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Short Review

All About Facial Nerve Palsy, A Review of Current Literature

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ABSTRACT

With a prevalence between 11 to 53 cases, for every 100,000 people per year, facial paralysis is a disease of multiple possible etiologies, being neural infection by herpes virus simplex type 1 the most common cause, mainly manifested by weakness of facial musculature and loss of sensation. It does not have a specific treatment and its full recovery depends on the application of multitherapy in a timely manner.

INTRODUCTION

Acute facial nerve palsy is a common disorder that manifests with the acute to subacute onset of unilateral paralysis of the muscles of facial expression. The Scottish physician Sir Charles Bell described cases of facial paralysis due to trauma in 1800, and the idiopathic condition is named after him today. However, there are other many possible causes of facial paralysis, and each case demands taking a careful history and performing a comprehensive neurologic examination to determine an etiology to start proper treatment. Symptoms appear suddenly over a period of 48 to 72 hours and generally begin to improve with or without treatment after a few weeks, with the recovery of some or all facial functions within six months. In some cases, residual muscle weakness lasts longer or might be permanent [1,2].

EPIDEMIOLOGY

Different authors agree on an incidence of between 11 to 53 cases, per 100,000 people per year. It has been documented that the incidence between men and women is similar, with a bimodal presentation between the ages of 20 to 29 years and 50 to 59, and 3.3 times more frequent in pregnant than in the non-pregnant woman. The American Academy of Otolaryngology and Head and Neck Surgery describes certain risk factors for the development of the disease, such as diabetes, obesity, high blood pressure, upper airway infections, immunocompromised state, and pregnancy [3].

ETIOLOGY

Bell´s palsy is the leading cause of acute unilateral facial nerve paralysis. Traditionally, it has been considered idiopathic (60-75%) although the most recent research shows a reactivation from latent state of herpes simplex virus infection (HSV-1) in the geniculate ganglion.4 Among other infectious etiologies identified, Ramsay-Hunt syndrome and neuroborreliosis have been described. Herpes zoster should be considered if the patient has severe pain and even if no herpetic blisters are present (herpes zoster sine herpete) [5]. Other less common causes of infectious facial

paralysis include rickettsia, HIV, human herpesvirus 6, mumps virus, cytomegalovirus, rubella virus and infections of the middle ear and mastoid [6]. Neoplastic etiologies are less common. Tumors arising from the facial nerve or from its nearby structures can cause functional compression of this nerve. The main tumors that have been associated to facial paralysis are acoustic neuromas, glomus, meningiomas, parotid gland tumors, and metastatic breast and lung cancer [7].

Table 1 shows types of nerve paralysis that can occur depending on the location of the injury and etiology [8].

Table 1: Differential diagnosis of Facial nerve paralysis [8].

Type	Etiology	Causal agent/ Pathophysiology
Central Facial Paralysis	Vascular	Infarction/hemorrhage
Supranuclear: Involvement of the motor cortex or corticobulbar tract Nuclear: Lesion of the VII craneal nerve at level of the protuberance	Demyelination	
	Vascular malformation	
	Tumor	Primary/metastasis
	Motor neurone disease	
Unilateral peripheral facial paralysis	Viral	Bell: Idiopathic and other viruses (Epstein Barr, Hepatitis B, Cytomegalovirus, Rubella, Mumps, Measles, Influenza, Adenovirus, Mononucleosis.
		Otic herpes zoster
		HIV
		Post rabies vaccine
	Acute and chronic otitis media, Otomastoiditis	*Haemophylus influenzae, Streptococcus pneumonaie, Streptococcus pyogenes, Staphylococcus aureus.*
	Tuberous otitis, Cholesteatoma	Tympanic rupture
	Malignant otitis externa	*Pseudomona aeruginosa and Staphylococcus aureus*
	Meningitis	Secondary and tertiary syphilis, HIV and tuberculosis
	Lyme's disease	*Borrelia burgdorferi*
	Carcinomatous meningitis	Lymphoma, leukemia
	Brucellar meningitis	*Brucella*
	Immune-based diseases	Collagenosis
		Vasculitis
		Sarcoidosis
		Behcet's disease
	Peripheral nervous system diseases	Guillain Barre's syndrome
		Diabetes Mellitus
		Myasthenia gravis, Steinert's disease, and Polymyositis
		Leprosy and Amyloidosis
		B1, B3 and B12 vitamin deficit
	Traumatic	Labyrinth and extralabyrinth fracture of the temporal bone
		Jaw fracture
		Penetrating wounds
	Neoplastic	Cerebellopontine and parotid angle
		Facial neuroma
		Otic carcinoma
	Iatrogenic	Otologic and skull base surgery
		Parotidectomy
		Mandibular block
	Congenital	Osteomandibular syndrome
		Moebius Syndrome
Bilateral peripheral facial paralysis	Neuroborreliosis	*Borrelia Burgdorferi*
	Guillain Barre's syndrome	
	Neurosarcoidosis	
	Multiple sclerosis	
	Meningeal carcinomatosis	
	Viral multineuritis	
	Bell's palsy	
	Meningitis involving skull base	

PATHOPHYSIOLOGY

Any mechanism that injures the fibers of the facial nerve will result in alterations of its function, whether they are motor, secretory, or sensorial. The clinical picture will depend on the site of injury and the involved nerve branches. One of the theories that have been supported forward over the years is that any process that causes ischemia, inflammation, or demyelination can result in compression of the nerve as it

passes through the bony facial canal in the temporal bone preventing nerve's conduction at this site [9,10].

If the lesion originates near the geniculated ganglion, the motor dysfunction will be accompanied by taste disorders and autonomic dysfunctions such as decreased tearing and salivation. Injuries between the geniculate ganglion and the chorda tympani nerve of the eardrum will produce the same alterations except for tearing. If the lesion is at the level of the stylomastoid foramen, the patient will present only motor alterations [11,12] see (Figure 1).

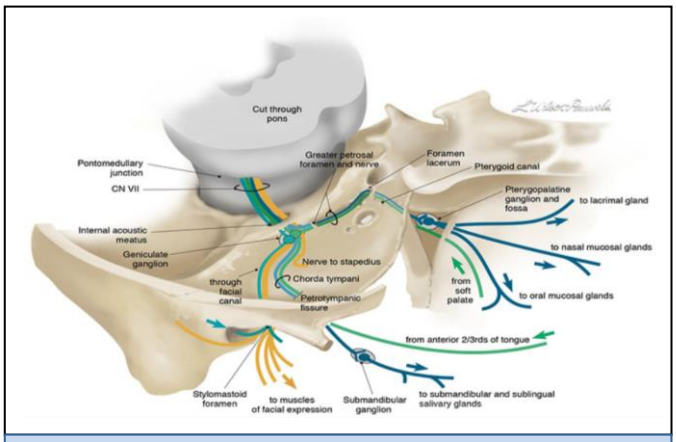

Figure 1: Superior view of the route of cranial nerve VII from the pons through the cranium (brainstem is elevated).

Description: General sensory afferent (blue-green), special sensory afferent (green), branchial motor efferent (yellow), visceral motor (parasympathetic) efferent (blue) [12].

Table 2: House Brackmann scale [18].

Grade	Function level	Simmetry at rest	Eyes	Mouth	Forehead	Synkinesias
I	Normal	Normal	Normal	Normal	Normal	None
II	Mild	Normal	Easy and complete closure	Slightly asymmetrical	Reasonable function	minimum
III	Moderate	Normal	With effort, complete closure	Slightly affected with effort	Slight to moderate movement	Existence of synkinesias and / or increased tone of facial muscles
IV	Moderately severe	Normal	Incomplete closure	Asymmetrical with maximum effort	None	
V	Severe	Asymmetry	Incomplete closure	Minimal movement	None	
VI	Total paralysis	Total paralysis				

CLINICAL MANIFESTATIONS

The clinical manifestations of Bell's palsy originate from compromised muscle contractility with damage to the neural control functions of the facial nerve, leading to progressive hemifacial muscle weakness and loss of sensation with complete or incomplete nerve involvement. Before paralysis, a banal picture and more or less intense pain may appear in the retroauricular region, especially in patients with Ramsay-Hunt Syndrome. The paralysis then sets within a few hours, and can sometimes worsen in 24 to 48 hours [13]. Usually, a decrease in facial muscle strength is observed, characterized by a sudden deviation of the oral commissure, difficulty to raise the eyebrow and smile, inability to close the eyelids, and an asymmetrical smile or lip position. Smooth forehead, changes in nasal breathing, biting of the oral mucosa, accumulation of food and saliva in the oral cavity or in the oropharynx, occur quite frequently [14].

DIAGNOSIS

The diagnosis of Bell's palsy should be based on the existence of peripheral facial paralysis with or without loss of taste of the anterior 2/3 of the tongue or altered secretion of the salivary or lacrimal glands. The onset must be acute, reaching the maximum degree of paralysis within the first three weeks, and it may or may not be associated with otalgia or hyperacusis [15]. Inspection should be performed with the patient at rest and during facial motility activity, emphasizing the search for indefinite or absent facial folds, impaired palpebral closure, epiphora, deviation of the oral commissure, decreased of movements of facial expression, conjunctival hyperemia, and Bell's sign.

Laboratory tests should not be routinely requested in patients diagnosed with Bell's palsy. MRI can only be justified when the patient has little or no recovery after three months [16,17].

Bell's palsy ophthalmologic evaluation should include:

- Elevating function of the upper eyelid
- Closure of the upper eyelid
- Bell phenomenon
- Presence of ectropion
- Retraction of the lower eyelid
- Conjunctival injection
- Corneal sensitivity

To assess the involvement and severity of the facial nerve in Bell's palsy, the House Brackmann Classification was accepted in 1985 by the American Academy of Otolaryngology and Head and Neck Surgery (Table 1) [18]. Some differential diagnoses of facial paralysis are Stroke, Herpes Zoster, Otitis media, Lyme's disease, Guillain Barré's syndrome, HIV, sarcoidosis, Sjögren's syndrome, tumors, and Melkersson-Rosenthal's syndrome [19].

TREATMENT

The initial treatment will depend on the cause:

- Bell's palsy or idiopathic: In the case of idiopathic facial paralysis, the initial treatment generally consists of the administration of corticosteroids:

Medical treatment

Administration of oral corticosteroids in the first 48 to 72 hours after the onset of the disease in patients 16 years of age or older. The recommended dose is Prednisone 60 mg per day, for 5 days with a reduced dose of 10 mg per day until reaching 10 mg and discontinue, to aid in the inflammation of the nerve. Prednisone is also advised in the treatment of facial paralysis in diabetics, Heerfordt syndrome, and the paralysis that follows dental surgery [20]. The use of antivirals as monotherapy has been discouraged, as several meta-analyses have not shown better outcomes compared to placebo [21-24]. On the other hand, the prescription of antivirals along with steroids is controversial. Large trials and several meta-analyses have not found any benefit in facial nerve recovery when antivirals are added to steroids [25-29]. However, smaller trials have found limited improvements in long-term recovery. The American Academy of Otolaryngology and Head and Neck Surgery suggests, as an option, the use of antivirals only in combination with oral steroids therapy when patients are diagnosed within 72 hours since facial paralysis onset, as a small benefit cannot be excluded given viral pathophysiology [30].

Surgical treatment

Identifying candidates for surgical decompression includes the use of electrophysiologic testing. Surgical candidates usually have more than 90% of facial nerve degeneration along with negative electromyography within 14 days of onset of total paralysis. Surgical decompression must also be performed within this time frame in order to be effective. Several techniques exist; however, the middle cranial fossa approach is the most commonly used for decompression in patients with Bell's Palsy. This approach effectively exposes the facial nerve's course at the level of the meatal foramen and labyrinthic portion, the most common site for compression. One of its main advantages is inner ear preservation,in contrast to other techniques. Using the mentioned criteria to select surgical candidates, Gantz et al found recovery to normal facial function in 91% of their sample [31].

Adjunct measures

Eye protection for patients with Bell's palsy who have impaired eyelid closure, use of sunscreen, frequent administration of drops and lubricating ointments, 0.5% hypromellose during the day and 2% hypromellose at night, use of contact lenses if it does not respond to the use of lubricants or occlusive patch, for eyelid closure by using adhesive tape during the night and day or the placement of weight on the upper eyelid if the drying of the cornea is very important [32]. These measures apply for every patient with facial palsy of any etiology.

Ramsay-hunt syndrome

Medical treatment: In patients with Ramsay- Hunt syndrome, steroids along with antivirals has been observed to yield the best outcomes for facial nerve recovery. Dosage and route of administration vary within different clinical trials [33]. Kinishi et al, proposed the use of methylprednisolone 500mg for day 1, 250 mg for days 2 and 3, and 100mg for days 4 through 7 with 4g/day of acyclovir for seven days. This scheme resulted in a recovery rate of 93.5% compared to 68% in the only steroid group [34]. Ryu et al observed similar outcomes facial nerve recovery, using prednisolone 80mg/day for the first four days, 60 mg/day for two days, 40mg/day for two days, 20mg/day for two days and finishing with 10mg/day for five days. This was accompanied by 4g/day of acyclovir for seven days [35].

Surgical treatment: Candidates for surgery are selected upon previously mentioned electrophysiological testing characteristics for Bell's Palsy.

- Paralysis secondary to facial nerve injury: If the paralysis is due to trauma, wound cut, fracture, or resection of a tumor, corticosteroid therapy may be administered. Subsequently, it can be corrected with surgery for early repair [36].

Surgical treatment

Surgical treatment in these cases should be individualized. It is important to assess facial paralysis thoroughly, interrogating about time since injury, type of injury, age, overall health, and previous radiation therapy. Physical examination should not be overlooked and must include facial motion, the integrity of hypoglossal, vagal and trigeminal nerves, and the status of the eye. In some cases, electrophysiologic testing might aid in the decision for the best surgical approach.

1. Dynamic surgical rehabilitation procedures: their objective is to recover facial motion and they include facial nerve primary neurorrhaphy, nerve graft, nerve transposition, musculature transposition, and microneurovascular transference. In general, these procedures should be reserved for those cases of complete paralysis of a non-inflammatory cause, due to traumatic or surgical causes, or when a section of the facial nerve has occurred.

2. Static techniques: they do not aim to recover muscle movement and are mainly used in poor surgical candidates for dynamic techniques or in combination with them. They are most used at the level of the forehead and the eye. It includes procedures such as the browpexy, the front lift, the placement of a gold weight, to help close the eye, and blepharoplasty in some cases. Patients in whom the paralyzed side is very low and are not candidates for dynamic techniques, facial elevation at the level of the cheek with tendons from the same patient is a very good alternative for static resuscitation.

- Facial paralysis <2 years of evolution: The musculature is still viable and, therefore, is likely to be reinnervated. It can be achieved with different techniques, such as a cross facial graft or masseteric or hypoglossal nerve transfers [37].
- Facial paralysis > 2 years evolution: There is irreversible atrophy of the muscle and, therefore, it is necessary to carry a new muscular unit to restore movement. This involves performing a muscle transplant from the thigh region (gracilis muscle) to the patient's face [37].
- Complete unilateral facial paralysis: Cross graft from sural nerve to contralateral facial nerve and transfer of free muscle flap (gracilis or anterior serratus) in two stages. As an alternative, an ipsilateral sural nerve graft with V-pair anastomosis and free muscle transfer at one time is also considered [37].
- Complete bilateral facial paralysis or Möebius syndrome: Transfer of free muscle flap (gracilis) with anastomosis to the ipsilateral trigeminal motor branch (masseteric) at one time. In three months the second surgery is performed (contralateral side) [37].

Each case must be individualized to select the appropriate treatment [38,39].

PHYSICAL REHABILITATION

Facial paralysis improves completely without treatment in most patients. Physical therapies such as exercise, laser treatment, electrotherapy, massage, and thermotherapy have been proposed to speed recovery, improve facial function, and minimize sequelae [40]. A systematic review in this topic concluded that there is no high-quality evidence to support significant benefit or harm from any physical therapy for idiopathic facial paralysis. There is low-quality evidence that tailored facial exercises can help to improve facial function, mainly for people with moderate paralysis and chronic cases. There is low-quality evidence that facial exercise reduces sequelae in acute cases [41].

PROGNOSIS

The prognosis for patients with Bell's palsy is generally good; the extent of nerve damage determines recovery time. The single most important prognostic factor is whether de paralysis is complete or incomplete. If there is any function left, full recovery occurs within a few months. The probability of complete recovery after total paralysis is 90% if the nerve branches in the face retain normal excitability for supramaximal electrical stimulation and only 20% if electrical excitability is absent [7]. Other studies have claimed that advanced age, hypertension, diabetes mellitus are associated with poorer outcomes, however, these results have been inconsistent [42-45]. Rates of favorable recovery for facial palsy are importantly decreased in patients with Ramsay-Hunt Syndrome compared to Bell's palsy [21]. Left without treatment, complete recovery is only achieved in 22% of the cases [46]. After a timely medical treatment with steroids and antivirals, recovery rates increase up to 93.4% [33]. According to Yeo et al, advanced age, diabetes mellitus, hypertension, and associated vertigo are associated with a poor prognosis

for recovery [47]. Temporality of facial palsy after temporal bone trauma has been advocated as an important predictor of recovery in a recent systematic review. Delayed-onset palsy was associated with recovery in 80% of the cases compared to 40% in patients with an immediate onset. As with Bell's Palsy and Ramsay Hunt Syndrome, palsy's severity is an important prognostic factor [48].

CONCLUSION

Acute facial paralysis is a common disorder in the general population. Possible etiologies include Bell's palsy, Ramsay-Hunt Syndrome, traumatic injury, infectious diseases, among others. Thus, every case should be approached individually. A complete medical history and neurotological examination are the keys to establish a diagnosis. Treatment modalities are varied and include medical, surgical, and rehabilitation options. The prognosis for recovery will depend upon the cause and selection of the proper treatment for each case.

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