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Review Article

Physiotherapy outweighed multiple therapy methods of bell's palsy: a review study

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Abstract

Although its etiology is not known, Bell's palsy is commonly based on trauma, infection, nervous damage due to surgery, genetic predisposition, autoimmune disease, temporal bone fracture, cerebellar arachnoid cyst and viral infection associated with vascular ischemia and inflammation. Viral infection due to herpes zoster and herpes simplex is the most common known cause of Bell's palsy. It is seen in about 20-35 out of 100.000 people each year. Its incidence is low in children under the age of 14 and it is more frequent within the range of 15-45 years. It is also known to occur equally in men and women. In the third trimester of pregnancy and postpartum in the first week, its frequency increases 3-fold. Unilateral paralysis, lagophthalmos, keratitis, droop of eyelid and edge of mouth, mouth and eye dryness, conjunctivitis and decreasing sensation and 2/3 taste of luxury in front of lingua are its common symptoms. 3 days from the onset of palsy, patients begin to use corticosteroid and antiviral drugs. 71% of patients had improvement after recovery but 29% of patients were shown to have hemifacial weakness and residual sequela throughout life. Synkinesis is the most common sequela of Bell's palsy. The effects of physiotherapy methods and their different combinations with other methods on prognosis were discussed in the treatment of Bell's palsy.

Keywords: Bell's Palsy; Acupuncture; Steroid; Antiviral; İdiopatic; Chiropractic; Proprioceptif; Facilitation.

Bell's palsy, in other words peripheral facial dysfunction, is the idiopathic, acute and unilateral paralysis. A great number of resources define Bell's palsy as one-sided lower motor neuron weakness of facial nerve (1,2). Although the exact etiology of Bell's palsy, which is the most common facial paralysis, is not known, it is known to develop due to viral infection and autoimmunity caused by latent herpes simplex and herpes zoster viruses (3). Japanese researchers have even changed Bell's palsy in literature as herpetic facial paralysis. They have observed herpes simplex virus only around the facial nerve ganglions of patients with Bell's palsy. In addition, vascular ischemia, temporal bone fractures, mechanical decompressions, genetic predisposition, cerebellar arachnoid cyst formation and the swelling of the 7th cranial nerve are among the reasons for the formation of Bell's palsy (1,2,4). Ear diseases cause Bell's palsy in children with acute otitis media or masked mastoiditis. Inflammation resulting from direct harm of organisms in the middle ear causes pressure on the nerves in facial canal. The growth of inflammation in the middle ear causes crack in fallopian canal and the facial nerve within to compress (5). The objective of the review is to research the benefits of physiotherapy methods used in the treatment of Bell's palsy and to address the effects of combinations applied with different treatment methods on prognosis.

Pathophysiology

Nerve inflammation that occurs along the facial canal and compression and demyelinization of axons that occur as a result of this are accepted as the pathophysiology of Bell's palsy (6). According to Aviel, increase in B lymphocyte rate and decrease in T lymphocyte rate is the pathophysiology of Bell's palsy (3). Another view originates from autoimmune system that develops against myelin basic proteins. Viral infections or the reactivation of viruses in latent stage stimulate the autoimmune system against peripheral nerve myelin units (3,4). This situation causes demyelinization of cranial nerves, especially facial nerve. Anomalies in intracanalicular and labyrinthine segment of facial nerve have also been observed in Bell's palsy. In addition, lymphocyte sensitivity has been observed

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against P1L proteins, as well (3,7). According to Liston and Kleid, histological changes occur in peripheral facial paralysis (3,4,8). Inflammation cells are reported to invade nerve cells from internal acoustic meatus to stylomastoid foramen. Thus, macrophage cells have been observed to damage myelin sheaths of nerve cells and the edema that occurs as a result of the damage has been observed to cause an increase in the area between neurons. With the increase in the area, communication between neurons and response to stimulant are delayed. This situation causes Bell's palsy pathologically (3).

Etiology, Epidemiology and Incidence

Although the exact etiology of Bell's palsy is not known, it is known to develop due to latent herpes simplex and herpes zoster and autoimmunity. Myelin triggers autoimmune system paralysis that develops against basic proteins. This situation causes demyelinization of the cranial nerve, especially the facial nerve. In addition, varicella zoster virus has also been proven to be the cause with serological evidence (3,9). Viral infection, vascular ischemia, inflammation, temporal bone fractures, mechanical decompression and cerebellar arachnoid cyst are also among the reasons for Bell's palsy. In addition, influenza, upper respiratory tract infections or the flu, tooth extraction and being in areas with air condition or wind are also among the reasons from paralysis. According to Schulz, viral reactivation is activated by metabolic change, stress factors, infection, cold airflow, psychological stress and immunosuppressant (3,4,10). Strong wind increases the risk for viral shedding and reactivates latent herpes infection. According to some studies, air conditions together with viral neuropathology also trigger Bell's palsy formation (3,4). Diego et al. reported that there was a positive correlation between low temperature and Bell's palsy, while they reported no correlation between atmospheric pressure and Bell's palsy. The common view about the effect of meteorological factors is that Bell's palsy' occurs most commonly in autumn. In addition, the speed of wind is a significant factor and it is positively correlated with Bell's palsy (4). On the other hand, the edema in the 7th cranial nerve causes ischemia and compression in cranial nerves which are in fallopian canal inside mastoid bone. This situation causes unilateral total weakness and paralysis (5). In addition, regular statin use also causes increase in Bell's palsy prevalence (11). According to Dixit, immunoglobin M (IgM) antibody is positive for Hepatitis E Virus (HEV). The association between Bell's palsy and HEV are supported with clinical, biochemical and serological evidence. The fact that HEV clinical findings and Bell's palsy clinical findings are parallel and also the fact that they form the basis of neurological problems support this view (3,4,9). Masked otomastoiditis blocks the ventilation functions of mastoid cells and causes inflammation by harming the organisms in the middle ear directly. As a result of this, inflammation compresses the nerves in the facial canal and causes ischemic neuritis formation. This situation triggers the formation of Bell's palsy (5). Bell's palsy is more frequent between the ages of 15 to 45 and the risk of Bell's palsy is three-fold in the third trimester and postpartum first week of pregnancy in women (12). Bell's palsy risk factors are frequently seen in pregnancy, diabetes mellitus, hypertension and individuals older than 60. Its incidence is 20-35 in every 100.000 people. While its incidence is 92/100.000 in women, it is 82/100.000 in men. In addition, according to some sources, the incidence of Bell's palsy increases in cold seasons. There is a 18% increase in patients who are admitted with a diagnosis of Bell's palsy in December and January (6).

Symptoms

It involves all the muscles in the right or left side of the face and on the paralyzed side, the face becomes motionless and like a mask. On the paralyzed side, the person cannot lift the eyebrows, wrinkle and cannot keep the eye shut as a result of the pressure on the paralyzed side. Due to the involvement of pars palpebralis of orbicularis oculi muscle, the eyelids cannot be shut.

Because the eye is open due to orbicularis oculi muscle involvement and because puncta lacrimalia cannot absorb, tears go from orbicular canal. Conjunctival membrane which closes the interior surface of the eyelid the anterior surface of the eye ball dries and conjunctivitis occurs. The mouth pulls to the side of the face which is not affected and the mouth and the eye corner on the paralyzed side droops. Since buccinators muscle is not working, food bites are collected in the vestibulum oris of the paralyzed side while chewing. Asymmetry occurs on the face while laughing and the mouth pulls to the side of the face which is not affected. There arise difficulties in closing the eyelids and the patient has to show effort. Synkinesis, reflex moves like the closing of the eyelid while laughing, occurs. Blanking occurs in the sulcus nasolabialis of the paralyzed side (9,13). Retroauricular pain is reflected on the chin and occiput. Intolerance to sound (hyperacusis) occurs (2). Decrease in blinking reflex (14), lagophthalmos and keratitis (corneal inflammation) occur (6).

Bell's Palsy Diagnosis and Assessment Methods

First, anamnesis and physical examination should be performed for Bell's Palsy diagnosis. Later, diagnostic examinations are made. It should be kept in mind that peripheral facial paralysis progresses within a few days and progression reaches the highest level in the third week. After the diagnosis of the patient is made as Bell's palsy as a result of the symptoms, the patient is checked for sequels such as synkinesis, facial contracture and facial pain caused by 7th cranial nerve dysfunction (2, 15). Face Clinometric Evaluation (FaCE), Face Grading Scale (FGS) and House-Brackmann Scale are used to find out the progression and level of Bell's palsy (8,16). In addition, there are definitive diagnoses between Bell's palsy and post-traumatic facial paralysis, Lyme disease, Melkersson-Rosenthal syndrome, Sjögren syndrome, cholesteatoma. Of these, although Melkerson-Rosenthall syndrome, which is also known as recurrent paralysis, has symptoms such as blanking of sulcus nasolabialis, not being able to close the eyleids and fissure on tongue

which are similar to Bell's palsy, its pathophysiology is different (7,16,17).

Definitive Diagnosis in Bell's Palsy

Audiometric Analysis: In Bell's palsy patients, cochlear nerve and facial nerve course together anatomically, thus, the threshold of distinguishing speech is measured with stapes reflex. Hyperacusis (being sensitive to sound) is seen in 30% of patients with Bell's palsy.

Topographic Analysis: The aim is to find out the level of damage in facial nerve. It is examined as Schirmer test, stapes reflex test, taste test, saliva secretion test, saliva pH test. Major function of superficial petrosal nerve is assessed with Schirmer test. After same size paper is kept on both eyelids for 5 minutes, they are removed. More than 25% difference between the moist on both papers is recorded as Bell's palsy diagnosis. The objective of Stapes reflex test is to assess the stapes muscle function that stimulates stapes muscle. Stapes reflex cannot be taken from the paralyzed side of Bell's palsy patients. In 69% of patients with Bell's palsy, stapes reflex disappears. In the taste test, chorda tympani, which is a branch of the facial nerve carrying the sense of taste in 2/3 anterior part of the tongue, is taken as the basis. Sweet, salty, sour and bitter solutions are used to assess the function of chorda tympani. In addition, electrogustometry is preferred in this test since it is fast and easy. In a study conducted with electrogustometry in the acute phase of Bell's palsy, it was found that the threshold value of taste were different in both sides of the tongue (16,17). The sense of taste has been seen to collapse on the paralyzed side. In saliva secretion test, saliva flow is measured by placing catheter on Wharton canals. Thus, secretory function of chorda tympani, which is a branch of facial nerve, is measured. In saliva secretion, more than 25% decrease was found between the paralyzed side and the non-paralyzed side. Thus, a mouth dryness symptom was reported in paralysis (12,16). In saliva pH test, less than 6.1 submandibular saliva secretion pH is accepted as a proof that there may be recovery in paralysis (12). In addition, facial expression analyses are made in photographs and video in assessing the patient. With FaCE, the patient is asked to give personal report about the facial expressions they cannot do. With FGS, researchers ask the patient to make 5 voluntary face expressions (smiling, curling lips, raising eyebrows, frowning, closing eyes tightly) in resting position and check for synkinesis. This grading scale assesses what a patient can do over 100. Low scores get facial dysfunction diagnosis. Electrophysiological tests are also conducted for diagnosis. Nerve Stimulation Test-NET is an electro physiological test and it is first applied on the non-paralyzed side. Researchers place electrodes near the stylomastoid foramen, increase the current gradually and record the threshold value in which contraction occurs. They apply the same procedure to the paralyzed side and they record as bad diagnosis if the threshold difference between is more than 2-3 Ma. However, NET test cannot be applied in bilateral paralysis. Another test is Maximal Stimulation Test (MST) and its objective is to stimulate all the fibrils and measure the maximal current. While making a comparison, the measurements of the both sides are assessed as equal, decreased or non-response. Decreased or non-response show advanced degeneration in the patient. While the rate for recovery is 88% in Bell's palsy patients with normal MST, this rate falls to 27% in patients with decreased MST. Since muscle movements are assessed visually in MST, it is subjective and its reliability is controversial. Another electrophysiological test is Electroneurography (ENoG). In this test, bipolar electrode is placed on sulcus nasolabialis and muscle contraction is assessed. The difference between the paralyzed and non-paralyzed sides is expected to be 3%; however, if this difference is more than 10%, this is an indicator of the patient's bad prognosis. The last electrophysiological test is Electromyography (EMG). With this test, action potentials that occur during muscle contraction are measured with electrodes. According to researchers, NET, ENoG and EMG tests should be conducted on a Bell's palsy patient starting from the third day. If the patient is found to have 90% degeneration as a result of assessments, direct surgical intervention and postop physiotherapy are conducted (10,12).

Types of Treatment in Bell's Palsy'

Types of treatment after assessment are grouped in three as medical and surgical, physiotherapy and combined therapy.

Medical and Surgical Methods

Prednisone with glucocorticoide effect is given in periods of 1 month or 3 months in the acute period (14,18). In 3 month-period, with the decrease in axonal degeneration of the patients as a result of steroid use, positive differences are obtained in ipsilateral blinking reflex (14,19). In some resources, it has been proven that corticosteroid treatment started three days after the onset of Bell's palsy is more effective (18,19). Dehydration, abrasion, dryness and conjunctiva may occur in the cornea. Because of this, synthetic tears during the day and night pomades have a positive and protective effect. In advanced Bell's palsy patients and those who do not respond to steroid treatment, the method most commonly used are transmastoid or subpetrous decompression. Decompression is seen to protect the non-paralyzed facial nerve fibers and cause axonal regeneration. Physiotherapy after operation provides reeducation in facial muscles. In addition, transcutaneous electrical current and golden plaque applications are also used for reinnervation today. In golden plaque application, with the help of golden implant placed on the upper eyelid, the eyelid can be closed (3,12). Tightness and swelling are controlled with palpation on the paralyzed side of the patient with Bell's palsy. If tight fibers are felt in the cervical or trochal area, high speed and low amplitude chiropractic manipulation is applied on the tetanus area. Later, on the paralyzed side, high voltage galvanic current is applied on the facial muscles with pencil electrodes (5). If the reason for Bell's palsy is tumor, arachnoid cyst or lymphadenopathy, transmastoid decompression is applied (4,5). Another surgical method

in Bell's palsy is cutting from the posterior and inferior sides of hypoglossal nerve angulus mandibulae and anastomosis the proximal tip to the distal of n. facialis. Hypoglossal nerve and facial nerve are peripheral nerves, thus, their regeneration is possible (8,20).

Physiotherapy Methods

Following surgical interventions, neuromuscular facilitation and massage are given to provide nerve stimulation and functionality (12). Proprioceptive Neuromuscular Facilitation (PNF) therapy is given bilaterally and symmetrically in Bell's palsy. All the muscle groups are stimulated with pivot points of mouth, nose and eye. The objective is to release movement in paralyzed side by giving resistance with strong moves. Thus, the strength is spread (1,21,22). For example, if the left eyebrow elevation of the patient is weak, first pressure and stretching is applied on the lower inner direction. Later, the patient is asked to raise his/her eyebrows. Down-resistance is applied to the movement of the right eyebrow and eyebrow elevation is revealed in the left side. While facial movement in the direction of elevation is about neck extension, facial movement in the direction of depression is about neck flexion. Neck rotation can stimulate the facial muscles on the side it is turned proprioceptively. In order to stimulate facial muscles respectively in PNF, hand contact and order should be in harmony. How should this harmony be?

Frontalis muscle: Provides the upper elevation of the eyebrow. The hand is placed on the forehead of the patient, with the fingers looking down. Bilateral downward and inward resistance is given in diagonal direction. The patient is asked to raise his/her eyebrows. Thus, the desired movement will occur in the paralyzed side with the resistance in the non-paralyzed side. Corrugator supercilii muscle: It is the frowning muscle. Fingers are placed to the inner part of the eyebrows. Resistance is given upward and outward. The patient is asked to raise his/her eyebrows. Thus, the movement that occurs against resistance on the non-paralyzed side will reveal the movement in the paralyzed side.

Orbicularis oculi muscle: It is the muscle that closes the eyelid tight. The thumb and index finger of both hands are placed to encircle the patient's eyes. The patient is asked to close his/her eyes tight. The physiotherapist tries to open the patient's eyes on the diagonal and opposite direction. Thus, the movement of pressure on the non-paralyzed side forms the movement of closing the eyes on the paralyzed side.

Levator palpebrae superioris muscle: It is the muscle that lifts the eyelid upward. The pressure is given downward. The patient is asked to raise his/her eyelids. Thus, strength spreads to the paralyzed side from the non-paralyzed side.

Procerus muscle: It is the nose wrinkling muscle. The index fingers of both hands are placed on two sides of the nose and downward inward resistance is given. The patient is asked to wrinkle his/her nose. Thus, the strength that is revealed from the non-paralyzed side to the resistance spreads to the paralyzed side. Orbicularis oris muscle: It is the muscle that closes the mouth and pushes the lips forward. Fingertips are placed on the sides of the mouth and resistance is applied to open the mouth.

Thus, the non-paralyzed side will cause strength by resisting and cause the desired movement on the non-paralyzed side. Risorius muscle, zygomaticus major muscle and zygomaticus minor muscle: They are the muscles that form the smiling mimic. The resistance is given downward and inward. The patient is asked to smile.

Buccinator muscle: Resistance is given outward with abeslang. The patient is asked to make a laughing or sucking move. It is also a type of exercise for mimics.

Levator labii superioris muscle: The upper lip is raised upward. Resistance is given downward and inward. The patient is asked to raise his/her upper lip upward. With the mouth corner on the non-paralyzed size raised upward and resistance, desired movement occurs on the paralyzed side. Depressor labii inferioris muscle: It is the muscle that pulls the lower lip downward. The patient is asked to push the lower lip downward. The strength caused by the non-paralyzed side to the resistance supports the formation of the desired move.

In PNF, coordinated moves are done to stimulate especially eye and tongue movements. The eye looks down with neck flexion and up with neck extension. Thus, in a patient with Bell's palsy, the muscles innerved by oculomotor nerve are stimulated. Tongue elevation occurs with neck extension. Tongue depression occurs with neck flexion. Thus, tongue muscles innerved by hypoglossal nerve are stimulated. Neck rotation caused lateral movement of the tongue and with the rotation to the paralyzed side, the muscles on the paralyzed side are stimulated. The tongue is depressed by opening the mouth and the tongue is elevated by closing the mouth (8,16,21).

Physiotherapy methods with medical and surgical methods

In Bell's palsy, a great number of sequels can occur following surgical intervention. These sequels are hemifacial spasm, motor deficit, syndrome of crocodile tears, synkinetic contraction, Marcus Gunn syndrome, and synkinesis. Another objective of Bell's palsy rehabilitation is synkinesis method. Specific neuromuscular reeducation method is used for this (10,15). Before therapy, facial muscles are stimulated with electric stimulation and then reeducation therapy is conducted. The patient is given mimics to do at home in front of the mirror. Sometimes, the patients are given botox besides face rehabilitation. Thus, facial symmetry is aimed. Recently, cross-face nerve graft is used in idiopathic facial paralysis. It is the operation of taking nerve graft from the leg and the nerve tip of the non-paralyzed side is conveyed to the nerve tip of the paralyzed side MIMIC exercise (MIM) are face expression

exercises in front of a mirror (blowing up a balloon, sending kiss, raising eyebrows, closing eyes tightly) and they provide neural and muscle stimulation especially on the paralyzed side. Since rehabilitation provides facial muscle control and functionality, it has an important place in Bell's palsy (10). In pediatric and adult Bell's palsy patients, acupuncture is applied as an alternative therapy in addition to physiotherapy. Acupuncture as a supplement to medical treatment and physiotherapy has been shown to have a positive effect (1,16,23,24).

CONCLUSION

In general, researches state that corticosteroid therapy and physiotherapy should be started in the acute phase of Bell's palsy. With this combined therapy, inflammation has been found to decrease and significant decreases have been recorded in autonomic dysfunction and motor synkinesis (2,3). Steroid use decreases nerve compression, neural edema, ischemic area in facial canal and physiotherapy provides nerve reeducation (12,19). According to researches, routine corticosteroid use and physiotherapy should be started within the 72 hours following the onset of Bell's palsy (9,12,19,25) because 6 weeks after the onset of Bell's palsy, it is difficult to regain functions in treatment (6). However, this is not true for all ages. Although Bell's palsy is not common in children, swelling in the soft tissue inside the external hearing canal, fluid increase in mastoid canal and tympanic cavity and inflammation show otitis media or mastoiditis (1,7,25). Ear diseased, acute otitis media or masked mastoiditis cause Bell's palsy in children. Corticosteroid, which is applied within 72 hours following the diagnosis of Bell's palsy, should not be given to children routinely to decrease compression on cranial nerve resulting from inflammation, and to decrease neural edema and ischemic area. Physiotherapy program should be started based on the assessments of the 2-6th week of the onset of the disease and 26-42nd week of the treatment (1,25) because corticosteroids affect the metabolism of children differently when compared with adults and their routine use can cause hyperglycemia, mood changes, increase in gastritis risk, peptic ulcer formation and gastrointestinal hemorrhage (1). Anatomically, the sectional area of the facial nerve in children is smaller and thus they have less damage from neural edema with compression (1, 5). As long as it is not routinely used, steroid and Neuromuscular Reeducation (NMR) and PNF exercise given to children within 3 days following the diagnosis cause more useful results (1,19,21). Mizumachi et al. compared the recovery period of Bell's palsy pediatric patients who received steroid, B12 vitamin and physiotherapy with those who did not receive any therapy. As the result of one-month long therapy, complete recovery is seen in all patients (1,19). However, this situation does not give the same results for children with grade 4 paralysis according to House-Brackmann scale. Intravenous antibiotics that are ceftazidime and PNF are recommended to children with severe lesion. Thus, as a result of the 3-month long antibiotic treatment on children, inflammation is completely cured and PNF application provides symmetric muscle contraction. As a supplement therapy to decrease the period of treatment, in Bell's palsy treatment resulting from otitis media, parental antimicrobial treatment and myringotomy are applied (1,2,12). Thus, with the decrease in edema, the pressure on the 7th cranial nerve decreases and muscle reeducation occurs with physical treatment. If myringotomy fails, mastoidectomy and facial nerve decompression are applied (1,2,5,12,21,25). In a study by Remli et al., facial nerve conduction and blinking reflex of 21 Bell's palsy patient group were analyzed. The results were planned to be analyzed within 10 days, 1 month and 3 months period. These 21 patients were given prednisone and PNF and mimic exercises twice a day. Their progress was followed with House-Brackmann scale, visual analogue scale and facial clinometric measurement. Within this 3-month-long period, significant increases were found in the patients' axonal degeneration, ipsilateral blinking reflex, eye movement scores and lacrimal control scores. The most significant positive phenomenon was observed in the increase in blinking reflex with the decrease in axonal degeneration in the first and third month (14). Oral corticosteroid is no successful in complete recovery of acute Bell's palsy even though it is high dose and only for a short time (26). In the acute period of treatment, only steroid and antiviral drug use was observed not to have such a big effect on eye complications frequently observed in elderly people (6,10,12,25). In patients who do not respond to steroid therapy and show 90-95% degeneration in electro diagnostic tests, surgical treatment is conducted within 2-4 weeks after the onset of symptoms (5,10). Surgical treatment is frequent especially in neuroma, axonotmesis and neurotmesis injuries. The most frequently used surgical method is transmastoid or subpetrous decompression. The objective of decompression is to keep the remaining facial nerve fibers and allow axonal regeneration (10). Coordination exercises and relaxation exercises before surgery are also applied in preop period. Transcutaneous electrical current also provides reinnervation (12,21). In patients supported with physiotherapy following post-op, significant muscle activity is expected within 6-20 weeks. For the education of the muscle grafted to the patient post-op, lip mobility exercises and symmetric laughing exercises are given. Physiotherapy has a significant place in muscle education after grafting (6,7,10). If the patient does not show the expected moves, the treatment program should be developed like that of non-operated patients. A great number of patients avoid surgery and prefer non-invasive treatment methods.

With recently popular chiropractic method, high speed and low amplitude therapy is applied on the cervical and thoracic vertebrates on the paralyzed side manually. Thus, the tense fibers in cervical and thoracic areas and tension in trapeze muscle decrease. With the patient's treatment program organized as chiropractic manipulation, interfarencial muscle stimulation and hydroculation,

90% recovery occurs within a year. Chiropractic therapy programs can give better results with facial muscle exercises (4,5,8,10,25). Acupuncture therapy, which is defined as an alternative therapy method, is applied on both children and adult Bell's palsy patients. However, its effect on Bell's palsy is controversial. In a systemic research conducted by a group of researchers, while acupuncture was called a therapy with positive effects of Bell's palsy, its complication incidence was ignored (16). Other researchers compared acupuncture and medication therapy (vitamin B+steroid) and stated that acupuncture was better when compared with medication therapy. However, this issue could not find sufficient evidence and in other words, Vitamin B was combined with steroids in other studies. The result of this study: acupuncture should be performed as a supplement therapy in addition to patients' physical examination (23,24). Ahn et al. compared acupuncture techniques applied for 6 sessions for three weeks. They compared traditional acupuncture and combined acupuncture in terms of relieving retroauricular ache, headache and pain resulting from trigeminal neuralgia and observed that both acupuncture techniques had the same effect in decreasing pain score (24). In short, adding acupuncture as a supplement to Bell's palsy treatment makes positive contribution to prognosis. As a conclusion, it was generally accepted that corticosteroid and physical therapy exercises should be started within 72 hours following the onset of Bell's palsy in order to decrease the compression resulting from inflammation (1,2,10,14,19).

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