Original Research Article

Evaluation and management of facial nerve paralysis in MGM medical college

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Abstract **Background:** The management of facial paralysis is developing gradually. It is very important to understand the facial nerve anatomy and the several methods of assessing the degree of facial nerve injury for the successful management. In case of nerve transaction direct coaptation and inter-positional nerve grafting lead to best outcome. A nerve transfer should be the option in case of intact motor end plates and where grafting is not feasible. Regional muscle transfer or free flap reconstruction should be employed in case of complete muscle atrophy. Static procedures offer some benefit when dynamic reanimation canneo be undertaken. So there are several ways to assess and manage facial paralysis. Hence the present study is undertaken to find out the different aspects of evaluation and management of facial paralysis. **Methods:** This was an observational review study in which we discussed the different etiology of facial nerve paralysis, and the different aspects of evaluation and management of the same. **Conclusion:** Facial nerve palsy is commonly seen and requires prompt evaluation and diagnosis. The majority of cases can be managed with medical treatment alone but ENT or neurology referral should be considered in atypical cases.
Key Word: facial nerve.

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INTRODUCTION

Paralysis of the facial nerve is a well-recognized phenomenon and Bell's palsy is the commonest form. **Roob *et al.***[1] reports that it occurs most commonly in children and young adults. In most cases no definite aetiology is identified (idiopathic/Bell's palsy). The facial nerve leaves the brainstem and enters into the internal auditory canal with the auditory nerve. It has three bones inside the temporal bone:1) greater superficial petrosal nerve- to lacrimal glands and glands within nasal and platal mucosa, 2) Chorda tympani- to taste buds and anterior two-thirds of the tongue, and 3) stapedial nerve- stapedius muscle in middle ear. As it exits the skull, it divides into branches within the parotid gland to supply the muscles of facial expression. The objective of this work is to outline the different etiological factors of facial nerve paralysis and to find out the different aspects for the evaluation and management.

Etiology: There are several etiologies behind facial nerve paralysis. Different causes of unilateral facial paralysis should be considered that include: idiopathic, traumatic infective, neoplastic, congenital and autoimmune factors. 70% of facial nerve paralysis is diagnosed as Bell's palsy with 11-40 new cases per 100000 each year[2,3]. Bell's palsy is the commonest finding and constituted for almost 90% of facial paralysis. About 10% of Bell's palsy patients experienced recurrence after a mean latency of 10 years[3]. In the last years there has been a growing range of studies proposing that the Bell's palsy origin is the reactivation of Type 1 Simple Herpes Virus, which is latent in the geniculate ganglion[4]. The second most common cause of facial paralysis is traumatic injury involving 8-22% of cases[5]. A higher incidence of these injuries occur during delivery either due to birth canal trauma or forceps delivery[6]. The other etiologies of facial paralysis are: surgical trauma, penetrating parotid or middle ear trauma,

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barotrauma, facial fractures, and temporal bone fractures[7]. The next common etiology of facial nerve paralysis is infection[5]. The herpes zoster is latent in the geniculate ganglion and its reactivation generally originates the Ramsay Hunt Syndrome, in which the patient presents with acute facial paralysis followed by severe pain and vesicular eruptions of the external auditory meatus: only 50% of these patients recover completely[8]. Acoustic neuroma of the adjacent nerve VIII or other cerebellopontine angle tumors, such as a meningioma or a tumor of the glomus jugulare are typically related with facial nerve weakness after surgery, in contrary to a preoperative facial palsy. Malignant tumors of the external auditory canal, like squamous cell carcinoma or an adenoid cystic carcinoma, can spread into the temporal bone and lead to proximal facial nerve paralysis. Malignant parotid tumors (e.g., mucoepidermoid carcinoma, adenoid cystic carcinoma) and facial nerve schwannomas cal also lead to facialnerve paralysis. Metastatic lesions from the orbit, lung, breast or kidney can also very rarely affect the facial nerve. Malignant facial skin lesions (e.g., basal cell carcinoma, squamous cell carcinoma) may lead to peripheral facial paralysis. In particular, perineural spread of cutaneous squamous cell carcinoma is a common cause of slowly progressive facial palsy. Bilateral facial paralysis are rarely found; as nearly 2% of facial paralysis; and typically represents a systemic disorder with multiple manifestation[9]. Bilateral palsy is important as it is much more likely to represent a systemic manifestation of the disease, with fewer than 20% of cases being idiopathic. Lyme disease represents a higher incidence of bilateral facial nerve palsies, involving around 35% of cases. Several other important differential considerations refer Guillain-Barre syndrome, diabetes, and sarcoidosis. Neurological causes of bilateral facial nerve palsy include Parkinson disease, multiple sclerosis, and pseudobulbar/bulbar palsy[10]. Significant disfigurement can be resulted by facial nerve paralysis, which have severe implications on both the emotional and physical well-being of the patient. Lacrimation, brow ptosis, ectropion, epiphora, and lagophthalmos are the ophthalmologic consequences of facial nerve paralysis. These consequences may lead to corneal damage from exposure keratopathy which potentially procced to blindness or globe rupture[11,12]. Loss of muscular support to the nasal valve can be resulted to nasal obstruction. Ineffective contaction of the perioral musculature can lead to insufficient oral competence, poor swallowing function, dysarthria and ptyalism[13]

Clinical Evaluation:

Upper Eyelid retraction contributes to lagophthalmos caused the unopposed action and tone of the levator and Muller's muscles.

Blink reflex is usually missing in facial nerve paralysis instead there is only a slight flutter.

Eyelid Closure assess lagophthalmos on gentle and forced closure. The extent of lagophthalmos will often dictate the extent and timing of medical and surgical intervention to protect the eye.

Brow ptosis leads to secondary eyelid ptosis interfering with visual acuity. At the initial stages of weakness this can be helpful in protecting the globe.

Lower eyelid involvement evaluates paralytic ectropion and should be considered to medical canthal tendon laxity.

Midface: Evaluation of midface position has a significant mechanical effect on the lower eyelid, nasolabial fold, cheek tone and elevation.

Mouth: assessment of mouth symmetry and ability to drink and whistle.

Neck: Evaluation of platysma muscle strength.

Hearing: Evaluation of hearing can be grossly tested by gentle finger rubbing to compare hearing on each side to detect severe loss.

Corneal Sensation: This is very crucial and should be performed very carefully and compared to the normal side. Acute loss of corneal sensation indicates a guarded prognosis for patients with facial nerve paralysis and requires aggressive management.

Bells Phenomenon: It should be considered because patients with good Bell's phenomenon may tolerate poor closure much better than those with poor Bell's phenomenon.

Tear Finction: A Schirmer's test is performed to determine the tear production. If the salivatory muscles or branches to the lacrimal gland have been affected the tearing may be affected in facial nerve paralysis. On the other hand tearing may be increased with aberrant regeneration or reflex tearing from ocular irritation secondary to exposure and drying of the ocular surfaces.

Synkinesis: spontaneous twitching or cross innervations due to aberrant regeneration may occur in long standing or recovering facial nerve paralysis. The most noticeable areas of synkinesis involve the orbicularis oculi, nasobial fold area and mouth.

Assessment: Proper characterization of facial nerve paralysis should be accomplished to provide precise patient counseling regarding evaluation and management. The House-Brackmann 6-point scale of facial nerve function is the most frequently used tool for assessing the severity of facial weakness[14,15]. This scale is not satisfactory in case of characterizing facial paralysis localized to one particular facial distribution. Terzis-Noah, BurresFisch, Nottingham, and Sunnybrook are the other scales used to assess the severity of facial weakness[16-19]. Proper assessment and documentation should include a detailed description of the status of motion of the upper,

middle and lower face. Special consideration should be made on the eye not only in terms of eyelid closure, lower lid laxity and brow height but also on visual acuity, existence of Bell's palsy and corneal irritation. Nasal valve collapse and oral competence should be given special consideration beside the overall symmetry. Different tests are used to evaluate the lesion of the facial nerve like: Schirmer, Stapedial Reflex, Electrogustometry and Salivary Flow are important to set up the topodiagnosis or the probable lesion place in addition to contribute for the prognosis evaluation. However, imaging including Computed Tomography and Magnetic Resonance are also relevant to compose the diagnosis. Electrophysiological assessments are usually very crucial for the prognosis and the indication of some more aggressive treatment. Tests like: Hilger, Electroneurography (ENog) and Electromyography (EMG) are largely used in day to day life. Enog is an objective test that measures evoked compound muscle action potentials using skin electrodes. Nerve injury is expressed as percentage of function relative to the normal side. When ENog is undetectable, EMG testing can be attempted. EMG measures voluntary muscle response with needle electrodes placed in the target muscles that detect action potentials during muscle contraction with a functioning nerve. Muscle that has been denervated displays fibrillation potentials, while muscle that is in the process of reinnervation demonstrates polyphasic potentials.[20] Electrical silence in a patient with long standing facial paralysis indicates severe muscle atrophy and degradation of motor end plates. Nerve conduction studies are best performed at least 10 days after injury to allow for Wallerian degeneration of axons to occur to avoid confounding results[21]. EMG cannot reliably ascertain the severity of nerve injury before this time[20,22].

Management: The treatment of paralysis focuses on the basic cause therapy. In cases of Bell's palsy, the form of treatment is not fully established yet. Some studies have sought to use Antiviral Agents, Corticoids and even surgical decompression of the nerve in search of some significant result. Ramsay Hunt syndrome management includes steroid therapy; acute otitis media and mastoiditis related bacterial infection require a complement of intravenous antibiotic therapy (type and duration according to local microbiological guidance). Facial palsy secondary to an acute middle ear infection is likely to require grommet insertion with or without mastoidectomy. Surgical intervention may also be required if the nerve has been damaged by cholesteatoma or due to surgery or temporal bone fracture.

Conservative:

Eye-care is incredibly essential in patients with facial nerve paralysis where there is corneal exposure. Artificial tears, adequate lubrication and taping the eyes at night ensure the prevention of corneal ulceration. Facial massage along with exercise promotes active rehabilitation and is essential for patients with facial nerve paralysis.

Medical:

Bell Palsy: current management Bell's palsy supported by a Cochrane review with over 1500 patients suggested that the use of steroids and analgesia will increase recovery of motor function if started within 72 hours of symptom onset[23]. The introduction of antivirals only showed minimal benefit for Bell' palsy and one study concluded that this combination should be reserved for Ramsay Hunt syndrome.[24] Over 70% of patients with Bell palsy will recover motor function completely within six months without any treatment[25]. Management of Ramsay Hunt syndrome includes steroid therapy with a full complement of treatment, facial nerve function is expected to recover in about 75% of patients.[26] There is a lack of evidence for whether the antiviral therapy is beneficial for Ramsay hunt syndrome but their use is still widely practiced[27]. Lyme disease management depends on patient age and disease severity.

Surgical:

Bell palsy: Over 90% degeneration on ENoG is associated with poor prognosis, and therefore, surgical decompression of the facial canal should merit consideration. However, this has not shown significantly positive outcomes compared to conventional medical treatment.[28] For acute suppurative otitis media along with mastoiditis, myringotomy with or without ventilation tube and or cortical mastoidectomy is advised.

Iatrogenic causes: If a facial nerve paralysis is apparent immediately after otological surgery, then a watch and wait policy should be adopted as this can be due to local anesthetic use. After the exclusion of a local anesthetic cause and assuming the surgeon is confident that the facial nerve epineurium is intact, a conservative approach with steroids is an option. Otherwise, an urgent re-exploration, facial decompression with or without nerve grafting must take place. Delayed palsies post-operatively can be due to edema and infection (requiring steroids and antibiotics) but also from over tight packing in open mastoid surgery, which requires removal.

Temporal bone fracture – if immediate and complete facial nerve paralysis occurs, then specialist nerve decompression is required as immediately as the patient's condition allows (usually within 2 to 3 weeks). If there is a delayed diagnosis and ENoG degeneration of more than 90%, then surgical exploration is required. Specific assessment of the facial nerve will help to dictate the approach as determining the site of nerve damage.

Different types of Surgeries for Facial nerve Paralysis: Facial nerve decompression is an option in cases of virally induced facial nerve paralysis and also for Bell palsy. A trans-mastoid approach would be best for cases of tympanic or mastoid segments damage to the facial nerve. If the damage expands to the labyrinthine segment, then a middle fossae approach allows precise decompression. The trans-labyrinthine method is reserved for cases of intratemporal decompression, where cochleovestibular function is absent. Facial nerve repair techniques can sub-classify into primary repair and cable grafting. Primary repair offers the maximum chance of return of facial nerve function. The aim is to provide a tension-free epineural repair, to avoid traction around the anastomosis and axonal injury[29]. If tension-free repair is not possible, then a cable graft approach is used. Frequently utilized nerves include the medial and lateral antebrachial cutaneous nerves as well as the sural nerve and great auricular nerve[30]. Nerve grafting options tend to be utilized in intermediate durations of facial paralysis (3 weeks to 2 years) with some studies suggesting the best outcomes if performed within six months[31]. This two-stage procedure involves an initial incision on the functional side of the face, nerve selection for sacrifice depending on desired functional outcome, and coaptation of the sural nerve graft to the donor facial nerve branches with tunneling to the contralateral side of the face. Following a 9 to 12 month waiting period, selected facial nerve branches and the cross-nerve grafts undergo secondary neurorraphies.[30] Muscle transfer techniques are suitable for those patients with chronic facial nerve palsy (older than two years). Regional muscle transfer most commonly utilizes the temporalis muscle; however, the digastric (marginal mandibular nerve injury) and masseter (smile reanimation) are also options. If using temporalis, it is essential to ensure adequate trigeminal nerve function before proceeding. A 1.5 to 2 cm strip of the temporalis is raised and rotated inferiorly beyond the zygoma to the oral commissure to align with the smile vector.[30]

Transcutaneous Nerve Stimulation: Transcutaneous nerve stimulation is an additional new treatment option for those with unilateral facial nerve palsy. The technology uses EMG signals from muscles on the intact side of the face to simultaneously stimulate the corresponding muscles on the side of paresis. The ultimate aim of therapy is to achieve facial symmetry. Early trials have shown positive results in significant domains of facial expression where the affected side is paretic, with some degree of reinnervation.[32]

CONCLUSION

Evaluation and management of facial nerve paralysis is perhaps one of the most challenging decision-making procedure for the otolaryngologists. In cases of transected nerves direct coaptation can be resulted to the best outcome followed by interpositional nerve grafting. A nerve transfer should be employed where the motor end plates are intact and primary repair or graft is not feasible. When complete muscle atrophy has occurred, regional muscle transfer or free flap reconstruction is an option. Static procedure can be beneficial when dynamic reanimation is a failure. While controversies in the management of facial nerve paralysis have always been and will always remain, most surgeons have devised their own strategies for their management. The outcome of facial nerve paralysis is not very gloomy as it was in the past, provided the patients report early. Incomplete palsies almost always recover. A facial nerve paralysis that is total or becomes complete within two to three days may not recover fully. During surgery, the fear of the facial nerve has resulted in more iatrogenic palsies than when the nerve and its landmarks have been routinely identified.

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