If the recording of some muscles clearly argued for neuropraxia but the other muscles for axonotmesi/neurotmesis, the recordings were classified as “mixed lesion”.15 The role of early surgery in treating Bell’s palsy has been controversial.16 In the past, surgical decompression within three weeks of onset has been recommended for patients who have persistent loss of function at two weeks, but the latest guideline suggest against the routine use of surgical decompression as a treatment choice in Bell’s palsy.2,12

Electrotherapy, massage, facial exercises, and biofeedback are different physical therapy modalities that have been used for the treatment of Bell’s palsy with a concentration on the role of exercise therapy more than other interventions. The aim of these modalities is to increase muscle and nerve function either through exercise or electrotherapy. Furthermore, thermal methods and massage work can decrease swelling and increase blood flow to the affected tissues, thereby increasing the amount of oxygen available to damaged hypoxic tissues with the aim of promoting recovery.19

Laser therapy is a modality that can be used in the treatment of Bell’s palsy. It is considered a non-invasive and painless therapeutic modality that can be used for any type of patient, including those who cannot use corticosteroids, such as diabetic and hypertensive patients.20 Approximately 70 to 80 percent of patients will recover spontaneously, up to 95% would be recovery without physical therapy if treated with prednisone and valacyclovir.17 A common short-term complication of Bell's palsy is incomplete eyelid closure which can lead to irritation and corneal ulceration. A less common long-term complication is permanent facial weakness, muscle contractures, synkinesis, sweating while eating or during physical exertion, ‘crocodiles tears’-lacrimation of ipsilateral eye during chewing and ‘jaw-winking’-closure of the ipsilateral eyelid when the jaw open.2,10 In this case report is about rehabilitation in a man with left Bell’s palsy.

**CASE REPORT**

**INITIAL PRESENTATION**

A 53 years old male come to the PMR department on July 5th 2018. Patient complained Left corner of patient’s mouth drooped since 7 months ago when he woke up in the morning. He saw that his left facial creases and nasolabial fold disappeared, the forehead unfurrowed, and the left corner of the mouth drooped. His left eyelids couldn’t close. While he ate the food and saliva pooled in the left side and spilled out from the left corner of the mouth. He felt numbness on his left face. He could taste food normally. Before that, he work as a sailor and often hit by the wind blowing directly to his face. Patient also often open the glass cover of the helmet while driving motorcycle. Sometimes he felt his left ear buzing, no pain behind his left ears, he had no history of trauma. Moreover, there were no weaknesses on his arms and his legs. This was the first time patient had an experience like this. On the second day onset, patient had already taken medicine from neurology department which are methylprednisolone 16 mg 3 times daily (5 days), followed 8 mg 3 times daily (5 days), ranitidine 150mg 2 times daily, and mecobalamin 500 mg 2 times daily. He experienced slight disturbances in activities in daily living (ADL) especially while feeding (chewing food and drinking), grooming (gargling while tooth brushing).

Table 2.UGO FISCH scale

|  |  |  |  |
| --- | --- | --- | --- |
| Position | Value | Percentage | Score |
| Rest | 20 | 70 | 14 |
| Frown the forehead | 10 | 70 | 7 |
| Closed eyes | 30 | 100 | 30 |
| Smile | 30 | 70 | 21 |
| Whistle | 10 | 70 | 7 |
| Total |  |  | 79 |

Table 3. House-Brackmann score to grade severity of facial nerve palsy by assessing motility of forehead, eye, nose, and mouth as 1-6.3

|  |  |
| --- | --- |
| **Grade** | **Score** |
| Normal, symmetrical function in all areas | I |
| Slight weakness on close inspection, complete eye closure with minimal effort, slight asymmetry of smile with maximal effort, slight synkinesis, absent contracture or spasm | II |
| Obvious weakness but not disfiguring, unable to lift eyebrow, complete and strong eye closure, asymmetrical mouth movement with maximal effort, obvious but not disfiguring synkinesis, mass movement or spasm | III |
| Obvious disfiguring weakness, inability to lift eyebrow, incomplete  eye closure, and asymmetry of mouth with maximal effort, severe synkinesis, mass movement, spasms | IV |
| Motion barely perceptible, incomplete eye closure, slight movement corner of mouth, synkinesis, spasm usually absent | V |
| No movement, loss of tone, no synkinesis, contracture, spasm | VI |

There is no history of hypertension, diabetes, toothache, ear infection, varicella, common cold in the past few weeks, and head trauma. There is no family member who had this problem. - Patient work as a sailor for 16 years and often hit by the wind directly in the face while on the ship. Patient also often open the glass cover of the helmet while driving motorcycle. Patient does not smoke and drink alcohol. Patient lives in the permanent house. The toilet is sitting toilet. The source of water is from PDAM and electricity from PLN. Patient used BPJS for paying the rehabilitation therapy. He worked on the expedition ship for about 16 years. He is the second from 5 siblings, he has one brother and three sister. On physical examination we found paralysis on left peripheral seventh cranial nerve with no other muscle weaknesses. The House-Brackmann’s score is grade III.

**DIAGNOSIS**

In medical diagnosis, the clinical diagnosis is Left Bell’s palsy 7th months of onset, the topical diagnosis Suspect facial nerve below the temporal part of facial canal. Etiological diagnosis is idiopathic. Functional diagnosis is disutbance in muscle power function, eating and drinking, mobility and transportation. The short term goal for management of this patient are to educate and reassure the patient about the condition, to facilitate or improve muscle contraction, to facilitate or improve facial symmetry. Whereas the long term golas for this patient are to prevent complication and to recover patient’s nerve function

**REHABILITATION TREATMENT PLAN**

1. Physiatrist

Explain the condition to the patient (its causes, incidence, prognosis and treatment), re-assure the patient, but be realistic (don’t give high expectations), advice the patient to following the rehabilitation program and to avoid therapy given by non-professionals, explain to the patient how the psychological state can affect the treatment, so avoid any emotional conflict and seek family or friend support to increase self-awareness and self-esteem, follow the given home program.

1. Physiotherapy

Modality: LLLT on left face (on the range of facial nerve ramification in eight different places), deep kneading massage on his left face, neuromuscular retraining on facial muscles in front of the mirror

1. Psychology

Mental support to reduce patient’s anxiety and giving a confidence that if he do exercise every day, his face may return to normal. Because this disease has approximately 80% of full recovery.

1. Home program

Warm compress on the left side of face for 10-15 minutes daily, massage on the left side of face, neuromuscular retraining on facial muscles in front of the mirror, blowing candle exercise, use straw while drinking, and gargle training

1. Social Worker

Gave advises and assisted family or friends in modifying his environment to support his treatment, such as wearing cover face helmet while driving motorcycle.

**DISCUSSION**

In this case report, we discuss about a 53 years old male patient who was diagnosed with Left Bell’s Palsy. The diagnosis was constructed by anamnesis and physical examination. From the anamnesis of this patient, there was asymmetry at the left side of face since 7 months ago when he woke up in the morning, left facial creases and nasolabial fold disappeared, the forehead difficult to frown, and the corner of the mouth drooped. When he ate, the food and saliva pooled in the affected side and spilled out from the corner. Before that, patient work as a sailor for 16 years and often hit by the wind directly in the face while on the ship. Patient also often open the glass cover of the helmet while driving motorcycle. There was no pain on his mastoid, he had no history of trauma. He could taste food normally. Moreover, there were no weaknesses on his arms and his legs. This was the first time patient had an experience like this.

According from the literature Bell’s palsy may begin with symptoms of pain in the mastoid region and produce full or partial paralysis of movement of one side of the face. The corner of the mouth droops, the creases and skin folds are effaced, the forehead is unfurrowed, and the eyelids will not close, permitting tears to spill over the cheek. Upon attempted closure of the lids, the eye on the paralyzed side rolls upward (*Bell’s phenomenon*). Food collects between the teeth and lips, and saliva may dribble from the corner of the mouth. The patient complains numbness in the left face but sensory loss is rarely demonstrable. If the lesion is in the middle-ear portion, taste is lost over the anterior two-thirds of the tongue on the same side.6,8 In this patient there was no impairment in sense of taste at 2/3 anterior of tongue which means the lesion was below the temporal part of facial canal (where the nerve gives rise to the stapedius and [chorda tympani](https://en.wikipedia.org/wiki/Chorda_tympani)).

Table 4. House-Brackmann score to grade severity of facial nerve palsy by assessing motility of forehead, eye, nose, and mouth as 1-6.3

|  |  |
| --- | --- |
| **Grade** | **Score** |
| Normal, symmetrical function in all areas | I |
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| Obvious disfiguring weakness, inability to lift eyebrow, incomplete  eye closure, and asymmetry of mouth with maximal effort, severe synkinesis, mass movement, spasms | IV |
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| No movement, loss of tone, no synkinesis, contracture, spasm | VI |

From the physical examination, patient was compos mentis, cooperative, communication and comprehension was good and vital sign was normal. Manual Muscle Test of all facial muscles was 2 except for M. Orbicularis occuli was 3 and M. Corrugator supercilli was 1. House-Brackmann grade was III. UGO FISCH scale was 79. To clinically assess the severity of peripheral facial nerve palsy various scoring systems are available. The initial severity of facial weakness provides valuable prognostic information for facial recovery. The most widely applied is the House–Brackmann facial nerve grading system (HBS) (table 1).3,11 In this patient the House-Brackmann score was III in the first visit then increased to II on the follow up.

UGO FISCH Scale is used to assess the condition of symmetric or asymmetric between the healthy side and the impaired side at 5 positions: At rest, smiling, closing eyes, raising eyebrows and whistling. The assessment of percentages are 0% is complete asymmetrical, there is no voluntary movement, 30% is symmetrical, poor, the recovery is likely closer to complete asymmetrical, 70% is symmetrical, fair, partial recovery is likely closer to complete symmetrical, 100% is symmetrical, normal or complete. In this patient the UGO FISCH Scale was 79 in the first visit then increased to 82 on the follow up. Clinical assessment should be repeated (approximately every month) to assess improvement.15

There are many other causes of acute facial palsy that must be considered in the differential diagnosis of Bell’s palsy. Lyme disease can cause unilateral or bilateral facial palsies; in endemic areas, 10% or more of cases of facial palsy are likely due to infection with *Borrelia burgdorferi*. The *Ramsay Hunt syndrome*, caused by reactivation of herpes zoster in the geniculate ganglion, consists of a severe facial palsy associated with a vesicular eruption in the external auditory canal and sometimes in the pharynx and other parts of the cranial integument; often the eighth cranial nerve is affected as well. Facial palsy that is often bilateral occurs in sarcoidosis and in *Guillain-Barré syndrome*. Leprosy frequently involves the facial nerve, and facial neuropathy may also occur in diabetes mellitus, connective tissue diseases and amyloidosis.. *Acoustic neuromas* frequently involve the facial nerve by local compression. Infarcts, demyelinating lesions of multiple sclerosis, and tumors are the common lesions that interrupt the facial nerve fibers; other signs of brainstem involvement are usually present. Tumors that invade the temporal bone (carotid body, cholesteatoma, dermoid) may produce a facial palsy, but the onset is insidious and the course progressive. All these forms of nuclear or peripheral facial palsy must be discriminated from the supranuclear or upper motor neuron (UMN) type.6

The diagnosis of Bell’s palsy can usually be made clinically in patients with (1) a typical presentation, (2) no risk factors or pre-existing symptoms for other causes of facial paralysis, (3) absence of cutaneous lesions of herpes zoster in the external ear canal, and (4) a normal neurologic examination with the exception of the facial nerve. Particular attention to the eighth cranial nerve, which courses near to the facial nerve in the pontomedullary junction and in the temporal bone, and to other cranial nerves is essential. In atypical or uncertain cases, testing for diabetes mellitus such as fasting glucose or A1C testing may be performed in patients with additional risk factors (e.g., family history, obesity, older than 30 years). Lyme antibody titers should be performed if the patient’s history suggests possible exposure. Patients with insidious onset or forehead sparing should undergo imaging of the head. In the presence of complete facial paralysis, nerve function can be further assessed with electroneurography or electromyography (EMG). EMG of the facial muscles determines signs of denervation and/or reinnervation as well as the degree of recruitment of motor units. In long standing denervation without signs of reinnervation, EMG might help in evaluating the facial muscle status whether there is complete muscle fibrosis or there are still viable contractile muscle fibers. Those with bilateral palsies or those who do not improve within the first two or three weeks after onset of symptoms should be referred to a neurologist.2,6,13 In this patient, we did not perform the laboratory testing because she did not have additional risk factors or possible exposure to Lyme disease. We also did not perform the imaging of the head because she did not have insidious onset. The facial paralysis was not complete, so we did not perform electroneurography or electromyography; also improvement at second follow up, so we didn’t referred her back to neurologist for further evaluation.

Generally, the treatment of Bell’s palsy can be conducted in 2 ways: first by using pharmacologic agents (medicaments) and second by rehabilitation. Physical medicine and rehabilitation (PM&R) also referred a physiatrist as a medical specialty concerned with diagnosis, evaluation, and management of persons of all ages with physical and/or cognitive impairment and disability. A physiatrist will work together with the other subunit such as physiotherapy, occupational therapy, orthotic-prosthetic, psychologist and medical social worker to achieve the best treatment for the patient.17

The aims of treatment in the acute phase of Bell's palsy include strategies to speed recovery and to prevent corneal complications. Most physicians prescribe corticosteroids as a primary treatment due to its potential to reduce the inflammatory process in Bell’s palsy and this facilitates remyelination of facial nerve. Prednisolone should be used in all patients with facial palsy of less than 72 hours duration who do not have contraindications to steroid therapy, with dose 60 mg per day for 5 days then reduced by 10 mg per day (for a total treatment time of 10 days).9,10,12 Patient had already taken methylprednisolone from neurology department since day 2nd with dose 16 mg (equally to 20 mg prednisone) 3 times daily (5 days), followed 8 mg (equally to 10 mg prednisone) 3 times daily (5 days).

The addition of antiviral treatment (AVT) such as Acyclovir or Valacyclovir is aimed at eradication of HSV infection. Current practice of adding AVT (either Acyclovir or Valacyclovir) in the regimen with Prednisolone may increase disease recovery rates compared with Prednisolone alone, but at this point this difference is not statistically significant. The possible explanation for the lack of any incremental effect of AVT is because Bell’s palsy is a post-infectious immune mediated facial neuropathy rather than direct viral infection.9 Furthermore, the latest guideline management for Bell’s Palsy suggest the combine use of antiviral and corticosteroids in patient with severe to complete paresis.12 This patient was not given AVT from neurology department.

It was reported by one investigator that injection of 500 µg of vitamin B12 (in form of methylcobalamin) given 3 times weekly for at least 8 weeks was of benefit in enhancing recovery in Bell’s Palsy.16 This patient was given mecobalamin 500 mg 3 times daily from Neurology Department. The routine use of eye-protective measures for patients with incomplete eye closure is a strong recommendation to prevent corneal complications, such as exposure keratitis, corneal ulceration and eventually loss of vision. Lubricating drops should be applied frequently during the day and ocular patches during the night is mandatory.10,12,14 This patient was not given artificial tears regularly and didn’t suggested to passively closed his eyes with finger or used eye patches before sleep because he could close his eyes with no gap.

Laser therapy has a favourable prognosis in the regeneration of peripheral nerves in both neurosensory and neuromotor deficits, 20 such as trigeminal neuralgia, neuropathy, lower back pain with sciatica, and herpes zoster.21 Application of a laser produces both local and systemic effects that can enhance the nerve regeneration process.21 Moreover, laser improves the recovery of the injured peripheral nerve and decreases post-traumatic retrograde degeneration of the neurons in the corresponding segments of the spinal cord.22 Research studies have shown that low level laser therapy (LLLT) increases the functional activity of the injured peripheral nerve, prevents or decreases degeneration in corresponding motor neurons of the spinal cord, and improves the axonal growth and myelinisation.23 This patient was given LLLT (880 nm) dose : 3,00 J/cm2, duty factor : 80%, frequency : 5,00 Hz, area : 1,00 cm2, time : 01:00 (m:s) at every point. Massage, which has frequently been prescribed for facial palsy, improves circulation and may prevent contracture.16 The guideline for soft tissue massage was 10 repetitions 1 or 2 times per day.17 This patient was given facial massage at the 7th month of paralysis onset according to recommendation from the latest guideline to give physiotherapy for patient with persistent weakness, but no recommendation for acute Bell’s Palsy of any severity.12 The stages of Bell’s palsy includes acute stage (1-7 day onset of disease), resting stage (8-20 days onset of disease) and restoration stage (21-90 days of disease).13

Facial neuromuscular re-education is a process of relearning facial movement using specific and accurate feedback to (1) facilitate facial muscle activity in functional patterns of facial movement and expression and (2) suppress abnormal muscle activity interfering with facial function.11 There are four distinct treatment based categories (initiation, facilitation, movement control and relaxation stages) matched with specific treatment techniques for each category. Surface EMG (s-EMG) has been advocated as an appropriate form of visual and/or auditory biofeedback for the re-education of muscle activity in facial movement disorders. However, if s-EMG is not available, a mirror may be substituted in order provide visual feedback as well. At the initiation phase, the exercises consisted of actively assisting specific facial movements which couldn’t be initiated or flaccid facial regions, and were advised to avoid mass movement patterns.1 On the first follow up (July 30th , 2018), the patient was instructed on using his fingers to passively move the left corner of his mouth into a ‘smiling’ posture. He was informed to then slowly release his finger pressure, all the while attempting to actively hold the ‘smiling’ posture with the involved musculature. In addition to the ‘smiling’ exercise, the patient was instructed to passively frown his left forehead with his finger and activate the appropriate musculature upon release of his passive support finger. When he began to able initiate slight movement at facilitation phase, the neuromuscular re-education exercises were prescribed on the basis of a participant’s impairments of facial motor control, with emphasis on small movements to gain symmetry between the affected and unaffected sides of the face.17

The exercise consisted of active and resistive exercise to increase facial movement excursion and facilitating the affected-sided musculature.1,11 The patient was instructed to perform slow, controlled, graded facial expressions to generate symmetry between the sides of the face with a mirror for visual feedback, and to use his finger to provide resistance to the desire facial movements with the precaution of muscular fatigue of the involved side and over facilitation of the uninvolved side. If there were any typical abnormal movement pattern or synkinesis developed, the meditation-relaxation strategies should be initiated as well as controlling synkinetic movements in addition to neuromuscular re-education therapy at the movement control phase. In the case of severe pan-facial tightness attributable to synkinesis and hypertonicity, the meditation-relaxation was the strong focus strategies. This patient didn’t develop any signs of synkinesis or the facial tightness at the initial examination and follow up (July 30th 2018), so the neuromuscular re-education performed at initiation and facilitation phase only. The typical guideline for neuromuscular re-education exercise was 20 to 40 repetitions 2 to 4 times per day.16 The results attained in facial training may be explained by the theory of nervous system plasticity.18

The use of nonspecific electrical stimulation of the peripheral facial neuromuscular system during the recovery process reinforces abnormal (synkinetic) patterns of facial muscle activity. Evidence from animal studies suggests electrical stimulation of facial neuromusculature during recovery from nerve injury may be disruptive to reinnervation. If the electrical stimulus is not carefully localized to the facial muscle nerve branch serving the specific intended facial movement or expression, peripheral nerve fibers within a nerve trunk serving many facial muscles will be simultaneously recruited. 11 Although many authors do not recommend ES for the fear of enhancing contracture, interfering with reinnervation or increasing cost of treatment, the findings of other authors may recommend its use. Electrical stimulation of muscle aims at preserving muscle bulk especially in complete paralysis; and it has also psychological benefit as the patient observes muscle contraction in his face that gives him hope for recovery from facial paralysis. ES was found to enhance axonal regeneration and skeletal muscle reinnervation in facial nerve lesion. The type of electrical stimulation should depend on the pathology of the facial nerve. If there is no electrophysiological signs of muscle denervation, faradic stimulation using 0,1-1 ms duration pulses delivered at a frequency of 1-2 pulses/s or more may be given for 50-200 contractions/session, 3 session/week until recovery. At PMR department, faradisation was given with ½-2 MA stimulation up to 30 impulses/minutes for each muscle, 90 times/day. Electrical stimulation may no longer be given once voluntary facial movement is regained even partially.16 Nevertheless, the latest guideline suggest against the use of ES in acute Bell’s palsy at any severity since the safety profile of such therapy is unproven, and there is an added cost.12 This patient was not given ES on his initial examination and the next follow up due to well recovery facial muscle and consideration of the risk and benefit of the treatment it self.

In the past, surgical decompression within three weeks of onset has been recommended for patients who have persistent loss of function at two weeks.2 It has potentially serious risks, including hearing loss (3-10% of patients), further damage to the facial nerve, and leaks of cerebrospinal fluids (4%).There is no evidence that surgical procedures to decompress the facial nerve are of benefit, that’s why patients should consider this option only if they have severe facial nerve degeneration on electroneuronography, if they are willing to accept the surgical risks and if the surgery is to be performed in an advanced treatment facility. The latest guideline suggest against the routine use of surgical decompression as a treatment choice in Bell’s palsy.7,12

The orthotics and prosthetics program is to do the installation of the “Y” plaster in the corner of the mouth in order not to fall. It is recommended that the plaster was replaced every 8 hours. It should be noted leather intolerance reactions that often occur. Installation of “Y” plaster is performed within 3 months if there has been no change in patients after undergoing physiotherapy.16 This patient didn’t use the “Y” plaster due to recovery at the follow up. About 80–85% of the patients recover spontaneously and completely within 3 months, whereas 15–20% experiences some kind of permanent nerve damage. About 5% may remain with severe sequelae. Long-term sequelae of facial nerve palsy may be persisting weakness, contractures, facial spasms, synkinesis, decreased tearing, crocodile tears, or psychosocial effects. The psychosocial impact of such a disorder can be life-altering in relation to social functioning. Indicators for poor prognosis include complete facial palsy, no recovery of symptoms by three weeks, age over 60 years, severe pain, herpes zoster virus, co-morbid status e.g. hypertension, diabetes, pregnancy and severe degeneration of the facial nerve shown by electrophysiological testing.1,3 At first visit, this patient had an anxiety and felt shame about his face. After gave him education about this illness, re-assured and consulted him to a psycholog, the shame and anxiety was decreased gradually. At the visit on November 12th 2018 (11 months of onset) his face was recovery with UGO FISCH scale was 100 and House-Brackmann score was grade I. The physical therapy and education was given to the patient about the possible complication or recurrence later on, so the patient could seek a medical help if needed.

**DAFTAR PUSTAKA**

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